



## **BASIC SURGERY**





# BASIC SURGERY

Edited by  
**LESLIE OLIVER**

M.B., B.S. (London), F.R.C.S. (Eng.), F.R.C.S.

*Neurosurgery*—West London Hospital and Medical School, Royal Victoria Hospital, London, West End Hospital for Neurology and Neurosurgery, London, The Park Hospital, London, The Southwick General Hospital, Southwick, Sussex, Plastic Neurosurgery, The Park Hospital, London, Essex, The Park Neurosurgery, The Southwick General Hospital, Southwick, Essex.

*With 650 illustrations (including 4 coloured plates)*

LONDON  
**H. K. LEWIS & Co. Ltd.**  
1958



H. K. LEWIS & Co. Ltd.

1958

PRINTED IN GREAT BRITAIN BY THE WHITEFRIARS PRESS LTD.  
LONDON AND TONBRIDGE

## PREFACE

THIS book has been written primarily for undergraduate students by a team of writers with special experience of their subjects. Sufficient operative surgery has been included to meet the needs of those students intending to specialise in surgery.

I am most grateful to J. H. Evans, M.D., M.R.C.P.; Bernard Fairbairn, M.B., F.R.C.S.; Douglas A. Langley, F.R.C.S.; William L. B. Leese, M.B., M.R.C.P.; J. N. Milnes, M.D., M.R.C.P.; and K. W. E. Paine, M.B., F.R.C.S., for their kindness in reading parts of the manuscript and for their helpful suggestions.

Some of the illustrations were contributed by the photographic departments of St. Mary's Hospital, London, The Hospital for Sick Children, Great Ormond Street, London, and Oldchurch Hospital, Romford.

LESLIE OLIVER.

London, 1958.



## CONTRIBUTORS

**A. G. POSSIS, M.B., B.S.(Orel.), F.R.C.S.(Eng.)**

Orthopaedic Surgeon, Central Middlesex Hospital, London  
*Orthopaedic Surgery - Injuries to Joints and Bones - The Hand*

**D. L. B. FARLEY, M.B., B.S.(Lond.), F.R.C.S.(Eng.)**

Senior Surgical Registrar, Royal Northern Hospital, London. Late  
Assistant to the Surgical Unit, St. Mary's Hospital, London. Late  
Senior Surgical Registrar, The Hospital for Sick Children, Great Ormond  
Street, London

*Hernias in Adults - The Neck - Peripheral Vascular Surgery*

**GEORGEY FLAVELL, F.R.C.S.(Eng.), M.B.C.P.(Lond.)**

Thoracic Surgeon, The London Hospital. Thoracic Surgeon, Royal  
Masonic Hospital. Senior Thoracic Surgeon, Broomfield Hospital.  
Consultant Thoracic Surgeon to the hospitals of the North-East Metro-  
politan Region

*Thoracic Surgery*

**R. S. MURLEY, M.B., M.S.(Lond.), F.R.C.S.(Eng.)**

Surgeon, Royal Northern Hospital, London. St. Albans City Hospital,  
Wood Green and Southgate Hospital, London

*Abdominal Surgery*

**LESLIE OLIVER, M.B., B.S.(Lond.), F.R.C.S.(Eng.), F.A.C.S.**

Neurosurgeon: West London Hospital and Medical School; Royal  
Northern Hospital, London; West End Hospital for Neurology and  
Neurosurgery, London; The French Hospital, London; The Southend  
General Hospital, Southend-on-Sea; Senior Neurosurgeon, Oldchurch  
Hospital, Romford, Essex; Visiting Neurosurgeon, The Jackson Clinic,  
Madison, Wisconsin, U.S.A.

*Neurosurgery*

**BASIL H. PAGE, M.A., M.Ch.(Camb.), F.R.C.S.(Eng.)**

Surgeon and Urologist, North Middlesex Hospital, London  
*Genito-Urinary Surgery - The Breast*

## CONTRIBUTORS

J. E. PIERCY, F.R.C.S.(Eng.), F.R.C.S.(Ed.)

Senior Surgeon, New End Hospital, London. Surgical Director, New End Thyroid Clinic and Department of Endocrinology, London

*The Surgery of the Endocrine Glands*

VALENTINE A. J. SWAIN, F.R.C.S.(Eng.)

Surgeon, Queen Elizabeth Hospital for Children, London. Surgeon, Institute of Child Health, University of London. Clinical Research Assistant, Department of Surgery, Postgraduate Medical School of London

*Pediatric Surgery—The Face, Mouth, Lips, Tongue and Jaws—Burns*

---

*Adviser in Pathology*

P. M. PETERS, M.A., D.M.(Oxf.)

Pathologist, Royal Northern Hospital, London

---

## Associate Contributors

CHALMERS BALLANTYNE, F.R.C.S.(Eng.), D.L.O. E.N.T. Surgeon, Royal Northern Hospital, London. Assistant Director, Audiology Unit, National Throat, Nose and Ear Hospital. Hon. First Assistant E.N.T. Department, St. Mary's Hospital, London.

ARNOLD S. JACKSON, M.D., F.A.C.S. Senior Surgeon, Jackson Clinic, Madison, Wisconsin, U.S.A.

A. G. LEWIS, B.Sc., M.D., B.S.(Lond.), M.R.C.P.(Lond.). Physician, Connaught Hospital, London, and Jubilee Hospital, Woodford.

I. CLIVE SIMS, M.D., Ph.D., D.M.R. Radiotherapist, Oldchurch Hospital, Romford, Essex.

LT.-COL. P. ROSS WHEATLEY, D.S.O., M.B., B.S.(Lond.), F.R.C.S.(Eng.). R.A.M.C.

WINIFRED F. YOUNG, M.A., M.D.(Camb.), D.C.H.(Eng.). Research Physician, Queen Elizabeth Hospital for Children, London.

# CONTENTS

## SECTION I

### ABDOMINAL SURGERY

By R. S. MURPHY

CHAPTER	PAGE
1. THE APPENDIX . . . . .	1
2. THE STOMACH AND DUODENUM . . . . .	12
3. THE GALL BLADDER AND BILE DUCTS . . . . .	47
4. THE PANCREAS . . . . .	65
5. THE LIVER . . . . .	77
6. THE SPLEEN . . . . .	
By A. A. G. LEWIS and R. S. MURPHY . . . . .	94
7. THE PERITONEUM, OMENTUM AND MESENTERY . . . . .	99
8. THE SMALL INTESTINE . . . . .	109
9. INTESTINAL OBSTRUCTION . . . . .	121
10. THE COLON, RECTUM AND ANAL CANAL . . . . .	132
11. AMOEBIASIS . . . . .	
By Lt. Col. P. DEAN WHEATLEY, D.S.O., R.A.M.C. . . . .	142
12. ABDOMINAL INCISIONS . . . . .	191
13. ABDOMINAL HERNIAS IN ADULTS . . . . .	
By D. L. B. FARLEY . . . . .	197

## SECTION II

### PÆDIATRIC SURGERY

By VALENTINE A. J. SWAIN

14. THE INGUINO-SCROTAL REGION . . . . .	223
15. ABNORMALITIES OF THE ANTERIOR ABDOMINAL WALL . . . . .	234
16. CLEFT LIP AND CLEFT PALATE . . . . .	241
17. CONGENITAL ABNORMALITIES OF THE OESOPHAGUS AND DIAPHRAGM . . . . .	250
18. THE STOMACH, DUODENUM AND BILIARY TRACT IN CHILDREN . . . . .	255
19. CONGENITAL DEFECTS OF THE JEJUNUM, ILEUM AND COLON . . . . .	264
20. MALFORMATIONS OF THE RECTUM AND ANUS . . . . .	270
21. TUMOURS AND SINUSES IN THE SACROCOCCYGEAL REGION . . . . .	284
22. SOME ACUTE ABDOMINAL CONDITIONS IN CHILDREN . . . . .	287
23. FLUID REQUIREMENTS FOR CHILDREN UNDERGOING SURGICAL TREATMENT . . . . .	297
24. CONGENITAL ABNORMALITIES OF THE URINARY TRACT . . . . .	302



## CONTENTS

### SECTION III GENITO-URINARY SURGERY

By BASIL H. PAGE

AFTER	PAGE
INVESTIGATION OF A GENITO-URINARY CASE . . . . .	319
DISEASES OF THE KIDNEY . . . . .	328
DISEASES OF THE URETER . . . . .	357
THE BLADDER . . . . .	365
DISTURBANCES OF MICTURITION AND DISEASES INVOLVING THE BLADDER NECK . . . . .	383
DISEASES OF THE URETHRA AND PENIS . . . . .	407
DISEASES OF THE TESTIS AND ITS APPENDAGES . . . . .	423
GENITO-URINARY TUBERCULOSIS . . . . .	437

### SECTION IV THORACIC SURGERY

By GEOFFREY FLAVELL

13. BRONCHOSCOPY . . . . .	445
14. LUNG ABSCESS . . . . .	457
15. NON-TUBERCULOUS EMPYEMA . . . . .	476
16. BRONCHIECTASIS . . . . .	489
37. BRONCHIAL CARCINOMA . . . . .	496
38. INNOCENT TUMOURS OF THE LUNG . . . . .	508
39. CYSTS OF THE LUNG . . . . .	511
40. CYSTS AND TUMOURS OF THE MEDIASTINUM . . . . .	518
41. THE SURGERY OF PULMONARY TUBERCULOSIS . . . . .	529
42. PULMONARY RESECTIONS . . . . .	549
43. DIAPHRAGMATIC HERNIAS . . . . .	567
44. THE ŒSOPHAGUS . . . . .	576
45. THE SURGICAL TREATMENT OF MITRAL STENOSIS . . . . .	593
46. CONSTRICTIVE PERICARDITIS . . . . .	605
47. CONGENITAL HEART DISEASE . . . . .	610
48. PATENT DUCTUS ARTERIOSUS . . . . .	626
49. COARCTATION OF THE AORTA . . . . .	630

### SECTION V NEUROSURGERY

By LESLIE OLIVER

50. HEAD INJURIES . . . . .	635
51. INTRACRANIAL SUPPURATION . . . . .	655
52. THE CRANIAL NERVES . . . . .	666
53. INTRACRANIAL TUMOURS . . . . .	695
54. OPERATIVE TECHNIQUE AND EXPOSURES OF INTRACRANIAL SURGERY . . . . .	732

## CHAPTER

## PAGE

55.	INFANTILE HYDROCEPHALUS . . . . .	732
56.	THE SURGERY OF INTRACRANIAL VASCULAR DISEASES . . . . .	745
57.	THE SURGERY OF EPILEPSY AND MENTAL DISEASES . . . . .	755
58.	TUMOURS OF THE SKULL . . . . .	764
59.	THE SPINAL CORD AND ITS NERVE ROOTS . . . . .	770
60.	THE AUTONOMIC NERVOUS SYSTEM . . . . .	794
61.	THE PERIPHERAL NERVES . . . . .	803

## SECTION VI

## THE FACE, MOUTH, LIPS, TONGUE AND JAWS

By VALENTINE A. J. SWAIN

62.	DISEASES OF THE FACE . . . . .	825
63.	DISEASES OF THE MOUTH AND LIPS . . . . .	844
64.	THE TONGUE . . . . .	849
65.	DISEASES OF THE JAWS . . . . .	854

## SECTION VII

## THE NECK

By D. L. B. FARLEY and ARNOLD S. JACKSON

66.	SWELLINGS OF THE NECK . . . . .	861
67.	THE CERVICAL LYMPH GLANDS . . . . .	871
68.	THE LARYNX AND PHARYNX . . . . .	
	By J. CHALMERS BALLANTYNE . . . . .	878

## SECTION VIII

## THE SURGERY OF THE ENDOCRINE GLANDS

By J. E. PIERCE

69.	THE THYROID GLAND . . . . .	885
70.	THE THYMUS . . . . .	912
71.	THE PARATHYROID GLANDS . . . . .	922
72.	THE ADRENAL GLANDS . . . . .	930

## SECTION IX

## 73. THE BREAST

By BASIL H. PAGE . . . . . 939

### SECTION III

#### GENITO-URINARY SURGERY

By BASIL H. PAGE

CHAPTER	PAGE
25. INVESTIGATION OF A GENITO-URINARY CASE . . . . .	319
26. DISEASES OF THE KIDNEY . . . . .	328
27. DISEASES OF THE URETER . . . . .	357
28. THE BLADDER . . . . .	365
29. DISTURBANCES OF MICTURITION AND DISEASES INVOLVING THE BLADDER NECK . . . . .	383
30. DISEASES OF THE URETHRA AND PENIS . . . . .	407
31. DISEASES OF THE TESTIS AND ITS APPENDAGES . . . . .	423
32. GENITO-URINARY TUBERCULOSIS . . . . .	437

### SECTION IV

#### THORACIC SURGERY

By GEOFFREY FLAVELL

33. BRONCHOSCOPY . . . . .	445
34. LUNG ABSCESS . . . . .	457
35. NON-TUBERCULOUS EMPYEMA . . . . .	470
36. BRONCHIECTASIS . . . . .	489
37. BRONCHIAL CARCINOMA . . . . .	496
38. INNOCENT TUMOURS OF THE LUNG . . . . .	508
39. CYSTS OF THE LUNG . . . . .	511
40. CYSTS AND TUMOURS OF THE MEDIASTINUM . . . . .	518
41. THE SURGERY OF PULMONARY TUBERCULOSIS . . . . .	529
42. PULMONARY RESECTIONS . . . . .	549
43. DIAPHRAGMATIC HERNIAS . . . . .	567
44. THE OESOPHAGUS . . . . .	576
45. THE SURGICAL TREATMENT OF MITRAL STENOSIS . . . . .	593
46. CONSTRICTIVE PERICARDITIS . . . . .	605
47. CONGENITAL HEART DISEASE . . . . .	610
48. PATENT DUCTUS ARTERIOSUS . . . . .	626
49. COARCTATION OF THE AORTA . . . . .	630

### SECTION V

#### NEUROSURGERY

By LESLIE OLIVER

50. HEAD INJURIES . . . . .	635
51. INTRACRANIAL SUPPURATION . . . . .	655
52. THE CRANIAL NERVES . . . . .	666
53. INTRACRANIAL TUMOURS . . . . .	695
54. OPERATIVE TECHNIQUE AND EXPOSURES OF INTRACRANIAL SURGERY . . . . .	732

**PART I**

	PAGE
98. FRACTURES OF THE SPINE . . . . .	1229
99. FRACTURES OF THE PELVIS AND SACRUM . . . . .	1234
100. FRACTURES OF THE UPPER END OF THE FEMUR . . . . .	1239
101. FRACTURES OF THE SHAFT OF THE FEMUR . . . . .	1244
102. FRACTURES AROUND THE KNEE JOINT . . . . .	1254
103. FRACTURES OF THE TIBIAL SHAFT AND THE FIBULA . . . . .	1260
104. FRACTURES AND FRACTURE-DISLOCATIONS OF THE ANKLE . . . . .	1269
105. FRACTURES OF THE TARSUS, METATARSALS, AND PHALANGES . . . . .	1279
106. INJURIES AND INFECTIONS OF THE HAND . . . . .	1284
107. BURNS . . . . .	
By VALENTINE A. J. SWAIN . . . . .	1293

**SECTION XIII**

**REACTIONS TO INJURY. BLOOD TRANSFUSION.  
SURGERY IN DIABETICS. ANTIMIOTIC THERAPY**

By A. A. G. LEWIS

108. REACTIONS TO INJURY. SURGERY IN DIABETICS . . . . .	1301
109. BLOOD TRANSFUSION . . . . .	1310
110. ANTIMIOTIC THERAPY . . . . .	1314

**SECTION XIV**

**111. THE PRINCIPLES AND SURGICAL APPLICATIONS OF RADIO-  
THERAPY**

By H. CLAY KIMM . . . . . 1319

INDEX . . . . .	1328
-----------------	------

## SECTION X

## PERIPHERAL VASCULAR SURGERY

By D. L. B. FARLEY

CHAPTER	PAGE
74. THE PERIPHERAL CIRCULATION. THE ARTERIES . . . . .	961
75. THE VEINS . . . . .	996
76. HÆMANGIOMAS AND ALLIED CONDITIONS	
By VALENTINE A. J. SWAIN . . . . .	1018
77. THE LYMPHATICS . . . . .	1023

## SECTION XI

## ORTHOPÆDIC SURGERY

By J. G. BONNIN

78. CONGENITAL DEFORMITIES . . . . .	1033
79. HÆMATOGENOUS OSTEOMYELITIS . . . . .	1038
80. TUBERCULOSIS . . . . .	1046
81. CHRONIC ARTHRITIS . . . . .	1052
82. DISEASES OF BONE METABOLISM . . . . .	1064
83. TUMOURS OF BONES AND JOINTS . . . . .	1069
84. THE SPINE . . . . .	1080
85. THE SHOULDER . . . . .	1096
86. THE ELBOW . . . . .	1100
87. THE WRIST AND HAND . . . . .	1102
88. THE HIP . . . . .	1109
89. THE KNEE . . . . .	1123
90. THE ANKLE AND FOOT . . . . .	1138

## SECTION XII

## INJURIES TO JOINTS AND BONES

## THE HAND. BURNS

By J. G. BONNIN

91. INJURIES TO JOINTS . . . . .	1157
92. FRACTURES AND THEIR TREATMENT . . . . .	1172
93. COMPOUND FRACTURES AND THEIR TREATMENT . . . . .	1188
94. INTERNAL FIXATION OF FRACTURES . . . . .	1196
95. THE COMPLICATIONS OF BONY INJURIES . . . . .	1200
96. COLLES' FRACTURE AND FRACTURES OF THE WRIST, HAND AND FOREARM . . . . .	1211
97. FRACTURES AROUND THE ELBOW JOINT, FRACTURES OF THE HEAD AND SHAFT OF THE HUMERUS, THE CLAVICLE, SCAPULA, RIBS AND STERNUM . . . . .	1222

# SECTION I

## ABDOMINAL SURGERY

by

R. S. MURLEY

*including*

AMOEBIASIS, by LT. COL. P. ROSS WHEATLEY, D.S.O., R.A.M.C.  
HERNIAS IN ADULTS, by D. L. B. FARLEY.

### CHAPTER I

#### THE APPENDIX

##### ACUTE APPENDICITIS

ACUTE appendicitis is the commonest abdominal emergency seen in hospital and general practice in Great Britain. Although there had been prior descriptions of the pathology and previous operative removal of the appendix, it was Reginald Fitz of Boston who, in 1886, first named appendicitis and emphasised the importance of early diagnosis and operation. Appendicitis became much more common, both in Britain and the United States, during the latter part of the nineteenth century. This increased incidence was certainly real and not simply due to improved standards of diagnosis. Appendicitis is essentially an affliction of civilised communities living on a sophisticated diet and it may be related to low cellulose and high protein intake. The varying incidence in different countries is probably related to these last factors rather than any intrinsic racial difference. In contrast to its frequency in Britain and the United States, appendicitis is uncommon in Spain and Italy; rare in eastern Europe, and most rare in Africans who are eating their native diet.

**Anatomy.** The appendix is attached to the postero-medial aspect of the caecum; its proximal end is about 2.5 cm. below the ileocaecal valve. If the organ is not immediately visible at operation, it can be identified by tracing the anterior *tenia* of the colon downwards to its base. The organ varies from 2.5 to 20 cm. in length; it is, on average, about 7.5 cm. long. Congenital absence of the appendix is exceptionally rare, but sometimes it is buried in the wall of the caecum and difficult to find at operation. More common than burial of the whole organ is the concealment of the proximal 1 or 2 cm. in the wall of the caecum. Failure to recognise this may result in an incomplete appendicectomy. The blood supply is from



glands of the mucosa, or through the submucosal lymphoid tissue, the histological features are those of typical acute inflammation. Sometimes an intramural abscess forms, and this may discharge into the lumen or through the muscle and serous coats. Perforation of the appendix may be due to the discharge of such an intramural abscess; it may follow distension and rupture of an obstructed organ, and sometimes it is due to occlusion of the appendicular vessels with resultant ischaemic necrosis. Vascular occlusion can also cause gangrene of a substantial part, or even the whole of the appendix.

The course of the disease, the macroscopic changes in the organ and the state of affairs at operation depend on several factors, including: (1) The virulence of the infection and resistance of the host; (2) the presence or absence of obstruction; (3) the position of the organ; and (4) the time elapsing before operation. If the organ is in a retrocaecal position, the dangers are less than if it lies free in the peritoneal cavity. In the latter position, perforation favours a rapidly spreading peritonitis, whereas this rarely occurs with a retrocaecal appendix. Proximity to a well-developed greater omentum and other viscera assists the localisation of infection. In this connection, it should be noted that the relatively undeveloped omentum of the child is not easily able to wrap itself around the inflamed organ. According to the balance of these various factors, perforation of the appendix will result in a localised abscess in one person, whereas in another there will be diffuse peritonitis. On occasions, a well-localised abscess, instead of resolving, steadily enlarges and ruptures into the general peritoneum. The combination of obstruction and less virulent infection can cause the appendix to distend with pus without perforating or, with a mild infection, merely to fill up with mucus. The former condition is called an *empyema*, and the latter a *mucocele* of the appendix.

**Clinical Features.** In typical cases, and these constitute about two thirds of those seen, there is the following sequence of symptoms: (1) Central abdominal pain; (2) anorexia, nausea or vomiting; (3) shift of pain to the right iliac fossa. The initial pain, visceral in origin, is colicky or continuous and situated at the umbilicus, but sometimes above or below this point. The patient often says that he feels full of wind. Usually, after a few hours, but sometimes after a day or two, the central pain is succeeded by pain and tenderness in the right iliac fossa due to irritation of the parietes by the inflamed organ. The iliac fossa pain is often aggravated by movement such as walking or the act of sitting up. In about a third of cases, the pain is situated in the right iliac fossa from the outset. *Vomiting* is an early and sometimes repeated feature of obstructive appendicitis. In the unobstructed type, however, the patient may vomit only once or simply experience slight anorexia or nausea. With a short history, there is commonly no change in the bowel habit, though *constipation* may be a feature after a day or two. *Diarrhoea* and *increased frequency* of micturition, due to direct irritation of bowel and



the ileocolic vessels; the artery and vein pass behind the terminal ileum to enter the delicate mesentery of the organ. Anterior to the base of the appendix, and frequently attached to it, is a fat-containing and sometimes vascular ileocaecal fold of peritoneum. This has been named (and certainly rather inappropriately in many cases) the bloodless fold of Treves. The position of the appendix is subject to variation. It may be situated below, behind, medial or lateral to the caecum, and its situation is sometimes a factor of some importance in determining the subsequent course of inflammation.

**Aetiology and Pathology.** Acute appendicitis is invariably due to

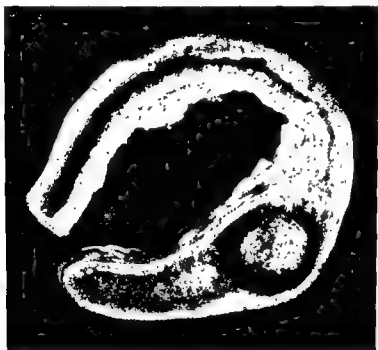


FIG. 1. Faecolith in the lumen of a thickened and inflamed appendix.

*bacterial infection.* Inflammation may be predisposed to by *obstruction* and the presence of abundant *lymphoid tissue*. This last factor may be of importance in determining the onset of inflammation in children and adolescents. Although inflammation is generally thought to be due to bowel organisms, it is possible that blood stream infection of the lymphoid tissue also occurs. Obstruction of the lumen may be due to kinks, fibrosis from previous inflammation, faecoliths, foreign bodies, hypertrophy of lymphoid tissue or oedema of the mucosa (Fig. 1). Inflammation starting in an unobstructed organ may cause swelling of the wall of the organ and consequent obstructive appendicitis. The conditions in the obstructed appendix are comparable to those in an infected closed-loop gut obstruction, and the results are nearly always more serious than in non-obstructive appendicitis. Whether the bacteria gain access through the crypts and

insidiously and the patient may present with *abdominal distension* suggestive of intestinal obstruction. In infants, inability to elicit a history makes diagnosis difficult, and if there is diarrhoea there is a risk of attributing the trouble to *gastro-enteritis*.

When the appendix is adherent to the terminal ileum, the symptoms may be like those of *intestinal obstruction*: indeed the inflammation here can produce a partial small gut obstruction. High pyrexia, rigors and mild jaundice, due to *pylephlebitis* occasionally occur without any preliminary features characteristic of appendicitis.

Quite apart from those patients who do not report the early stages of their illness, and then see the doctor for the first time with a well-established abscess, there are some who have quite slight initial symptoms preceding formation of a large abscess. Rarely an abscess tracks down the inguinal canal or into the thigh beneath the inguinal ligament. Sometimes a feverish illness, without any abdominal symptoms, is only diagnosed correctly following discovery of an abscess in right iliac fossa, pelvis or subphrenic region.

A problem of recent origin is the case of appendicitis, undiagnosed as such, and because of unexplained fever, treated for some time with a broad spectrum antibiotic. In these circumstances, perforation of the organ may be masked; thus a very dangerous state of affairs can develop without recognition.

There is certainly no other abdominal disease in which prompt diagnosis and treatment is so important. However, every general practitioner and surgeon of experience can recall cases he has failed to recognise until too late.

**Differential Diagnosis.** The initial central pain may be mistaken for *biliary* or *intestinal colic*. When there is severe epigastric pain at the onset, a *perforated peptic ulcer* may be suspected. In such cases, the demonstration of liver dullness, or a plain X-ray which shows no gas under the diaphragm, obviate this mistake. When the pain is right-sided from the onset, it may suggest *cholecystitis*, and even in cholecystitis there is sometimes central pain at the start with subsequent shift to the right. In such cases, especially if the gall bladder is low down, it is not uncommon for appendicitis to be wrongly diagnosed. *Pyelitis* can be confusing, but careful examination of the urine (including microscopy of a centrifuged deposit) is essential in doubtful cases. *Non-specific mesenteric lymphadenitis* is usually characterised by sharp bouts of short-lived pain and the patient is relatively comfortable in between. Inflammation of a *solitary diverticulum of the caecum* or a *Meckel's diverticulum* may exactly simulate appendicitis. In the female, *salpingitis*, *ruptured ectopic gestation*, *twisted ovarian cyst*, and *corpus luteum hæmorrhage* must be considered. The first of these can be difficult to exclude; the second is usually associated with a missed period and possibly with shock; the third and last diagnoses are suggested by local symptoms and signs without gastro-intestinal upset.

bladder, may occur with pelvic appendicitis or pelvic abscess formation. Diarrhoea occasionally occurs with a pre-ileal or paracolic inflamed appendix. The injudicious use of a purgative may exacerbate the infection and provoke perforation.

*Pyrexia* and *tachycardia* vary greatly according to the severity and duration of symptoms. Temperature above 102° F. is unusual; rigors occasionally occur, and these may be due to pylephlebitis or even to an uncomplicated obstructive appendicitis. *The tongue* tends to be furred but moist. A clean tongue may be seen in early cases and with milder forms of inflammation. *Tenderness* is maximal over the inflamed appendix, and varies both in position and intensity according to the position of the organ and its relationship to surrounding viscera. It is most often in the region of *McBurney's point* (on the line between the umbilicus and right anterior superior iliac spine, and 1½ to 2 inches from the latter). Tenderness may be more lateral and less marked in retrocaecal appendicitis, or only detected on rectal examination if the appendix is in the pelvis. It is unnecessary to catalogue the possible points of maximum tenderness if the varying positions of the cecum and appendix are kept in mind. *Cutaneous hyperaesthesia* is a sign which does not warrant the attention given it in some accounts of this disease. Rebound pain is sometimes elicited on releasing pressure over the cecum. *Rigidity* of the parietal muscles is related to the site of the appendix. Thus, if the appendix is subcaecal in position, rigidity may be marked, but it is absent in pelvic appendicitis. In the retrocaecal variety, rigidity may be minimal or only located well out towards the flank. The absence of both rigidity and tenderness shortly after perforation may deceive the doctor who first sees the case at this stage. An *abdominal mass* may be due to several causes. In the first forty-eight hours, a palpable lump is most likely to be omentum adherent to the appendix. Later on in the course of the illness, a lump is usually due to abscess formation, but even after several days a mass may simply consist of the appendix and adjacent adherent viscera. Occasionally, especially in a thin patient, the appendix itself is felt as a tender swelling.

**Aids to Diagnosis.** A careful history and thorough physical examination give adequate information in most cases. One must beware of the white cell count, for a leucocytosis may be found with a normal appendix, whereas a normal white cell count may lead to wrongful exclusion of genuine appendicitis. The white cell count may mislead even an experienced clinician, and is probably best ignored. A plain X-ray of the abdomen is occasionally helpful. Thus, a small gas bubble and fluid level in an abscess cavity, or a "sentinel" loop of terminal ileum, adherent to the appendix, may help to establish an otherwise doubtful diagnosis.

**Unusual Types of Appendicitis.** Although appendicitis is uncommon at the extremes of life, in the very young and in the aged the diagnosis can be especially difficult. In old folk, the disease sometimes develops most

incidentally and the patient may present with *abdominal distension* suggestive of intestinal obstruction. In infants, inability to elicit a history makes diagnosis difficult, and if there is diarrhoea there is a risk of attributing the trouble to *gastro-enteritis*.

When the appendix is adherent to the terminal ileum, the symptoms may be like those of *intestinal obstruction*; indeed the inflammation here can produce a partial small gut obstruction. High pyrexia, rigors and mild jaundice, due to *pylephlebitis* occasionally occur without any preliminary features characteristic of appendicitis.

Quite apart from those patients who do not report the early stages of their illness, and then see the doctor for the first time with a well-established abscess, there are some who have quite slight initial symptoms preceding formation of a large abscess. Rarely an abscess tracks down the inguinal canal or into the thigh beneath the inguinal ligament. Sometimes a feverish illness, without any abdominal symptoms, is only diagnosed correctly following discovery of an abscess in right iliac fossa, pelvis or subphrenic region.

A problem of recent origin is the case of appendicitis, undiagnosed as such, and because of unexplained fever, treated for some time with a broad spectrum antibiotic. In these circumstances, perforation of the organ may be masked; thus a very dangerous state of affairs can develop without recognition.

There is certainly no other abdominal disease in which prompt diagnosis and treatment is so important. However, every general practitioner and surgeon of experience can recall cases he has failed to recognise until too late.

**Differential Diagnosis.** The initial central pain may be mistaken for *biliary* or *intestinal colic*. When there is severe epigastric pain at the onset, a *perforated peptic ulcer* may be suspected. In such cases, the demonstration of liver dullness, or a plain X-ray which shows no gas under the diaphragm, obviate this mistake. When the pain is right-sided from the onset, it may suggest *cholecystitis*, and even in *cholecystitis* there is sometimes central pain at the start with subsequent shift to the right. In such cases, especially if the gall bladder is low down, it is not uncommon for appendicitis to be wrongly diagnosed. *Pyelitis* can be confusing, but careful examination of the urine (including microscopy of a centrifuged deposit) is essential in doubtful cases. *Non-specific mesenteric lymphadenitis* is usually characterised by sharp bouts of short-lived pain and the patient is relatively comfortable in between. Inflammation of a *solitary diverticulum of the caecum* or a *Meckel's diverticulum* may exactly simulate appendicitis. In the female, *salpingitis*, *ruptured ectopic gestation*, *twisted ovarian cyst*, and *corpus luteum hæmorrhage* must be considered. The first of these can be difficult to exclude; the second is usually associated with a missed period and possibly with shock; the third and last diagnoses are suggested by local symptoms and signs without gastro-intestinal upset.

bladder, may occur with pelvic appendicitis or pelvic abscess formation. Diarrhoea occasionally occurs with a pre-ileal or paracolic inflamed appendix. The injudicious use of a purgative may exacerbate the infection and provoke perforation.

*Pyrexia* and *tachycardia* vary greatly according to the severity and duration of symptoms. Temperature above 102° F. is unusual; rigors occasionally occur, and these may be due to pylephlebitis or even to an uncomplicated obstructive appendicitis. *The tongue* tends to be furred but moist. A clean tongue may be seen in early cases and with milder forms of inflammation. *Tenderness* is maximal over the inflamed appendix, and varies both in position and intensity according to the position of the organ and its relationship to surrounding viscera. It is most often in the region of *McBurney's point* (on the line between the umbilicus and right anterior superior iliac spine, and 1½ to 2 inches from the latter). Tenderness may be more lateral and less marked in retrocaecal appendicitis, or only detected on rectal examination if the appendix is in the pelvis. It is unnecessary to catalogue the possible points of maximum tenderness if the varying positions of the caecum and appendix are kept in mind. *Cutaneous hyperaesthesia* is a sign which does not warrant the attention given it in some accounts of this disease. Rebound pain is sometimes elicited on releasing pressure over the caecum. *Rigidity* of the parietal muscles is related to the site of the appendix. Thus, if the appendix is subcaecal in position, rigidity may be marked, but it is absent in pelvic appendicitis. In the retrocaecal variety, rigidity may be minimal or only located well out towards the flank. The absence of both rigidity and tenderness shortly after perforation may deceive the doctor who first sees the case at this stage. An *abdominal mass* may be due to several causes. In the first forty-eight hours, a palpable lump is most likely to be omentum adherent to the appendix. Later on in the course of the illness, a lump is usually due to abscess formation, but even after several days a mass may simply consist of the appendix and adjacent adherent viscera. Occasionally, especially in a thin patient, the appendix itself is felt as a tender swelling.

**Aids to Diagnosis.** A careful history and thorough physical examination give adequate information in most cases. One must beware of the white cell count, for a leucocytosis may be found with a normal appendix, whereas a normal white cell count may lead to wrongful exclusion of genuine appendicitis. The white cell count may mislead even an experienced clinician, and is probably best ignored. A plain X-ray of the abdomen is occasionally helpful. Thus, a small gas bubble and fluid level in an abscess cavity, or a "sentinel" loop of terminal ileum, adherent to the appendix, may help to establish an otherwise doubtful diagnosis.

**Unusual Types of Appendicitis.** Although appendicitis is uncommon at the extremes of life, in the very young and in the aged the diagnosis can be especially difficult. In old folk, the disease sometimes develops most

insidiously and the patient may present with *abdominal distension* suggestive of intestinal obstruction. In infants, inability to elicit a history makes diagnosis difficult, and if there is diarrhoea there is a risk of attributing the trouble to *gastro-enteritis*.

When the appendix is adherent to the terminal ileum, the symptoms may be like those of *intestinal obstruction*: indeed the inflammation here can produce a partial small gut obstruction. High pyrexia, rigors and mild jaundice, due to *pylephlebitis* occasionally occur without any preliminary features characteristic of appendicitis.

Quite apart from those patients who do not report the early stages of their illness, and then see the doctor for the first time with a well-established abscess, there are some who have quite slight initial symptoms preceding formation of a large abscess. Rarely an abscess tracks down the inguinal canal or into the thigh beneath the inguinal ligament. Sometimes a feverish illness, without any abdominal symptoms, is only diagnosed correctly following discovery of an abscess in right iliac fossa, pelvis or subphrenic region.

A problem of recent origin is the case of appendicitis, undiagnosed as such, and because of unexplained fever, treated for some time with a broad spectrum antibiotic. In these circumstances, perforation of the organ may be masked; thus a very dangerous state of affairs can develop without recognition.

There is certainly no other abdominal disease in which prompt diagnosis and treatment is so important. However, every general practitioner and surgeon of experience can recall cases he has failed to recognise until too late.

**Differential Diagnosis.** The initial central pain may be mistaken for *biliary* or *intestinal colic*. When there is severe epigastric pain at the onset, a *perforated peptic ulcer* may be suspected. In such cases, the demonstration of liver dullness, or a plain X-ray which shows no gas under the diaphragm, obviate this mistake. When the pain is right-sided from the onset, it may suggest *cholecystitis*, and even in cholecystitis there is sometimes central pain at the start with subsequent shift to the right. In such cases, especially if the gall bladder is low down, it is not uncommon for appendicitis to be wrongly diagnosed. *Pyelitis* can be confusing, but careful examination of the urine (including microscopy of a centrifuged deposit) is essential in doubtful cases. *Non-specific mesenteric lymphadenitis* is usually characterised by sharp bouts of short-lived pain and the patient is relatively comfortable in between. Inflammation of a *solitary diverticulum of the cæcum* or a *Meckel's diverticulum* may exactly simulate appendicitis. In the female, *salpingitis*, *ruptured ectopic gestation*, *twisted ovarian cyst*, and *corpus luteum hæmorrhage* must be considered. The first of these can be difficult to exclude; the second is usually associated with a missed period and possibly with shock; the third and last diagnoses are suggested by local symptoms and signs without gastro-intestinal upset.

An appendix mass must be differentiated from other *abscesses and tumours* in the right iliac fossa and from *empyema of the gall bladder*. Sometimes *carcinoma of the cæcum* or ascending colon causes symptoms like those of appendicitis and a palpable mass may be due to the tumour or an associated abscess.

Finally, a group of conditions must be considered in which operation is unnecessary or positively contraindicated. Right basal *pneumonia* or *pleurisy*, especially in a child, may cause abdominal pain and vomiting. Physical signs in the chest are sometimes not apparent for some time after the onset, but an unusually high temperature, and absence of strictly localised abdominal signs may warn the astute clinician. The acute specific fevers, such as *measles* and *chickenpox*, may cause abdominal pain in the early stages, and even the pain of *poliomyelitis* has sometimes been mistaken for appendicitis. Rarely such diseases as *osteomyelitis* of the pelvis, iliac *phlebitis* and *lymphadenitis* are diagnosed as appendicitis, and the very infrequency of such cases is liable to deceive even an experienced clinician.

**Treatment.** Within forty-eight hours of the onset, there is universal agreement that a diagnosis of acute appendicitis demands immediate operation. When the attack is clearly a mild one of the non-obstructed variety, and if it appears to be subsiding when the patient is first seen, the doctor may be persuaded to temporise. In such cases, operation is sometimes deferred to fit in with domestic and other circumstances. However, the greatest circumspection is necessary in making any such decision, as a flare-up occasionally occurs. In truth, the majority of patients so treated are those with mild symptoms and in whom there is doubt about the diagnosis.

It is in the longer-established cases of appendicitis that there is sometimes less agreement about the best method of treatment; yet, even here, there is probably less controversy than was the case twenty or so years ago. Indiscriminate appendicectomy in patients with advanced disease and abscess formation carries a mortality of 10 per cent. or more, and it was experience of this kind that formerly led many to advise against operation after forty-eight hours had elapsed. However, the expectant treatment of appendicitis, if applied on a strictly time interval basis, will lead to quite unjustifiable deferment of operation in many cases. Moreover, a palpable mass is by no means always an abscess, and it may be impossible to say what exactly is going on in such an abdomen. In cases of this type, if an incision is made over the swelling, it is generally possible on reaching the extraperitoneal tissues to see if an abscess is present. In these circumstances, it is feasible to drain many abscesses by an extraperitoneal approach. Unless the appendix is readily accessible within the abscess, no attempt is made to remove it. When it is necessary to transgress the peritoneal cavity to drain such an abscess, the greatest care is taken to avoid spreading contamination. It is the bad results of mischievous and

unnecessarily aggressive attacks on the appendix itself which have given operation an evil reputation in this type of case. Drainage of an abscess relieves pain and fever, and rapidly improves the general condition of the patient.

*The case with apparently resolving mass.* Sometimes the patient presents seven to fourteen days after the onset of the illness with a well-localised abscess and apparently improving health. Providing the patient is admitted to hospital where detailed observation is possible, it is perfectly justifiable to treat such a patient conservatively, and in most cases the abscess resolves. In these cases, as well as those patients who have had simple drainage of an abscess, appendicectomy is performed later. This

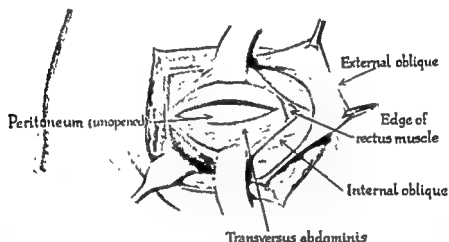


FIG. 2. Gridiron incision. To show exposure of the various layers of the abdominal wall. The skin incision is commonly more transverse than that shown.

*interval appendicectomy* is usually done three months after the initial attack, but occasionally a further attack occurs during this period.

*The case with diffuse peritonitis.* Patients who present with diffuse peritonitis from a perforated or gangrenous organ seldom need any prolonged preoperative treatment. However, it is important not to operate upon them too precipitately, and wise to pass a suction tube and aspirate the stomach before an anæsthetic is given. An hour or two spent in correcting dehydration and electrolyte loss and starting antibiotic treatment may be of value. Preliminary treatment of this sort is seldom wasted and this is especially true in infants and young children.

**Appendicectomy.** There is no one incision for appendicectomy, and certainly no point in laying down the law about one particular method. Most surgeons prefer a skin crease or oblique incision centred over the point of maximum tenderness. In the *McBurney (gridiron) incision*, each muscle is *split* in the line of its fibres, and improved access can be secured by opening the edge of the rectus sheath medially, or by *cutting* the muscles



laterally (Fig. 2). The inexperienced operator is sometimes handicapped by this incision, and in these circumstances, or with a patient of fat and heavy build, there is much to be said for *cutting* through all the muscles and peritoneum in the line of the skin incision. This *Rutherford Morison incision* is a sound and practical approach. A paramedian incision is sometimes indicated when there is doubt about the diagnosis, and a pelvic or upper abdominal condition may have to be dealt with.

After the peritoneum is opened, the abdominal wall is gently lifted with a retractor. The iliac fossa is inspected, and if the appendix is not

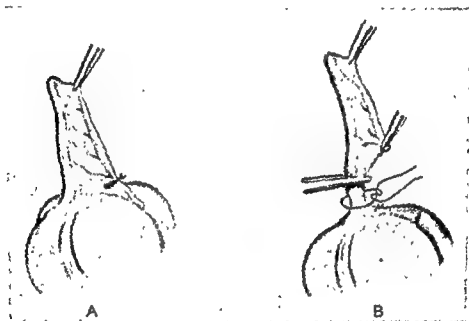


FIG. 3. : Appendicectomy. A. Ligation of appendicular vessels. B. Ligation of crushed appendix base.

visualised it is brought into view by tracing down the anterior tænia of the caecum. The organ is then mobilised gently and the greatest care is taken to avoid damage to its mesentery as well as any structures to which it may be adherent. The vessels in the mesentery of the appendix are carefully secured and ligated (Fig. 3); the base of the appendix is crushed, tied, and, following division of the ligature, inverted into the caecum with a Z-stitch or purse-string suture (Fig. 4). The last step is occasionally impossible due to the thickened, turgid state of the tissues. Drainage of the peritoneum is only necessary when a thick-walled abscess is encountered or there is a good deal of raw surface. A small collection of pus adjacent to the appendix does not necessitate the insertion of a drain.

**Ancillary Care.** Reference has already been made to the occasional need for gastric aspiration and intravenous transfusion before operation, and this needs to be continued in the more severe cases. Antibiotic

therapy is indicated when : (1) There is diffuse peritonitis ; (2) an abscess has to be drained across the peritoneal cavity ; (3) there is a gangrenous and foully-infected organ the removal of which is difficult and necessitates undue manipulation and contamination of the abdomen. In uncomplicated cases, there is no need for antibiotics, and there is no indication for them when a localised abscess is drained without disturbing the peritoneal cavity.

As to the choice of antibiotic and chemotherapeutic agent, there is much to be said for penicillin and sulphadimidine (500,000 units and 1.5 gm. respectively six-hourly). Although streptomycin tends to be used by some surgeons, the rapidity with which strain resistance develops is a

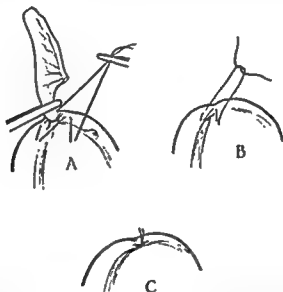


FIG. 4. Appendicectomy. A. Ligature tied on appendix base and Z-stitch inserted. B. Z-stitch being tied. C. Stump of appendix completely buried.

strong argument against its use. In grave peritonitis, and when the response to penicillin and sulpha-combinations is deemed unsatisfactory, terramycin (oxytetracycline) 0.5 gm., six hourly is probably best. See also *Antibiotic Therapy*, Chap. 110.

**Complications.** The commonest complication following appendicectomy is *infection of the wound* and *abscess* formation in the abdominal wall. A *sinus* may form due to persistent infection or retained foreign material. An *intraperitoneal abscess* may form in the right iliac fossa, pelvis, subphrenic region, or between coils of small gut. *Faecal fistula* is uncommon and generally due to damage to the cæcum or terminal ileum at the time of operation. It is occasionally due to an ill-placed drain. Sometimes such a fistula is due to *Crohn's disease* or *actinomycosis* which has not been recognised at the time of operation. *Paralytic ileus* is not uncommon, and sometimes there is *mechanical ileus* due to adherent coils of small gut in

the region of the appendix base or wall of an abscess cavity. *Deep venous thrombosis* and *pulmonary embolism* occasionally occur but are fortunately relatively uncommon in young adults and exceptional in childhood. *Pulmonary collapse* may also occur and is sometimes complicated by *pneumonitis*. *Portal pyæmia* is uncommon and may be difficult to distinguish from subphrenic abscess. Remote complications are *incisional hernia* (especially when there has been infection of the wound), and *intestinal obstruction* due to adhesions.

### SUBACUTE RECURRENT APPENDICITIS

Recurrent attacks of mild appendicitis may occur and sometimes they are succeeded by a typical acute attack. Subacute recurrent appendicitis is a reasonable title for this type of case in which the organ shows evidence of recent or resolving inflammation. The symptoms may be those of a mild acute attack, or there may be recurrent central abdominal or right iliac fossa pain. It is, however, unquestionable that many patients presenting with recurrent pain in the right iliac fossa do not have appendicitis, and frequently they are not suffering from any other condition which necessitates operation. Such a patient is commonly a young woman, and this type of trouble may engender some anxiety on the part of the patient and her relatives. The family doctor may need to exercise considerable skill in the management of such patients; on the one hand he must avoid stampeding the patient into precipitate and unnecessary operation, and on the other hand he must beware of resisting the suggestion of operation when it is genuinely desirable. Such difficulties are often best resolved in the following fashion: (1) If there is reasonable possibility of the appendix being at fault, or likelihood of some other lesion requiring surgery (e.g. an ovarian cyst), then the position should be carefully explained and surgery recommended; (2) if there is doubt about the diagnosis of appendicitis and no reasonable ground for suspecting any other surgical disorder, then the patient may be advised that it is preferable to run a no more than average risk of subsequent appendicitis rather than face possible continuing symptoms after an unnecessary operation; (3) in some cases, the doctor feels pretty sure that the appendix is not diseased, but there is persistent anxiety in an otherwise reasonable and balanced individual. If, after carefully weighing up the physical and emotional factors, it is felt that operation is justifiable, then *full exploration* is recommended. "Key-hole" surgery through a small incision is most unwise since it is vitally important that the surgeon shall be able to satisfy the patient and himself that nothing has been overlooked.

In many of these cases, it is important to exclude renal, ovarian, biliary, duodenal, and spinal sources of pain. A barium follow-through examination of the appendix region is of more value in refuting than in confirming suspicions about a pathological appendix. For example, it may be possible

to demonstrate that the site of pain and tenderness in the right iliac fossa is nowhere near the position of the appendix. On the other hand, incomplete filling of the appendix and even the demonstration of tenderness over it should be interpreted with reserve.

### CHRONIC APPENDICITIS

Some have denied that true chronic appendicitis ever occurs. However, histological examination of the organ certainly justifies the pathological diagnosis in a number of cases. These patients generally exhibit similar symptoms to those already considered under subacute recurrent appendicitis, and the pathologist sometimes reports mild acute-on-chronic inflammatory changes. The term chronic appendicitis should not be used in describing those cases in which there is fibrous obliteration of the appendix. Such fibrosis may be the result of previous inflammation, or be due to atrophy in the aged. Some patients with recurring appendicular symptoms show fibrous obliteration of the distal part and chronic or even acute inflammatory changes in the proximal half.

### TUMOURS OF THE APPENDIX

**Adenocarcinoma.** This is rare in the appendix despite its frequent occurrence elsewhere in the large bowel. In appearance it sometimes simulates mucocoele of the appendix but it does not warrant fuller description here.

**Carcinoid.** This is the commonest tumour of the appendix and is seen in about 1 in 250 organs removed at operation. Usually the tumour is an incidental finding and is situated in the submucosa at the tip. Occasionally other carcinoids are found in the appendix or small gut. The cut surface of such a tumour is generally yellow due to the high lipid content of the cells. The latter are mainly spheroidal in shape and arranged in tightly packed sheets or groups. These tumours arise from the Kulschitzky cells in the crypts of Lieberkuhn, and as the cells react with silver salts they are sometimes called argentaffinomas. Carcinoids of the appendix are locally invasive; but, unlike their counterparts in the small gut, they rarely metastasise.

### MISCELLANEOUS CONDITIONS

**Foreign Bodies and Parasites.** Various foreign bodies such as seeds and lead shot may be found in the appendix, but they are usually of no significance. Fæcoliths of varying consistency are also seen, and, if hard, these may contribute to obstruction in the event of appendicitis. Oxyuris vermicularis infestation is a common incidental finding in the appendices of young children and sometimes in adults, but other types of worm are rarely found.

**Mucocele of the Appendix.** This condition has been briefly mentioned in connection with appendicitis and is due to a combination of obstruction and low-grade infection (Fig. 5). Uncommonly such a mucocele ruptures and gives rise to the condition of *pseudomyxoma peritonei* in which there

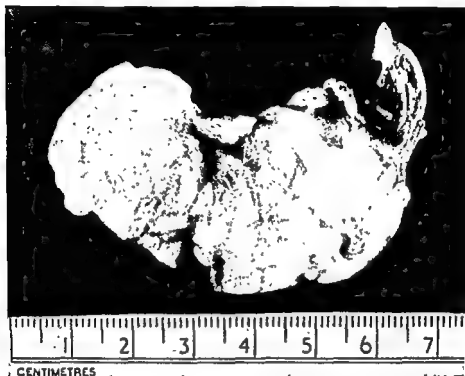


FIG. 5. Mucocele of appendix. Base of organ to right.

are gelatinous masses scattered over the peritoneum. It should be remembered that the latter condition can, on rare occasions, be secondary to adenocarcinoma of the appendix. Occasionally a normal appendix distends with mucus as a result of obstruction of its proximal end by a tumour of the caecum. *Endometriosis* occasionally involves the serous coat of the appendix and is usually secondary to this disease in the ovary or pelvis. *Secondary* neoplasms are rare occurrences. Intussusception of the appendix itself is rare but it commonly takes part in ileocaecal intussusception.

## CHAPTER 2

### THE STOMACH AND DUODENUM

#### Tumours of the Stomach

##### CARCINOMA

CARCINOMA is by far the most important and the commonest type of gastric tumour. In Britain close on 15,000 people die each year of this disease. Its apparently increasing incidence during the last fifty years is largely a reflection of the ageing population, for when expressed as a proportion of all cancer deaths, the percentage incidence has actually fallen a little in the last decade. This is in contrast to the striking increase in the number of deaths from bronchial carcinoma during the same period.

**Aetiology.** Carcinoma of the stomach is roughly twice as common in men as in women. Less than 5 per cent. of such tumours occur in patients under forty-five years of age and more than a third of all cases are diagnosed during the seventh decade. Very little indeed is known of the cause of gastric cancer. Dietary factors may be of importance, but their precise significance has escaped detection. In the past there was much argument about the possible relationship between peptic ulcer and cancer, but there can be little doubt that very few gastric ulcers ever undergo malignant change. Pre-existing achlorhydria is a definitely established aetiological factor, and there is no doubt that patients suffering from pernicious anaemia, in which achlorhydria is a constant finding, are more prone to gastric cancer. The actual incidence in such patients is about three times as high as in an unselected population of similar age group, and the longer established the pernicious anaemia, the greater is the chance of cancer. Simple tumours of the stomach only rarely undergo carcinomatous change.

**Pathology.** The commonest site of gastric carcinoma is the pyloric region in which some two-thirds of these tumours are situated. Roughly a quarter occur in the mid-gastric region, and about 5 per cent. at the cardiac end. In about 10 per cent. of all cases, the growth is a diffusely infiltrating one; this type is definitely more common in young patients. There are three main types of tumour: (1) Ulcerating; (2) polypoid; (3) infiltrating. The first type is the most common. In contrast to innocent peptic ulcer, the muscle coat is seldom completely destroyed by a malignant ulcer. Polypoid growths are less common, and their surface tends to become necrotic and ulcerated. The infiltrating type can spread extensively in the submucous and subserous layers, sometimes converting the stomach into a rigid, indistensible tube (Fig. 6). Growths

near the upper end of the stomach spread fairly readily into the œsophagus, but infiltration of the duodenum is less often seen.

**Microscopic Appearance.** The majority of these tumours are adenocarcinomas with a variable degree of differentiation. Sometimes there are quite different appearances in separate parts of the same tumour. In some there is a well-marked glandular arrangement, but often there is a more anaplastic cellular arrangement, especially with infiltrating growths. In the latter cases, there is sometimes considerable scirrhous reaction and it may be difficult to identify the tumour cells. Both intra- and extra-cellular mucin production may be seen. Squamous carcinoma of the

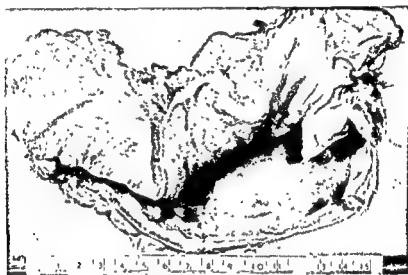


FIG. 6. Carcinoma of stomach. Extensive infiltration of proximal two-thirds of stomach. The name *linitis plastica* is sometimes applied to this type of neoplasm.

stomach is rare and, when situated near the fundus, it may be difficult to exclude a primarily œsophageal growth.

Apart from direct infiltration of the stomach, these growths spread by four principal routes: (1) The lymphatics; (2) direct involvement of neighbouring organs; (3) the peritoneum; (4) the blood stream. Lymphatic spread is initially to the juxta-gastric and intermediate glands in the omenta, thence to those related to the main vessels, and occasionally to the thoracic duct (Fig. 7). The actual route and extent of such spread is related to the site and duration of the primary growth as well as its inherent aggressiveness. With pyloric tumours, spread to the upper gastric, juxta-cardiac and gastrosplenic nodes is less common than when the tumour starts in the mid-gastric or cardiac region. Direct spread to neighbouring organs such as the pancreas, omentum, transverse mesocolon and liver is often seen. Transperitoneal spread is a prominent feature of some cases. Deposits are commonly seen in the pelvis where they may form a shelf of malignant tissue, or involve the ovaries. Hæmatogenous spread of gastric cancer to lungs, brain and skeleton is relatively late.

**Clinical Features.** It cannot be too strongly emphasised that there are no distinctive features of early gastric cancer. Eternal vigilance when investigating dyspepsia of recent origin, especially after the age of forty-five, will reduce some of the delays in diagnosis; but it is sad to record how many tumours are already advanced and inoperable before symptoms occur. The early symptoms of gastric carcinoma (and these are by no means synonymous with early disease) are often vague and indefinite. Therefore, it is not unusual for some time to elapse between the onset of symptoms and the first visit to a doctor.

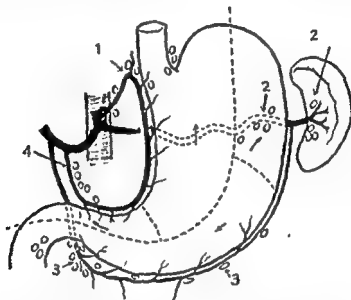


FIG. 7. Lymphatic drainage of stomach. Showing various groups of glands and approximate territory drained by them: (1) Superior cardiac. (2) Gastro-splenic and pancreatic. (3) Sub-pyloric and inferior gastric. (4) Supra-pyloric.

In four out of five cases the first symptoms are gastro-intestinal in character. Constitutional upset is the primary feature in most of the remaining cases, although a small percentage present first with secondary deposits outside the abdomen. In general, tumours at the pyloric end of the stomach tend to be easier to diagnose than those elsewhere, whereas the greatest difficulty is usually experienced with those near the cardia.

The commonest symptom is some form of "indigestion," variously described as a feeling of fullness, wind, or an upper abdominal aching pain which is either continuous or related to meals. Although a distinctive food/pain relationship is not so common as in peptic ulcer, this does occur in about 25 per cent. of cases, and then there is often relief with alkalis. Occasionally there is remission of symptoms as in benign peptic ulcer. Dysphagia may be the first symptom of a growth at the cardia, and sometimes anorexia or the appearance of an abdominal tumour is the initial feature.



A proportion of patients present with anæmia, weakness and lassitude as the only symptoms. Hæmatemesis and melæna occur in about 20 per cent. of cases, although the blood loss is rarely so severe as in cases of benign ulcer. Although perforation of gastric carcinoma is not common, about 2 to 3 per cent. of all gastric perforations are probably due to neoplasm, but the true nature of the disease is frequently unrecognised at the time of perforation.

Although it is desirable to establish the diagnosis as early as possible, it should be emphasised that those patients with a short history prior to operation, often have an unfavourable type of disease, and those with a long history frequently have slowly growing and more favourable tumours. This paradox of a better prognosis in patients with long-standing symptoms is, of course, common to cancer at many sites.

**Physical Examination.** In some 50 per cent. of cases there are no notable physical signs when the patient first reports, but in many of the remainder an abdominal mass is present. A palpable tumour is generally epigastric but sometimes lower down. Loss of weight is only evident in those patients whose food intake has been restricted, or in those with widespread disease. Anæmia is common with ulcerated tumours but infrequent with diffusely infiltrating tumours. Occasionally the patient first appears with a secondary deposit at the umbilicus; ascites; jaundice; or other features of disseminated disease. The presence of a palpable supraclavicular node (Virchow's gland) occasionally draws attention to a gastric neoplasm. However, it should be remembered that metastasis to the neck is not uncommon in other types of malignant disease (e.g. bronchial carcinoma).

**Investigations.** *Barium Meal.*—This is the most useful investigation (Fig. 8). In about 85 per cent. of hospital cases an abnormality is detected at the first examination, and a further 5 per cent. are suspected on repeat examination. A negative X-ray in these cases is often followed by a delay of many months before the diagnosis is finally established. The barium meal should, therefore, be repeated at an early date in all cases where there is reasonable clinical suspicion. Lesions at the cardia are the most difficult to diagnose. Infiltrating tumours, which neither form a mass nor ulcerate deeply, are also not so easily diagnosed on X-ray. The radiologist may also have particular difficulty in those patients with large gastric rugæ. *Gastroscopy.*—This examination is an aid to diagnosis in some cases, and it yields a higher success rate in the hands of a skilled and experienced gastroscopist. Gastroscopic biopsy of the mucosa has been tried but is not widely used. *Fractional Test Meal.*—About a quarter of these patients have normal acidity or even hyperchlorhydria. Achlorhydria is present in rather more than 50 per cent. and hypochlorhydria in the remainder. But as achlorhydria or hypochlorhydria is present in about 25 per cent. of cases of gastric ulcer, the fractional test meal is of little value in differential diagnosis. Occasionally, with a juxta-pyloric lesion, when

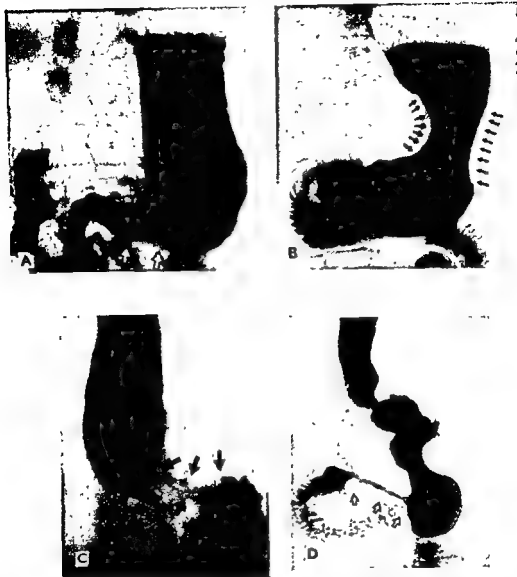


FIG. 8. Carcinoma of stomach. Barium meal films showing growths of various parts. A. Distal stomach with narrowing and rigidity of pyloric antrum. B. Middle third. C. Cardiac end with dilatation of oesophagus. D. Antral growth with reflux of barium into oesophagus as result of obstruction. (Courtesy of Dr. L. S. Carstairs.)

the radiologist is in doubt as to whether an ulcer is pre- or post-pyloric, the discovery of achlorhydria is of real value in drawing attention to the possibility of carcinoma rather than duodenal ulcer. *Cytological Examination.*—Malignant cells can sometimes be identified in the gastric contents after aspiration. Even with considerable experience of the method only a small proportion of growths can be diagnosed by the pathologist in this way. *Occult Blood.*—The chief value of the occult blood test is in focussing attention on the alimentary tract of a patient who has anæmia but no

obvious digestive disorder. Occult blood is present in the stools of some 90 per cent. of patients with gastric carcinoma. *Macrocytic anaemia* occurs in some patients with diffuse carcinoma of the stomach.

**Differential Diagnosis.** The most important condition in differential diagnosis is benign gastric ulcer. However, even a duodenal ulcer mass, due to marked oedema around the crater of a penetrating ulcer, may deform the pyloric end of the stomach and simulate a gastric carcinoma on X-ray. Notwithstanding certain "typical" cases, little reliance can be placed on the history. In the absence of a palpable tumour, a definite diagnosis is usually only established by X-ray or gastroscopy. Differentiation from other less common types of gastric tumour, such as benign lesions or sarcoma, is sometimes impossible. Similarly, those infrequent cases of benign hypertrophic pyloric stenosis in the adult are often indistinguishable from carcinoma before operation. An important group of patients are those presenting with anaemia and symptoms related thereto. A patient with unexplained secondary anaemia should be carefully investigated to exclude carcinoma of the stomach or right side of the colon, as growths at both sites are liable to present in this way. Occasionally a carcinoma gives rise to secondary deposits without it being possible to identify the primary tumour.

**Treatment.** Surgery offers the only hope of cure, but few would deny that the results of operation are somewhat dispiriting. Experience naturally varies in different places, but in most general hospitals, from 25 to 30 per cent. of all cases are clearly inoperable when first seen. In the remainder resection of the growth is done in about one in four to one in six cases. The mortality varies with the actual procedure used as well as the age and general condition of the patient. The operative mortality is in the region of 20 to 25 per cent. for any hospital receiving its patients from a local community. Better mortality figures have been reported with some of the selected material admitted to some hospitals.

Before embarking on exploration of the abdomen it is important to assess carefully the general health, and to relieve anaemia, hypoproteinaemia, avitaminosis and dehydration. Gastric lavage is often valuable in cleansing the stomach before operation and should not be neglected.

**A. Resectable Growths.** These can be divided into the possibly curable, with apparently localised disease, and those cases in which resection is justified although extragastric spread has occurred.

(i) *The "Curable" Growths.* Tumours at the pyloric end are dealt with by partial gastrectomy, but the operation is more extensive than that performed for peptic ulcer. Tumours of the mid-gastric region, and those of the fundus which do not encroach on the cardia, can be dealt with by abdominal total gastrectomy. Tumours at the fundus which involve the cardia and lower end of the oesophagus necessitate an abdominothoracic oesophageogastrectomy. In all these operations, the greater and lesser omentum, the first 1½ inches of the duodenum, and generally the spleen

and gastrosplenic omentum, are resected with the stomach. In some cases, it is necessary to remove part of the pancreas or transverse colon, or the left suprarenal, but the surgeon must beware undue enthusiasm in his resection. Although heroic surgery does occasionally pay dividends, this is a type of cancer in which the largest number of patients will derive most benefit in the hands of a surgeon who is reasonably conservative in the majority of cases. The quality of life counts for more than quantity, and furthermore fatal super-radical surgery is not to be regarded as a justifiable form of euthanasia.

(ii) *Incurable Disease with Resectable Growth.* In some cases, there is distant spread (for example, to the liver) yet resection of the primary growth is both feasible and desirable. A large ulcerating tumour with obstruction and anaemia will, unless the disease is rapidly progressive and very extensive, often justify palliative partial gastrectomy. A palliative total gastrectomy is less often justifiable. In the presence of limited peritoneal metastasis, with or without hepatic deposits; involvement of the para-aortic nodes or the diaphragm, gastrectomy may still be justified. Very occasionally, a localised hepatic deposit can be removed at the time of gastrectomy, but only the veridicious optimist would claim that such cases should be included in the apparently curable class. In all these cases, experience and mature judgement are called for in deciding what will best serve the true interests of the patient. In general, it is the sufferer with a relatively long history and fair general condition who is most likely to benefit from palliative resection.

**B. Irremovable Growths.** Inoperability, so far as the stomach itself is concerned, may be relative or absolute. The former cases include those with widespread extra-gastric disease in which gastrectomy, though still practicable, is considered undesirable. Absolute inoperability is typified by completely fixed and irremovable growths, but even in some of these cases, it is necessary to relieve obstruction by some form of short-circuit. If sufficient healthy stomach is available, an anterior gastrojejunostomy can be done, and an irremovable tumour at the cardia can be by-passed by *oesophageojejunostomy*.

**Results.** It is difficult to give a comprehensive assessment of results. Of every hundred cases attending a general hospital about thirty are inoperable when first seen. Of the seventy or so explored, resection may be possible in no more than twenty, and the resection may have been frankly palliative in half of these. About five of these twenty patients die in hospital, and no more than five of the survivors will be alive five years later. When the lymph glands are free from growth, the 5-year-survival rate is between 50 and 70 per cent. When the glands are involved, the 5-year-survival rate is between 10 and 15 per cent. These figures roughly epitomise the sorry tale of gastric carcinoma and, notwithstanding the many technical advances of recent years, the results are still most disappointing.

obvious digestive disorder. Occult blood is present in the stools of some 90 per cent. of patients with gastric carcinoma. *Macrocytic anaemia* occurs in some patients with diffuse carcinoma of the stomach.

**Differential Diagnosis.** The most important condition in differential diagnosis is benign gastric ulcer. However, even a duodenal ulcer mass, due to marked oedema around the crater of a penetrating ulcer, may deform the pyloric end of the stomach and simulate a gastric carcinoma on X-ray. Notwithstanding certain "typical" cases, little reliance can be placed on the history. In the absence of a palpable tumour, a definite diagnosis is usually only established by X-ray or gastroscopy. Differentiation from other less common types of gastric tumour, such as benign lesions or sarcoma, is sometimes impossible. Similarly, those infrequent cases of benign hypertrophic pyloric stenosis in the adult are often indistinguishable from carcinoma before operation. An important group of patients are those presenting with anaemia and symptoms related thereto. A patient with unexplained secondary anaemia should be carefully investigated to exclude carcinoma of the stomach or right side of the colon, as growths at both sites are liable to present in this way. Occasionally a carcinoma gives rise to secondary deposits without it being possible to identify the primary tumour.

**Treatment.** Surgery offers the only hope of cure, but few would deny that the results of operation are somewhat dispiriting. Experience naturally varies in different places, but in most general hospitals, from 25 to 30 per cent. of all cases are clearly inoperable when first seen. In the remainder resection of the growth is done in about one in four to one in six cases. The mortality varies with the actual procedure used as well as the age and general condition of the patient. The operative mortality is in the region of 20 to 25 per cent. for any hospital receiving its patients from a local community. Better mortality figures have been reported with some of the selected material admitted to some hospitals.

Before embarking on exploration of the abdomen it is important to assess carefully the general health, and to relieve anaemia, hypoproteinaemia, avitaminosis and dehydration. Gastric lavage is often valuable in cleansing the stomach before operation and should not be neglected.

**A. Resectable Growths.** These can be divided into the possibly curable, with apparently localised disease, and those cases in which resection is justified although extragastric spread has occurred.

(i) *The "Curable" Growths.* Tumours at the pyloric end are dealt with by partial gastrectomy, but the operation is more extensive than that performed for peptic ulcer. Tumours of the mid-gastric region, and those of the fundus which do not encroach on the cardia, can be dealt with by abdominal total gastrectomy. Tumours at the fundus which involve the cardia and lower end of the oesophagus necessitate an abdominothoracic oesophageogastrectomy. In all these operations, the greater and lesser omentum, the first 1½ inches of the duodenum, and generally the spleen



FIG. 9. Leiomyoma of stomach. Barium meal film showing circular tumour and central ulcer filled with barium. (Courtesy of Dr. J. M. Stewart.)

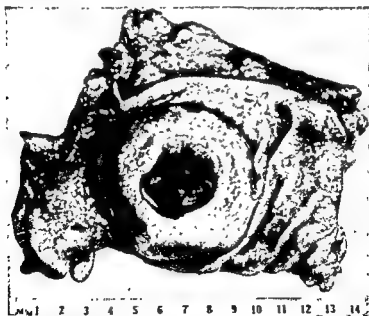


FIG. 10. Leiomyoma of stomach. Specimen from stomach shown in Fig. 9. The patient had a severe hæmatemesis. (Mr. E. B. Whittingham's case.)

35/1 and 6/1. Chronic peptic ulcer is commoner in town than country folk, and gastric ulcer is more prevalent in the lower social grades. These points are mentioned, partly from general interest, and partly to enable the reader to understand the wide variations in the figures quoted by different authorities.

## OTHER GASTRIC TUMOURS

**Sarcoma.** Sarcomas constitute about 1 per cent. of all malignant tumours of the stomach, and include lympho-, leiomyo- and fibrosarcomatous types. Lymphosarcoma may be localised to the stomach or part of a generalised condition. Clinically and radiologically, there may be a resemblance to carcinoma, but the prognosis is generally better; the operative mortality is lower and about a third of the survivors live for five years or more. Lymphosarcoma is usually radiosensitive, and may justify post-operative irradiation.

**Benign Tumours.** All types of benign tumour are certainly rare as clinical entities, although detailed scrutiny of the stomach at necropsy shows them to be not uncommon. Epithelial tumours include *papilloma* and *adenoma*. Although malignant change can occur, the consensus of opinion is that it is unlikely that many carcinomas arise in this fashion. *Diffuse adenomatous polyposis* is an uncommon condition which may be demonstrated as multiple filling defects on X-ray. Mesenchymal tumours include *leiomyoma* (Figs. 9 and 10), and *neurilemmoma*: the latter arise from the Schwann sheath of nerve fibres in the stomach. These last two tumours occasionally ulcerate, with consequent bleeding and anaemia, and occasionally there are obstructive symptoms. *Fibroma*, *lipoma* and *angioma* are all very rare.

## PEPTIC ULCER

Benign ulcers of the stomach and duodenum, and less commonly of other sites, are grouped under the generic title of peptic ulcer. The use of such a title does not imply that all such ulcers are produced in the same manner. The aetiology of gastric ulcer differs from duodenal ulcer in several important respects; and these differences will emerge in the course of subsequent discussion. Nevertheless, it is convenient to consider the aetiology and pathology of these ulcers together.

**Aetiology.** It has been estimated that there may well be 1,500,000 past or present sufferers from peptic ulcer in England and Wales alone. There are certainly marked variations in the incidence of peptic ulcer in different countries, and there are also quite surprising variations in different parts of the same country. In Britain generally, ulcers are about three times as common in men as in women. Under the age of forty-five, duodenal ulcer is commoner in both sexes. Over forty-five, duodenal ulcer is usually commoner in men but gastric ulcer preponderates in women. The proportions of duodenal and gastric ulcers in any series varies with the age, sex, geographical and other factors. Thus, in England during the 1940's, the proportion of duodenal to gastric ulcers in male hospital cases varied from about 12/1 in patients less than twenty-five years, to 2/1 in those over forty-five years. In Glasgow, during the same period, the duodenal/gastric ulcer ratio in men of these two groups varied between

Both acute and chronic ulcers tend to occur at similar sites, but it is hard to assess the true frequency of acute ulcers, since they are shallow, difficult to demonstrate on X-ray and may quickly heal. Gastrosomy has, however, shown that such ulcers are more frequent than was formerly appreciated. Intermediate types of ulcer are often dubbed subacute, but the frequency of the different varieties shows considerable differences in necropsy, gastroscopic, X-ray and surgical series.

*Acute Peptic Ulcers.* These are shallow, extending down to the submucosa, and sometimes multiple (Fig. 11). The ones which get into the surgeon's hands have usually caused profuse bleeding and yet they



FIG. 11. Acute ulcers of stomach. Necropsy specimen showing several shallow acute ulcers just below esophago-gastric junction.

may be impossible to locate from outside the viscus. Sometimes such an ulcer is only identified at operation by palpation of the clot in the vessel which has caused the bleeding.

*Chronic Peptic Ulcers.* These are usually single, but there may be scars of previous ulcers (Fig. 12). Coincident gastric and duodenal ulcers are found in about 10 per cent. of operation cases and three or four gastric ulcers are not uncommon. The serous aspect of the organ is often scarred and may become stippled with petechiae on handling. The crater is round or ovoid, and extends down to or through the muscular layer—the latter being completely destroyed. The diameter varies from a few millimetres to 10 cm. or more. As the ulcer extends it may penetrate adjacent structures, such as the liver or pancreas, or perforate into the peritoneal cavity. The edges of a chronic ulcer are commonly overhanging and the base is covered with exudate, beneath which are granulation tissue and fibrous layers. The muscularis mucosæ and main muscle layers tend to



*Acid and pepsin* though possibly not the most important factors in causing ulcers are probably among the most important in perpetuating them. An active peptic ulcer probably never occurs with achlorhydria. The normal secretion of acid on eating is partly due to stimuli mediated by the vagi, and partly due to *gastrin* secreted in the pyloric end of the stomach. Three aspects of secretion may be relevant to ulcer production; the quantity, the quality, and the amount of the interdigestive secretion. An isolated fractional test meal gives little idea of the stomach's secretory potentialities. Apart from qualitative differences in the juice secreted, there is an increased secretory volume in duodenal ulcer patients. The interdigestive and nocturnal secretion too is usually higher, and this is largely nervous in origin. In gastric ulcer patients, the acidity is normal or decreased. The quantity of secretion is related to the secretory cell mass, and the gastroscopic appearances can be correlated with this; the mucosa is thus atrophic in achlorhydria and hypertrophic in hyperchlorhydria. *Other Causal Factors.*—Peptic ulcer occasionally occurs as a complication of shock, burns and brain stem damage, and in these cases a *central nervous influence* is believed to act via the vagi. The relative fixity of the mucosa along the lesser curve of the stomach may make it more sensitive to *trauma* from food. *Vascular factors* too have been postulated. The recent demonstration of arteriovenous shunts in the gastric mucosa suggests a possible mechanism for local ischæmia although it does not explain the predilection for certain sites. *Nutritional factors* too are of possible importance although we know little about them. The relative infrequency of active peptic ulcer in pregnant women, as well as the tendency to heal at this time, suggest that *hormones* may play a part in some cases: a powerful histamine-inhibiting substance (urogastrone) has been isolated from the urine, and a similar inhibitory effect is caused by the secretion of enterogastrone secondary to the arrival of fat in the intestine.

The resistance of the normal gastric and duodenal mucosa is quite remarkable. *Mucus* exerts a protective effect on the mucosa, and it is believed that *reduced mucus secretion*, as well as the formation of mucolytic enzymes, may lower the resistance of the mucosa. In these and other factors which determine the resistance of the mucosa to autodigestion lie possibly important clues to the prevention and cure of peptic ulcer.

**Pathology.** Peptic ulcer may occur in the *stomach*, *duodenum*, *œsophagus*, the *jejunum* close to a gastro-enterostomy, and in a Meckel's diverticulum, indeed in any of the tissues exposed to pepsin and hydrochloric acid. Such ulcers may be acute or chronic. In the stomach, 95 per cent. of ulcers are on the lesser curve or the immediately adjacent parts of the posterior and anterior walls; the remainder are in the pyloric antrum or close to the cardia. Duodenal ulcers are almost always on the anterior or posterior walls of the first part of the duodenum.

Both acute and chronic ulcers tend to occur at similar sites, but it is hard to assess the true frequency of acute ulcers, since they are shallow, difficult to demonstrate on X-ray and may quickly heal. Gastrosocopy has, however, shown that such ulcers are more frequent than was formerly appreciated. Intermediate types of ulcer are often dubbed subacute, but the frequency of the different varieties shows considerable differences in necropsy, gastroscopic, X-ray and surgical series.

*Acute Peptic Ulcers.* These are shallow, extending down to the submucosa, and sometimes multiple (Fig. 11). The ones which get into the surgeon's hands have usually caused profuse bleeding and yet they



FIG. 11. Acute ulcers of stomach. Necropsy specimen showing several shallow acute ulcers just below esophago-gastric junction.

may be impossible to locate from outside the viscus. Sometimes such an ulcer is only identified at operation by palpation of the clot in the vessel which has caused the bleeding.

*Chronic Peptic Ulcers.* These are usually single, but there may be scars of previous ulcers (Fig. 12). Coincident gastric and duodenal ulcers are found in about 10 per cent. of operation cases and three or four gastric ulcers are not uncommon. The serous aspect of the organ is often scarred and may become stippled with petechiae on handling. The crater is round or ovoid, and extends down to or through the muscular layer—the latter being completely destroyed. The diameter varies from a few millimetres to 10 cm. or more. As the ulcer extends it may penetrate adjacent structures, such as the liver or pancreas, or perforate into the peritoneal cavity. The edges of a chronic ulcer are commonly overhanging and the base is covered with exudate, beneath which are granulation tissue and fibrous layers. The muscularis mucosæ and main muscle layers tend to

fuse at the edge and endarteritis is often seen in the base. In the event of hæmorrhage the rigidity of such vessels and the tough fibrous base hinder spontaneous arrest of bleeding. Similarly, the dense fibrous tissue and adherence to surrounding structures impair sound healing. Although the granulation tissue and the edges of the ulcer usually show lymphocyte and plasma cell infiltration, the inflammatory process is often more acute. Thus, when a perforation is repaired, biopsy of what looks like a chronic ulcer sometimes shows that the induration is due to œdema and not fibrosis and that the ulcer is a subacute one. Malignant change

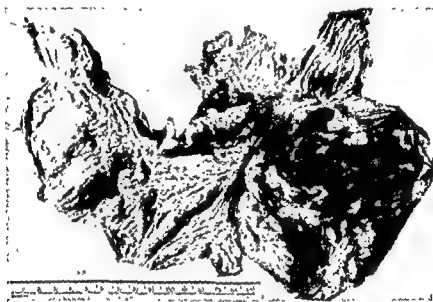


FIG. 12. Chronic gastric ulcer. Situated at junction of the upper and middle thirds of lesser curve. Necropsy specimen opened along greater curve with the pylorus to the left and œsophagus above and to the right.

is uncommon in gastric and unknown in duodenal ulcer. It was the downgrowth of epithelium at the edge of a gastric ulcer and its distortion by fibrosis which formerly tempted some pathologists to diagnose malignancy where none existed. Scar contracture adjacent to an ulcer may result in pyloric stenosis or hour-glass deformity of the stomach.

**Clinical Features.** It is important to appreciate that the severity of the symptoms frequently bears no relationship to the size of the ulcer. Severe pain can be caused by a tiny acute ulcer and a huge chronic ulcer may be symptomless for long periods. Nevertheless, a reasonably typical pattern occurs in most cases and pain is usually the most characteristic symptom.

In *chronic duodenal ulcer* pain tends to be episodic and seasonal. In some patients, the attacks occur especially in spring and autumn, and persist for two or three weeks at a time. The bouts of pain occur one to two hours after food, and sometimes pain is precipitated by hunger. This hunger pain and the quite frequent night pain are probably due to

the high interdigestive and nocturnal secretion of acid. The pain is usually situated in the epigastrium or right hypochondrium, and may also occur between the scapulae. It is generally relieved by food and alkalis. (3)

In chronic gastric ulcer pain tends to occur sooner after food, it rarely disturbs the patient at night and hunger pain is uncommon. Pain is usually relieved by alkalis and is not episodic as in duodenal ulcer. It is situated in the epigastrium, left hypochondrium or back. Pain in the back, both with gastric and duodenal ulcers, is often due to a penetrating posterior ulcer. High gastric ulcers may cause substernal pain, but this may also be due to acid regurgitation into the oesophagus of a patient with duodenal ulcer who has hyperchlorhydria and pylorospasm. (1)

Of the various other symptoms, vomiting is variable. In uncomplicated cases, it is more common with gastric than duodenal ulcer and tends to relieve the pain. Vomiting in duodenal ulcer cases is generally due to associated pyloric oedema, spasm or stenosis. Bleeding occurs at some time in the history of many chronic ulcer cases. It varies from slight blood-staining of the vomitus to catastrophic and rapidly fatal hæmorrhage. Either hæmatemesis or mælena may occur and the actual route of blood loss bears little relation to the ulcer site. It is unjustifiable to assume that hæmatemesis is due to a gastric ulcer and that mælena must be caused by a duodenal lesion.

**Diagnosis.** However typical the history it is important to establish a precise diagnosis. This is usually possible with barium meal X-ray, though gastroscopy may be the only way to demonstrate some gastric ulcers.

**Barium Meal.** It has sometimes been said that the most important thing on an X-ray report is the signature, and this is certainly true of the barium meal examination. With skilled radiology and modern technique a very high standard of accuracy is achieved (Fig. 13). The visualisation of the crater is the only certain evidence of an ulcer, and its demonstration may necessitate careful orientation of the patient, the application of pressure to the abdomen and various other refinements of technique. The size and position of the lesion, the presence of mucosal oedema or large gastric folds all influence the possibility of diagnosis. A large crater is by no means indicative of malignancy, for many huge innocent ulcers are seen. Scarring of the mucosa may indicate the site of a healed ulcer; the most characteristic feature of this is the presence of converging folds. **Gastroscopy.**—This is of value in some cases, both in showing a gastric lesion which is not seen on X-ray and in determining the nature of a doubtful lesion. The value and limitations of gastroscopy vary with the skill and experience of the operator.

**Differential Diagnosis.** Duodenal ulcer symptoms may be simulated by those of chronic cholecystitis, though usually there is not such marked periodicity, nocturnal discomfort and relief by food. Recurrent subacute

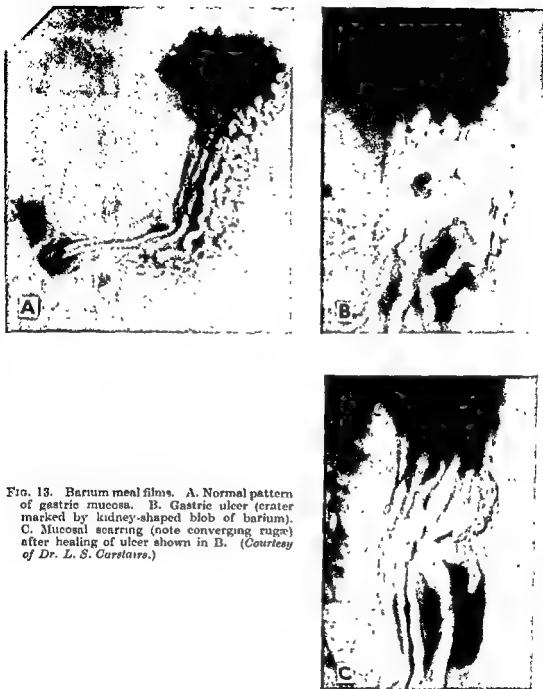


FIG. 13. Barium meal films. A. Normal pattern of gastric mucosa. B. Gastric ulcer (crater marked by kidney-shaped blob of barium). C. Mucosal scarring (note converging rugae) after healing of ulcer shown in B. (Courtesy of Dr. L. S. Carstairs.)

*appendicitis* can also simulate duodenal ulcer pain. Although the differentiation of gastric and duodenal ulcers is ordinarily perfectly straightforward on X-ray, the radiologist may find it extremely difficult to be sure of the diagnosis in some juxtapyloric lesions. In such cases, if a prepyloric ulcer cannot be certainly excluded and the clinician is fearful of a possible *carcinoma*, the finding of achlorhydria supports the latter diagnosis. Apart from the occasional case in which there is difficulty

in differentiating a duodenal from a prepyloric ulcer, a more common problem is the differentiation of a benign from a malignant ulcer in the stomach itself. An unusual ulcer site, as on the greater curve, is highly suspicious of carcinoma. A big crater on the lesser curve, especially if smooth and projecting well beyond the stomach profile, is rarely malignant, but a large crater in the pyloric region is often malignant. A crater in a filling defect is most likely due to a carcinoma, but mucosal folds covering on a crater are confirmatory of a benign ulcer. In the event of a large ulcer crater being demonstrated it is of more than academic importance to establish a correct diagnosis. Precipitate surgical interference (engendered by unreasonable fear of malignancy) may make operation difficult and hazardous, whereas careful medical treatment of a large benign ulcer will greatly facilitate subsequent surgery.

Other conditions worthy of note are *hiatus hernia* (see Chap. 43) which sometimes causes similar symptoms to peptic ulcer and indeed may coexist with an ulcer. *Chronic Gastritis* is another condition to be excluded, but it should be remembered that large rugæ may conceal an ulcer crater both on X-ray and gastroscopy and thus lead to an incorrect diagnosis of gastritis.

It is worthy of note that a number of patients with peptic ulcer are labelled *psychoneurotic* through failure to demonstrate the crater. This is especially liable to occur with a posterior gastric ulcer, with small duodenal lesions, with ulceration in a highly rugose stomach, and even with a huge benign ulcer. In the latter instance a truly huge crater may be overlooked on X-ray through being mistaken for the lesser curvature itself. When the radiologist demonstrates a "scar" without a crater it behoves the surgeon to be especially careful before embarking on operation for a presumed active ulcer. The scar may merely mark the site of an inactive ulcer when the symptoms are due to some other cause.

### TREATMENT

The great majority of peptic ulcer patients are never seen by a surgeon, and symptoms in many of the more troublesome cases are well controlled by medical treatment. Surgery is reserved for certain complications of peptic ulcer and for those patients whose symptoms cannot be properly relieved by the physician. Views on the most suitable methods of surgical treatment have slowly crystallised. It would, however, be wrong to presume that methods at present in vogue will necessarily stand the test of time. Some operations have not been used sufficiently long for their value to be accurately assessed. Indeed it has been said that it takes at least ten years to find a gastric operation out. Impressive early results are sometimes not borne out by long-term follow-up. It is also important to appreciate that a method which gives good results in one place may prove less satisfactory in equally skilled hands elsewhere.

The common indications for surgery are : (1) Repeated relapse and loss

of working capacity despite careful medical treatment; (2) continued pain and failure of the ulcer to heal despite intensive medical treatment in hospital; (3) suspicion of malignancy in gastric cases; (4) marked scarring with fixed pyloric narrowing or hour-glass stomach; (5) perforation; (6) selected cases of bleeding ulcer. Few absolute criteria for operation can be laid down, and indications are modified by individual circumstances. One patient sticks doggedly to his job with symptoms which drive another to his bed for several weeks. Therefore, when interpreting the degree of disability due to an ulcer the patient must not be judged superficially, and many aspects of his life and character must be considered. The patient is more likely to get the maximum benefit from surgery when there is full understanding and co-operation between family doctor, physician, radiologist and surgeon.

**The Choice of Operation.** At one time there were various local operations for gastric ulcer, including wedge resection, sleeve resection and diathermy excision. Recurrent ulceration was common after these procedures; they were therefore abandoned. In the case of duodenal ulcer, *gastro-enterostomy* was usually performed with the object of bypassing the crater and introducing alkaline juice into the stomach. However, reactivation of the ulcer or ulceration on or near the anastomosis occurred in a high percentage of cases. *Partial gastrectomy* gradually became more popular: in duodenal ulcer because it removed a large part of the acid-secreting area and substantially reduced the likelihood of recurrence; and in gastric ulcer because it removed most of the lesser curve, which is the only place where such an ulcer is likely to recur. After World War II, the operation of *vagotomy* was revived and widely practised in the treatment of duodenal ulcer, but recurrent ulceration was so frequent that vagotomy alone was soon abandoned in this type of case. A combination of *vagotomy* and *gastro-enterostomy* continues to be used quite extensively by some surgeons who have been impressed by its effectiveness in many cases of duodenal ulcer. Vagotomy, either alone or in combination with gastro-enterostomy, has no place in the treatment of gastric ulcer; indeed it is by no means rare for a gastric ulcer to succeed a vagotomy done for the relief of a duodenal ulcer. Vagotomy alone is of some value in the treatment of selected cases of anastomotic ulcer. At the present time the main choice of surgical treatment lies between: (1) *Partial Gastrectomy*. For all cases of gastric ulcer needing surgery and for many duodenal ulcers. Two main varieties are the Polya type with end-to-side gastrojejunal anastomosis, and the Billroth I type with end-to-end gastroduodenal anastomosis. (2) *Gastro-enterostomy alone*. Now chiefly limited to elderly or poor-risk cases with *pyloric stenosis* and a burnt-out or relatively quiet ulcer. (3) *Vagotomy*. (4) *Combinations of (2) and (3), or (1) and (3).*

**Surgery for Gastric Ulcer.** This will be dealt with first since the choice of operation is straightforward and limited to partial gastrectomy.

Although many surgeons prefer the Billroth I type of operation in these cases, other equally experienced and eminent authorities adhere to the Polya type of operation. Recurrent gastric ulcer is a rarity after a properly performed partial gastrectomy, and anastomotic ulcer is much less common than after gastrectomy for duodenal ulcer.

**Surgery for Duodenal Ulcer.** Many of these patients have hyperchlorhydria, and it is virtually unknown for active duodenal ulcer to occur with true achlorhydria. Drastic reduction of acidity is essential to long-term success, and this may be effected either by partial gastrectomy, or vagotomy together with gastro-enterostomy. *Partial gastrectomy* is still regarded as the operation of choice by the majority of surgeons. Following gastrectomy, about 80 per cent. of patients are really well and return to their normal occupation; anastomotic ulcer occurs in not more than 2 or 3 per cent., and the mortality in elective cases is around 1 to 3 per cent. in the good general hospital. Most surgeons prefer the Polya type of procedure, since experience has shown that recurrent ulceration and stenosis is liable to follow the Billroth I procedure for duodenal ulcer. It is not obligatory to remove the duodenal ulcer itself and the majority of surgeons only do so when it is accessible. Some make a special point of "going for" every ulcer, but the hazards of gastrectomy tend to be increased if this is done routinely. A duodenal ulcer left *in situ* normally heals following exclusion and gastric resection. When a large duodenal mass is present with marked œdema of the tissues, and especially if the patient is elderly or a poor risk, it is occasionally wise to do a two-stage operation by transecting the stomach proximal to the pylorus at the time of gastrectomy, and then removing the pyloric antrum six or eight weeks later. The second procedure is usually very straightforward as the œdema has then resolved. The second stage should not be delayed too long as, with the pyloric antrum present, reactivation of the ulcer may occur. An alternative to the two-stage operation is to divide the pyloric antrum and core out its mucosa. This obviates the need for subsequent excision of the antrum but does not give such a good "stump" for closure.

**Vagotomy and Gastro-enterostomy.** This certainly gives good immediate results in cases of duodenal ulcer, but experience of the long-term effects is variable. Some have reported anastomotic ulcer in 10 per cent. or more of cases whereas others have recorded anastomotic ulcer in a much smaller proportion of cases. In general the mortality of the procedure is lower than that of gastrectomy, but strict comparison is difficult owing to different standards of selection for the two operations. In a condition such as duodenal ulcer which, in the absence of complications, rarely kills the patient, there are some who feel that even the possibility of anastomotic ulcer in 10 per cent. of cases is more than compensated by the lower operative mortality. Even if one in ten patients do have to be subjected to subsequent gastrectomy, the protagonists of vagotomy and gastro-enterostomy feel that the overall risk is still appreciably lower.



It would seem unwise to attempt to adjudicate too emphatically between these two operations, for the circumstances of the individual case, as well as the surgeon's own training and experience, must appreciably influence treatment. There is a definite tendency, both in Great Britain and in the United States, to use vagotomy and gastro-enterostomy for many uncomplicated cases of duodenal ulcer, for bad-risk cases, and for many patients under thirty-five years. Even the advocates of the lesser-risk operation concede the case for gastrectomy in those patients with large penetrating or bleeding ulcers, or patients with a coexistent gastric ulcer. At the present time it is best for surgeons to keep an open mind about the merits of the lesser-risk operation. *Limited partial gastrectomy plus vagotomy*—As some of the ill-effects of the modern gastrectomy can be attributed to the relatively small size of the gastric remnant, some have sought to limit its possible disadvantages by combining vagotomy with a less radical resection. Experience to date suggests that, although the incidence of anastomotic ulcer is lower than for vagotomy plus gastro-enterostomy, the mortality does not differ much from that of orthodox partial gastrectomy.

**Anastomotic Ulcer.** Anastomotic ulcer, sometimes called marginal ulcer, as will have been appreciated already, is a possible complication of the operative treatment of gastric or duodenal ulcer (Fig. 14). It is much commoner following operation for duodenal ulcer, and commoner after the bypass than the resection operations. Long term follow-up of duodenal cases treated by simple gastro-enterostomy has shown that anastomotic ulcer may occur in as many as 10 to 30 per cent. of cases, but there is a good deal of variation in the incidence in different series. In this connection we are considering only those cases which cause appreciable symptoms, since it is likely that a number of patients have transient anastomotic ulceration which never demands surgical relief.

**Pathology.** The macro- and microscopic features are similar to those of primary peptic ulcer, but the ulcer tends to be more linear. The ulcer mass commonly becomes adherent to the adjacent colon and in some cases a gastro-jejuno-colic fistula occurs. Bleeding or perforation into the peritoneum occasionally occurs.

**Clinical Features.** Pain tends to be more persistent and continuous than in primary peptic ulcer. In at least 50 per cent. of cases the symptoms occur within six months of operation. The pain is often to the left of the umbilicus, sometimes radiating downwards. It is often severe and may be quite bizarre in its character and distribution. There is sometimes hæmatemesis and melæna. With gastro-jejuno-colic fistula, diarrhoea, marked loss of weight, eructation of foul-smelling gas and, occasionally, true faecal vomiting may occur. Sometimes there are marked nutritional changes without the true cause being identified.

**Diagnosis.** Anastomotic ulcer may be strongly suspected from the history. The physical signs are not distinctive, but there is often tender-

ness over the site of the stoma and sometimes a mass is felt. The ulcer may be positively diagnosed on X-ray (Fig. 14); it is most commonly in the jejunum and sometimes at the junction of stomach and small gut. A positive diagnosis of gastro-jejuno-colic fistula is established better by barium enema than barium meal X-ray, and indeed the latter may miss this lesion completely.

*Treatment.* The operation of choice for anastomotic ulcer following gastro-enterostomy is partial gastrectomy. When such an ulcer follows gastrectomy, then a higher resection is generally done. Attempts to cure anastomotic ulcer by vagotomy usually give no more than short-lived relief. Gastro-jejuno-colic fistula is dealt with similarly. In those cases with marked loss of weight and diarrhoea, and especially if there is much œdema and inflammation at the site of the fistula, it is best to do a preliminary colostomy at or near the hepatic flexure and then remove the fistula after an interval of one to three months. By the time of the second operation it is usually found that the formerly severely inflamed fistula can be easily dealt with.



FIG. 14. Post-gastrectomy barium meal film. Anastomotic ulcer following operation for duodenal ulcer. (Courtesy of Dr. L. S. Carstairs.)

## COMPLICATIONS OF PEPTIC ULCER

### PERFORATION

In either acute or chronic ulceration of the stomach or duodenum there may be progressive penetration of the wall of the organ with perforation into the peritoneal cavity. In Great Britain perforated duodenal ulcer is from five to eight times commoner than the gastric variety. With juxtapyloric ulceration, especially if there is much œdema present, it may be impossible to say whether the perforation is in the stomach or duodenum.

*Clinical Features.* There is a past history of ulcer pain in some 50 per cent. of cases. Perforation may be preceded by prodromal symptoms for a week or so, or it may come out of the blue. Typically there is agonising

abdominal pain of sudden onset, starting in the epigastrium and spreading over the whole abdomen. Reflex vomiting is common enough at the onset but rarely persists. There may be traces of blood in the vomitus, and occasionally perforation is associated with major bleeding, or one complication may follow the other. The pain is often referred to both shoulders due to irritation of the diaphragm. Sometimes the symptoms of perforation come on more gradually due to slow leakage into the peritoneum, and the diagnosis is then less easily made. If this possibility



FIG. 15. Perforated gastric ulcer. Plain (erect) film of abdomen showing unusually large gas collection under diaphragm on both sides. (Courtesy of Dr. L. S. Carstairs.)

is not borne in mind it is surprising how easily such a complication can be overlooked even in hospital. For this reason it has sometimes been said that there is no more dangerous place to perforate than in a medical ward, excepting, possibly, in a surgical ward!

*On Examination.* The patient usually lies quite still, either supine or on one side. In the first few minutes after perforation he is shocked, pale, anxious and sweating. Following transient increase in the pulse-rate there is usually little alteration in pulse and blood pressure during the first few hours, and the temperature is often subnormal. Tenderness and rigidity are generally widespread, and the muscle rigidity is commonly "board-like." In some cases, there is a more gentle leak into the right

paracolic gutter with more obvious signs on that side of the abdomen. Percussion of the abdomen often shows diminution or absence of liver dullness and this is pathognomonic of perforation, but it is not always present, for there may be only a small amount of free gas, or it may be trapped under the liver. Plain X-ray of the abdomen in the erect position will confirm the presence of free gas, or otherwise assist in the diagnosis of a doubtful case (Fig. 15). Bowel sounds are usually absent; thus the silent abdomen is characteristic on auscultation.

Within an hour or two of perforation there is often diminution of pain and decrease in the physical signs. If the patient is seen for the first time at this stage the diagnosis may not be so obvious. However, in the absence of treatment the signs of peritonitis and deterioration supervene. It may be said that the speed of development and the severity of the physical signs depend upon the site and size of the perforation and the volume of fluid escaping from the gut. Perforation of a gastric ulcer into the lesser peritoneal sac may cause symptoms which suggest no more than the "flare-up" of an ulcer.

**Differential Diagnosis.** In *acute cholecystitis* tenderness and rigidity are more localised, bowel sounds are present and no free gas is demonstrated. *Acute pancreatitis* sometimes simulates perforation quite closely, but the rigidity is less marked, vomiting is often rather persistent, and there is no evidence of free gas. *Exacerbation of peptic ulcer* symptoms may be confused with the less dramatic cases. *Acute gastritis* may also cause marked symptoms but less widespread signs. Other important conditions to be considered are *myocardial infarction*, *pneumonia with diaphragmatic pleurisy*, *dissecting or leaking abdominal aneurysm*, and, rarely, the gastric crisis of *tuberculosis*.

The presence of free gas in the peritoneal cavity has been mentioned in connection with differential diagnosis, but it should be emphasised that free gas cannot always be demonstrated after perforation. Previous adhesions between liver and diaphragm may prevent its demonstration or the leakage of gas may be minimal. The actual volume of free gas probably depends on the air-swallowing propensities of the patient and the amount of air in the stomach at the time of perforation.

**Treatment.** Early treatment is imperative and the mortality rate increases with the elapse of time after perforation. Gastric perforations are about twice as fatal as duodenal ones and increasing age adds to the hazards. A chronic ulcer is more dangerous than an acute one. There are three possible methods of treatment: (1) Operative closure of the perforation; (2) non-operative treatment with gastric aspiration and intravenous therapy; (3) partial gastrectomy in selected cases.

*Operative closure of the perforation* is the most widely practised method of treatment. Prior to operation, and as soon after diagnosis as possible, the stomach is emptied by aspiration and, if necessary, an intravenous transfusion is started. Although it is often stated that no pain-relieving

drug should be given until the patient is seen by the surgeon, this is really cruel advice for many cases. Movement of the patient from his home and the journey to hospital by ambulance can prove almost intolerable even to a strong man. Therefore, it is wrong to assert quite dogmatically that the family doctor should not give morphine. The only thing he *must* do is to be sure that the dose and time of administration is clearly recorded in his letter or on the patient.

Preoperative aspiration of the stomach should be as complete as possible, for not only does this help to relieve the pain but it also reduces subsequent contamination of the peritoneum to a minimum. Details of the operation are given later. Following operation, gastric aspiration and intravenous therapy is continued until it is clear that peristalsis is restored and the stomach emptying satisfactorily. In many cases this ancillary treatment is unnecessary, but delayed emptying of the stomach may occur in those patients with much pyloric stenosis or œdema. Slower progress is usual in those with extensive and longer-established contamination of the peritoneum. With these various points in mind the post-operative regime is adapted to the individual case. A light diet is started as soon as possible, and progressively increased to a reasonably normal diet. It is generally unwise needlessly to restrict feeding in these cases but, of course, the patient with an active chronic ulcer and persistent post-operative symptoms requires appropriate medical care.

*Non-operative treatment* by gastric aspiration and intravenous therapy is certainly a sound method in suitable cases and in practised hands. In these circumstances, the mortality rate compares favourably with that of operative closure. The method has been strongly and unjustly criticised by some authorities, but the fact remains that it is of real and established value. Non-operative treatment may be the method of choice for acute ulcer perforations with a short history. However, the clinical diagnosis of acute and chronic ulcer is not always easy since some long-established chronic ulcers are remarkably silent. This differentiation may also be difficult at operation although biopsy has shown that a proportion of ulcers formerly regarded as chronic are truly recent in origin.

Conservative (non-operative) treatment is contraindicated in cases with severe peritoneal soiling, in those who resent a stomach tube, or those in whom satisfactory aspiration is difficult. Conservative treatment is not recommended in any known chronic ulcer or when perforation is accompanied by bleeding. When there is doubt in the diagnosis and the possibility of some other condition needing early surgical relief operation should be advised. With these reservations it may be said that the choice between operative and conservative methods really depends upon the circumstances and practice of the individual surgeon and hospital.

*Method.* (1) Pain is relieved by a suitable dose of morphine or alternative drug. (2) An aspiration tube of adequate dimensions is passed and the stomach is completely emptied. In this connection it should be emphasised

that a tube about 8 mm. diameter with a good terminal eye and side holes is preferable to a Ryle's tube but is no more difficult to pass. (3) Continued aspiration of the stomach is effected either by intermittent or continuous suction. (4) Intravenous transfusion is maintained with due care to avoid unnecessary fluid and salt intake. (5) The patient is nursed in a comfortably recumbent position. (6) Oral fluids are withheld for not less than twenty-four hours and a light diet is started about the third or fourth day.

In conclusion, it may be said that although the conservative method may require more detailed supervision of the patient for the first two days the later course is generally uneventful.

*Partial Gastrectomy.* Since both of the above methods carry a higher mortality for chronic ulcers it is in these cases that immediate partial gastrectomy has been advocated. If a competent and experienced surgeon is available the patient with a long history of chronic ulcer is quite certainly best treated in this way. In these circumstances the mortality is lower than that achieved by simple closure or conservative treatment, and the bad ulcer patient is saved from two operations, or at any rate two periods in hospital. Immediate partial gastrectomy must be used with some discretion with poor risk cases and those who have been perforated more than twelve hours. It is, however, indisputable that even in these unfavourable circumstances the major procedure may still be more appropriate and safer than the lesser one. It is certainly wrong to do a partial gastrectomy for a perforated ulcer in a patient who has had relatively slight symptoms in the past. Any post-gastrectomy symptoms are likely to prove far more irksome and distressing by comparison with relatively mild preoperative symptoms. Immediate partial gastrectomy is the treatment of choice in the chronic ulcer patient with a bad history; in large gastric perforations and in chronic duodenal ulcer with pyloric stenosis.

*Chemotherapy and Antibiotics.* In discussing the above methods of treatment no reference has been made to the use of antibiotics. Although it is difficult to adduce detailed statistics in support of their use, few surgeons would feel justified in withholding antibiotics from those patients with severe contamination and when there is delay in treatment. Provided safe drugs are used there would seem to be no objection to routine antibiotic therapy, although it can certainly be dispensed with in the more favourable cases. For routine use crystalline penicillin (0.5 mega unit six-hourly) is most suitable, and this may be supplemented with a soluble sulphonamide such as sulphadimidine (gm. 1.5, six-hourly). Oxytetracycline should be reserved for the patient with dangerous peritonitis (see Chap. 110).

*Complications of Perforation.* Certain complications may occur after all methods of treatment. *Paralytic ileus* is not uncommon, and *peritonitis* is especially liable to occur when there is severe contamination and delay

drug should be given until the patient is seen by the surgeon, this is really cruel advice for many cases. Movement of the patient from his home and the journey to hospital by ambulance can prove almost intolerable even to a strong man. Therefore, it is wrong to assert quite dogmatically that the family doctor should not give morphine. The only thing he *must* do is to be sure that the dose and time of administration is clearly recorded in his letter or on the patient.

Preoperative aspiration of the stomach should be as complete as possible, for not only does this help to relieve the pain but it also reduces subsequent contamination of the peritoneum to a minimum. Details of the operation are given later. Following operation, gastric aspiration and intravenous therapy is continued until it is clear that peristalsis is restored and the stomach emptying satisfactorily. In many cases this ancillary treatment is unnecessary, but delayed emptying of the stomach may occur in those patients with much pyloric stenosis or œdema. Slower progress is usual in those with extensive and longer-established contamination of the peritoneum. With these various points in mind the post-operative regime is adapted to the individual case. A light diet is started as soon as possible, and progressively increased to a reasonably normal diet. It is generally unwise needlessly to restrict feeding in these cases but, of course, the patient with an active chronic ulcer and persistent post-operative symptoms requires appropriate medical care.

*Non-operative treatment* by gastric aspiration and intravenous therapy is certainly a sound method in suitable cases and in practised hands. In these circumstances, the mortality rate compares favourably with that of operative closure. The method has been strongly and unjustly criticised by some authorities, but the fact remains that it is of real and established value. Non-operative treatment may be the method of choice for acute ulcer perforations with a short history. However, the clinical diagnosis of acute and chronic ulcer is not always easy since some long-established chronic ulcers are remarkably silent. This differentiation may also be difficult at operation although biopsy has shown that a proportion of ulcers formerly regarded as chronic are truly recent in origin.

*Conservative (non-operative) treatment* is contraindicated in cases with severe peritoneal soiling, in those who resent a stomach tube, or those in whom satisfactory aspiration is difficult. Conservative treatment is not recommended in any known chronic ulcer or when perforation is accompanied by bleeding. When there is doubt in the diagnosis and the possibility of some other condition needing early surgical relief operation should be advised. With these reservations it may be said that the choice between operative and conservative methods really depends upon the circumstances and practice of the individual surgeon and hospital.

*Method.* (1) Pain is relieved by a suitable dose of morphine or alternative drug. (2) An aspiration tube of adequate dimensions is passed and the stomach is completely emptied. In this connection it should be emphasised

chronic ulcers (Fig. 16), but it is not uncommon for heavy bleeding to come from shallow acute erosions. Bleeding in a gastric ulcer usually arises from a branch of the *left gastric artery*, and less often from the left gastric itself or the splenic artery. Either hæmatemesis or melæna or both may occur; the actual route of blood loss gives no certain clue as to whether the ulcer is above or beyond the pylorus.

**Treatment.** The majority of these cases are well treated by the physician. Adequate blood transfusion, careful sedation and proper feeding will see most patients safely through. The physician may then carefully assess the patient and, if necessary, refer him to the surgeon for operation with all the advantages of full physical and psychological preparation.

**Absolute Indications for Emergency Surgery.** The minority of cases require emergency surgical treatment, and the following are the chief indications for such intervention: (1) Continued severe bleeding and deterioration despite blood transfusion; (2) recurrent bleeding in hospital especially with continued ulcer pain; (3) combination of bleeding and perforation.

Important subsidiary factors to be taken into consideration are the patient's *age*, the presence of *hypertension* and *arteriosclerosis*, a *long history* of chronic ulcer and the actual circumstances of the bleeding. Whereas young men of thirty may stand up well to repeated hæmorrhage and rarely die, patients over fifty, especially if obese, run much greater risk. Hypertension, arteriosclerosis and other chronic associated diseases all increase the risk. It is, however, illogical to deduce from an apparently hardened peripheral artery that the bleeding vessel is in a similar state, and that this is an argument for operation. It is perfectly true that the vessel in the ulcer base is often sclerotic, and that this rigidity seriously impedes spontaneous arrest of bleeding, but such sclerosis is the result of the ulcer pathology and rarely due to generalised arteriosclerosis. The state of the peripheral vessels has little influence on the discerning surgeon when he is weighing the pros and cons of operation. If an aspiration tube is kept in the stomach it is often useful in demonstrating fresh bleeding. In really aged patients, for example those over seventy-five, the risks of both continued hæmorrhage and surgery are high. A decision as to the advisability of operation may be hard to make. However, if the patient is of poor morale, in bad physical health, and living alone, it is generally best to avoid surgery. On the other hand, a lively septuagenarian should be given the benefit of operation if it is indicated.

**Operative Treatment.** It is vitally important to empty the stomach before an anæsthetic is given, for there is always serious risk of vomitus being aspirated. A large-bore stomach tube should be passed, for small ones are especially liable to block with blood clot. Highly skilled general anæsthesia is essential, and the use of an endotracheal balloon tube guards against possible aspiration into the lungs of stomach contents which even the stomach tube cannot fully preclude.



in treatment. Adhesions may cause subsequent *intestinal obstruction*. Intraperitoneal *abscess* occurs, especially between coils of gut, although many of these resolve spontaneously. More serious are *pelvic abscess* and *subphrenic abscess*. Chest complications such as *basal collapse* and *consolidation* are relatively common, and *pleural effusion* or *empyema* may be associated with subphrenic abscess. *Pyloric obstruction* may be due to the buckling produced by operative closure or, more often, to subsequent oedema and stenosis related to the ulcer.

### BLEEDING PEPTIC ULCER

It is usual for the patient with a bleeding peptic ulcer to be admitted under a physician who maintains active liaison with a surgeon. This

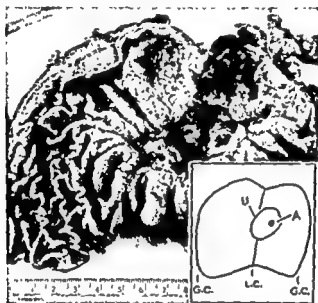


FIG. 16. Bleeding ulcer. Chronic ulcer on lesser curve of stomach with eroded vessel. Operation specimen with inset showing orientation of stomach. L.C. Lesser curve at upper end. G.C. Two edges of divided greater curve. U. Ulcer. A. Eroded artery.

arrangement proves very satisfactory, for it not only achieves a high standard of medical care but also ensures that the surgeon is kept in the picture. Operative treatment is only necessary in a minority of cases, but a cordial liaison between physician and surgeon is essential if sound selection is to be exercised. The incidence of emergency operations for the control of haemorrhage is generally between 5 to 10 per cent. of such cases admitted to hospital. Despite detailed statistical study it is exceedingly difficult to draw comparison between many published series, and circumstances differ widely in different places. For this reason the greatest circumspection is necessary in interpreting and comparing published series.

In general the biggest and most persistent haemorrhages occur from

elicited on shaking the patient. More important than the mechanical effects of obstruction are the dehydration and electrolyte disturbance which supervene. The heavy loss of chloride in the vomitus without equivalent sodium depletion causes alkalosis. The latter may be aggravated by injudicious use of stomach powders. Patients sometimes take excessive quantities of unsuitable agents, such as sodium bicarbonate, and thus develop consequent profound alkalosis. The blood urea is often raised and sometimes tetany occurs. *Treatment.*—The most careful preparation is essential prior to operation. The plasma electrolytes and blood urea are estimated and the chloride deficit is made good. In the majority of cases, as the chlorides are replaced the alkalosis and raised blood urea are also corrected. In long-standing cases of severe alkalosis, there is occasionally marked impairment of renal function which is only very slowly corrected. Whilst the blood chemistry is being attended to the stomach is thoroughly washed out each day with normal saline. Detailed fluid balance charts are kept and a suitable light diet is prescribed. It is wise to give vitamins by injection to ensure their absorption in the early stages of treatment. Only when patients are in good condition is it justifiable to submit them to operation. *Choice of Operation.*—In the majority of properly prepared patients partial gastrectomy is the operation of choice. In elderly patients with a "burnt-out" ulcer and strictly fibrotic stenosis, gastro-enterostomy is certainly justifiable. Exceptionally, an emergency gastro-enterostomy is justifiable in younger patients, but precipitate operation is commonly a reflection of inadequate preliminary treatment.

**Hour-Glass Stomach.** This is not a common condition. It is usually due to the scarring related to a large saddle ulcer on the lesser curve of the stomach and is more common in women. As in pyloric stenosis there is often an active ulcer present, and the symptoms may be due to this as well as the obstruction. Vomiting is neither so frequent nor so prominent as in pyloric stenosis; indeed, an hour-glass stomach is usually an incidental finding in the course of investigation and rarely presents as a clinical entity. When operative treatment is required this generally entails a partial gastrectomy.

#### OPERATIONS ON THE STOMACH

Before all elective operations for ulcer or growth any essential dental treatment is given and breathing exercises are taught. Patients with pyloric stenosis and those with a neoplasm with much stasis and ulceration are given stomach wash-outs. In most of these procedures a vertical upper abdominal incision is used; either midline or paramedian (muscle-reflecting or transrectal) incisions are made according to individual preference.

**Closure of a Perforation.** A short incision generally suffices. The perforation is exposed by suitable traction on the stomach, and in most

Once the abdomen is opened the ulcer is generally readily identified. Even the less obvious acute ulcers are often quickly spotted by the related oedema and thickening of the stomach or duodenal wall. When a gastroduodenal lesion is not seen it is wise to inspect the rest of the gut before opening the stomach. Occasionally the blood is coming from a Meckel's diverticulum, an argentaffinoma or some other tumour of the gut. If the source of the bleeding is still not evident it is wise to open the stomach. Sometimes a small seed-like clot is felt in the open end of the offending vessel and this may be the first sign of the ulcer. In expert hands the routine preoperative use of gastroscopy has sometimes enabled small lesions of this kind to be identified before laparotomy, but gastroscopic examination may be exceedingly difficult in these circumstances.

**Complications.** *Paralytic ileus* is especially common after emergency gastrectomy and this may partly be due to the fact that the gut is often distended and loaded with blood at the time of operation. *Sepsis* both in the wound and the peritoneal cavity is commoner than after elective gastrectomy. *Intraperitoneal abscess*, including *subphrenic abscess*, occasionally occurs. *Burst abdomen* is not uncommon, despite the most careful closure and the use of tension sutures. It is largely promoted by post-operative distension or coughing, and sepsis may also be a factor. In this connection it is wise to give these patients 1,000 mg. ascorbic acid, intramuscularly, on each of the first three days, for some of them may be depleted of the vitamin by previous dieting. *Chest complications* are more common than after elective operations, firstly, because the patient has often been confined to bed and immobile for a day or two prior to operation; secondly, because of abdominal distension which impedes respiration and, lastly, because of aspiration of blood and stomach contents. It is not uncommon for the patient to faint with the initial hæmorrhage and, if hæmatemesis then occurs, inhalation of vomitus may be considerable. Aspiration may have occurred, therefore, before the patient sees the doctor, and it must be guarded against at the time of operation. Every effort should be made to prevent *pulmonary collapse* and infection (see under *Bronchoscopy*). *Bronchopneumonia* and *lung abscess* may occur despite antibiotic therapy.

#### OBSTRUCTIVE COMPLICATIONS OF ULCER

**Pyloric Obstruction.** This condition usually results from a duodenal ulcer, but is occasionally due to a gastric ulcer close to the pylorus. Although actual cicatricial stenosis does occur, a proportion of these cases are due to oedema or sometimes pylorospasm close to an active ulcer. The stomach becomes dilated and hypertrophied. *Clinical Features.*—The obstruction causes accumulation of gastric secretions and food residue with consequent vomiting which characteristically occurs in the evening. The vomitus is generally large and contains old food residue. Visible peristalsis and distension of the stomach may be present, and a splash is

usually done with two layers of fine catgut. Some surgeons prefer to use silk or other non-absorbable material for the outer layer which picks up the serous and muscular coats. The inner layer of catgut passes through all coats. Either a simple running or loop-on-the-mucosa stitch may be used.

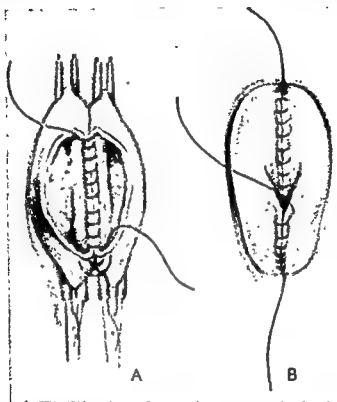


FIG. 18. Gastrojejunostomy. A. Insertion of posterior all-coats suture (previously inserted seromuscular suture not shown). B. Anterior all-coats suture being buried by anterior seromuscular suture.

**Vagotomy.** The left lobe of the liver is mobilised and retracted after dividing the triangular ligament. The peritoneum is incised over the lower part of the œsophagus and the latter is freed all round. Exposure is helped by traction on a length of rubber tubing slung around the lower œsophagus. In most cases there are two main nerve trunks; in about a quarter there are three, and in a few cases there are four. The right vagus lies behind and to the right of the œsophagus and usually a little separated from it, whereas the left is in close apposition anteriorly. A short length of each nerve is excised and the ends are ligated, or silver clips applied in case of bleeding from small vessels on the nerve. The excised lengths of nerve are sent for section and the anatomy is accurately recorded in case of subsequent question about the efficacy of the operation.

**Partial Gastrectomy.** Although many different operations have been

cases the opening is best closed with omentum (Fig. 17). A few Lembert sutures are inserted wide of the perforation and surrounding induration, and these are then tied over an attached portion of the greater omentum. This technique can be applied in almost any circumstances and avoids buckling the stomach or duodenum. Accessible free fluid and debris are sucked out. In some cases with excessive contamination a drain is inserted into the subhepatic (Morison's) pouch through a separate small flank incision.



FIG. 17. Closure of a perforated gastric ulcer. a. Interrupted Lembert sutures inserted wide of the ulcer. b. Attached portion of greater omentum laid across perforation and sutures tied over it.

**Gastro-enterostomy.** Simple gastro-enterostomy is done much less commonly now than formerly, but the operation is often combined with vagotomy in the treatment of duodenal ulcer. A loop of jejunum close to the duodenojejunal flexure may be joined either to the back or front of the stomach (Fig. 18). The anterior operation is more often used in the palliation of neoplasm and the posterior one for ulcer. In the latter procedure a hole is made in the transverse mesocolon to the left of the middle colic artery, and the jejunum is anastomosed to the stomach so as to form a vertically disposed stoma (Fig. 19). The edges of the defect in the mesocolon are sutured to the back of the stomach. In the anterior operation, the jejunum is brought in front of the omentum and transverse colon. In both gastro-enterostomy and gastrectomy, the suturing is

usually done with two layers of fine catgut. Some surgeons prefer to use silk or other non-absorbable material for the outer layer which picks up the serous and muscular coats. The inner layer of catgut passes through all coats. Either a simple running or loop-on-the-mucosa stitch may be used.

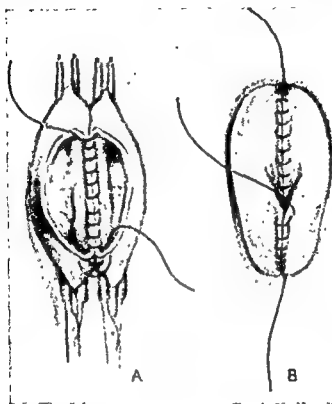


FIG. 18. Gastrojejunostomy. A. Insertion of posterior all-coats suture (previously inserted seromuscular suture not shown). B. Anterior all-coats suture being buried by anterior seromuscular suture.

**Vagotomy.** The left lobe of the liver is mobilised and retracted after dividing the triangular ligament. The peritoneum is incised over the lower part of the œsophagus and the latter is freed all round. Exposure is helped by traction on a length of rubber tubing slung around the lower œsophagus. In most cases there are two main nerve trunks; in about a quarter there are three, and in a few cases there are four. The right vagus lies behind and to the right of the œsophagus and usually a little separated from it, whereas the left is in close apposition anteriorly. A short length of each nerve is excised and the ends are ligated, or silver clips applied in case of bleeding from small vessels on the nerve. The excised lengths of nerve are sent for section and the anatomy is accurately recorded in case of subsequent question about the efficacy of the operation.

**Partial Gastrectomy.** Although many different operations have been

described there are two main prototypes, the Billroth I and Billroth II operations. The latter is never used to-day but developments of it are, and these are usually associated with the name of Polya. Present-day reference to the Billroth operation means that a gastro-duodenal anastomosis has been done, whereas the Polya operation implies closure of the duodenal "stump" and separate end-to-side gastrojejunal anastomosis (Fig. 19). Whichever method is used the curvatures of the stomach are

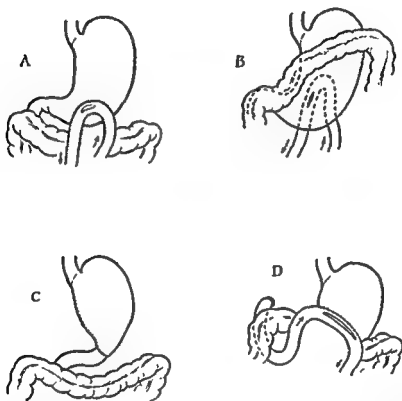


FIG. 19. Gastric operations. A. Anterior, and B. posterior gastrojejunostomy. C. Billroth I. D. Polya type antecolic partial gastrectomy with full width stoma.

cleared first; the left and right gastric and gastro-epiploic vessels are secured. If further mobilisation is required as many of the short gastric vessels as necessary are divided, care being taken to avoid damage to the lower pole of the spleen. With a mobile gastric or duodenal ulcer the procedure is straightforward. A large adherent gastric ulcer may have to be "pinched off" the pancreas, and a large or rather distally situated duodenal ulcer needs very careful dissection to avoid damage to the common bile duct and pancreas. Once the specimen is mobilised the duodenum is divided and subsequently the stomach too is divided to leave roughly a third or a quarter behind. However, after dividing the duodenum the further procedure varies according to whether a Billroth or Polya operation is done. In the Billroth operation the proximal end of

the divided duodenum is joined to the greater-curve side of the stomach, and the remainder of the cut end of the stomach is closed (Fig. 19C). In the Polya operation, the duodenal stump is closed very carefully (a number of different methods being used), and the cut end of the stomach is anastomosed to the antimesenteric border of the upper jejunum (Figs. 19D and 20). Either ante- or retrocolic anastomosis can be done with the afferent loop at greater or lesser curve; and the afferent loop from duodenojejunal flexure to stomach is made as short as it reasonably can be. There is little to choose between the various modifications of procedure although some surgeons are wont to argue their merits endlessly. The gastrojejunal anastomosis can be made the full width of the stomach or with a restricted stoma (Fig. 21A). It is frequently wise to drain the abdomen, especially if there has been a difficult duodenal stump closure. In cases of neoplasm the resection is modified to include an adequate margin of stomach clear of the growth, at least 3 cm. of duodenum and the whole of the greater omentum (Fig. 22B). With a high subtotal resection the spleen and the gastrosplenic omentum are included.

**Total Gastrectomy.** This is almost invariably done for neoplasm although it is very occasionally needed for a massive benign ulcer (Fig. 21B).

(i) *Abdominal Approach.* A vertical incision is used. The resection for neoplasm includes the whole stomach, 3 cm. of duodenum, the greater omentum and the spleen. After division of the vagi at least 5 cm. of œsophagus can be mobilised. The duodenal stump is closed, and the œsophageojejunal anastomosis can be either end-to-side (Fig. 21B) or end-to-end (Fig. 23B). Occasionally it is practicable to join the œsophagus to the duodenum.

(ii) *Abdominothoracic Approach.* When it is necessary to resect more of the lower part of the œsophagus beyond the upper margin of the growth (Fig. 23), the incision is extended into a suitable intercostal space.

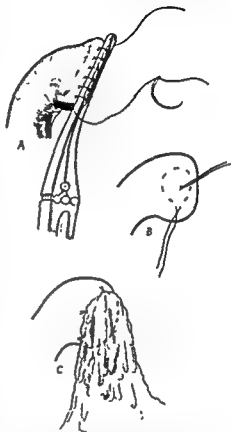


FIG. 20. Gastrectomy. One method of closing the duodenum. A. Inverting stitch inserted over crushing clamp. B. Ends of first suture tightened and tied after removing clamp. Purse-string suture inserted and ready to tie. C. Attached portion of omentum sutured over.



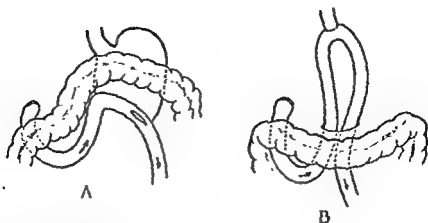


FIG. 21. Gastric operations. A. Polya type retrocolic partial gastrectomy with restricted stoma. B. Total gastrectomy with end-to-side oesophago-jejuno-stomy.

Many variations in the technique of anastomosis are used in joining the oesophagus to the jejunum (Fig. 24). Some have sought to improve results by including the pancreas, the left suprarenal and neighbouring parts in the resection. This may be justifiable in well-selected cases, but its routine practice greatly increases the morbidity and mortality rate.

**Gastrostomy.** The operation of gastrostomy is sometimes required in order to nourish a patient who is unable to swallow. It is more often used as a temporary measure to prepare the patient for some later definitive operation. Formerly it found some favour in the palliation of incurable carcinoma of the oesophagus, but nowadays many surgeons feel that this is an unkind way of prolonging life. Several methods are

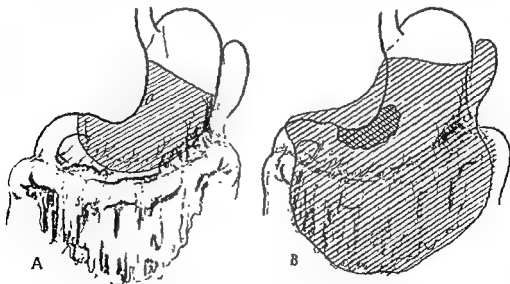


FIG. 22. Partial gastrectomy. To show extent of resection for: A. Benign ulcer, and B. carcinoma.

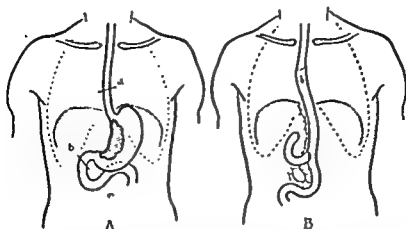


FIG. 23. Total gastrectomy and partial esophagectomy for carcinoma involving cardia. A. Extent of resection (a to b). B. End-to-end esophagojejunostomy with end-to-side jejunojejunostomy to restore continuity of small gut.

used, but in principle they are all similar. The operation can be done under local anæsthetic. A short *left* transrectal incision suffices. The stomach is exposed and a suitable sized self-retaining catheter is inserted; inversion of the opening is secured by two or three purse-string sutures of catgut or by making an oblique track for the catheter. The stomach is held up to the parietes either with a fine stitch or by slipping a little rubber collar on to the catheter at skin level when the wound is closed.

### THE POST-GASTRECTOMY SYNDROME

A great deal has recently been written about the post-gastrectomy syndrome which deserves some mention here. This includes a number of unpleasant sequelæ of varying severity, the chief of which are a faint feeling after food, nausea, vomiting, looseness of the bowels and a feeling of fullness. The symptoms are probably of mixed ætiology and various explanations are given. The possible causes are distension of the duodenal loop, rapid filling of the jejunum, traction on the mesentery, hypoglycæmia and plasma electrolyte changes. At least 50 per cent. of patients never experience any of these symptoms, and the majority of those who do only have them in mild or moderate degree for a few months. Persistent severe symptoms are certainly unusual and it is wise to handle the patient with confident and sympathetic encouragement. Restriction of fluids at meal times

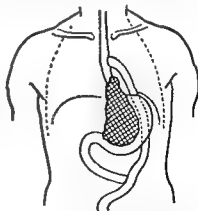


FIG. 24. Inoperable carcinoma involving cardia. Palliative bypass by side-to-end esophagojejunostomy.

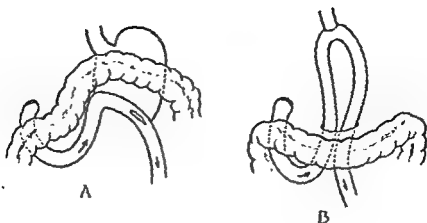


FIG. 21. Gastric operations. A. Polya type retrocolic partial gastrectomy with restricted stoma. B. Total gastrectomy with end-to-side œsophago-jejuno-stomy.

Many variations in the technique of anastomosis are used in joining the œsophagus to the jejunum (Fig. 24). Some have sought to improve results by including the pancreas, the left suprarenal and neighbouring parts in the resection. This may be justifiable in well-selected cases, but its routine practice greatly increases the morbidity and mortality rate.

**Gastrostomy.** The operation of gastrostomy is sometimes required in order to nourish a patient who is unable to swallow. It is more often used as a temporary measure to prepare the patient for some later definitive operation. Formerly it found some favour in the palliation of incurable carcinoma of the œsophagus, but nowadays many surgeons feel that this is an unkind way of prolonging life. Several methods are

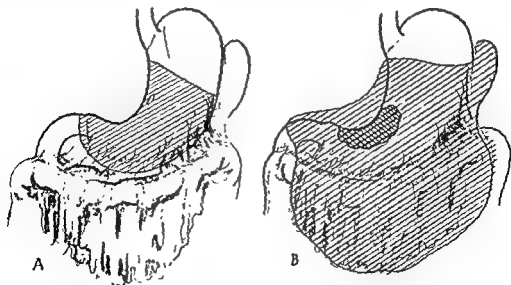


FIG. 22. Partial gastrectomy. To show extent of resection for: A. Benign ulcer, and B. carcinoma.

## CHAPTER 3

### THE GALL BLADDER AND BILE DUCTS

**General Considerations.** The chief functions of the gall bladder are to concentrate and store bile and to deliver it to the duodenum when needed. It is also believed to absorb cholesterol and other lipoids. Bile secreted by the liver is concentrated six- or tenfold in the gall bladder, and a small amount of mucus is added. When drainage from the gall bladder is obstructed for a few days mucus accumulates and bile pigment is either absorbed or oxidised so that the organ fills with clear, colourless fluid (often misleadingly termed "white bile"). With complete obstruction of the common duct, dilatation of the gall bladder delays the onset of jaundice for two or three days. However, if the gall bladder is chronically inflamed and fibrous, or if cholecystectomy has previously been done, complete duct obstruction is more rapidly succeeded by jaundice.

The main stimulus to emptying of the gall bladder is the entry of dilute hydrochloric acid or a fatty meal into the duodenum. Emptying is also stimulated by hypertonic saline and magnesium sulphate. All of these agents stimulate the secretion of cholecystokinase, an enzyme which is formed in the duodenum and upper jejunum and which causes the gall bladder to contract. The various bile ducts contain little smooth muscle in their walls and act largely as passive conductors of bile. However, biliary colic is probably due to muscular contraction of the bile ducts as well as of the gall bladder.

Some authorities ascribe certain biliary symptoms to hypertonia of the bile ducts or the sphincter of Oddi. Manometric pressure readings and cholangiography at operation have been said to confirm these conditions. The greatest circumspection is however necessary in interpreting such findings, and the diagnosis of hypertonia of the bile ducts or sphincter must be accepted with considerable reserve.

**Tests of Function.** Radiological visualisation of the gall bladder following the ingestion of radio-opaque substances which are excreted by the liver and concentrated in the gall bladder has revolutionised the investigation of biliary disease. The opaque substance usually used is *sodium tetra-iodo-phenol-phthalein*, and prior to its introduction by Graham, in 1926, diagnosis was largely dependent on the history and clinical findings. Failure of the medium to outline the gall-bladder on X-ray is presumptive evidence of gall-bladder disease. But non-filling of the gall bladder may be due to the patient's failure to take or absorb the medium, or to liver failure. Sluggish emptying of the gall bladder

and lying down for half an hour or so relieves most patients. Simple modifications in the diet, such as decreasing the carbohydrates, sometimes helps. Important though it is to take an interest in this sort of trouble, the surgeon and the family doctor can, by obsessive interest in it, merely aggravate the patient's anxiety and prolong his suffering. Many surgeons consider it wise to explain the possibility of such symptoms to patients in simple terms before operation. This certainly makes it much easier to handle patients if and when such symptoms subsequently occur.

## CHAPTER 3

### THE GALL BLADDER AND BILE DUCTS

**General Considerations.** The chief functions of the gall bladder are to concentrate and store bile and to deliver it to the duodenum when needed. It is also believed to absorb cholesterol and other lipoids. Bile secreted by the liver is concentrated six- or tenfold in the gall bladder, and a small amount of mucus is added. When drainage from the gall bladder is obstructed for a few days mucus accumulates and bile pigment is either absorbed or oxidised so that the organ fills with clear, colourless fluid (often misleadingly termed "white bile"). With complete obstruction of the common duct, dilatation of the gall bladder delays the onset of jaundice for two or three days. However, if the gall bladder is chronically inflamed and fibrous, or if cholecystectomy has previously been done, complete duct obstruction is more rapidly succeeded by jaundice.

The main stimulus to emptying of the gall bladder is the entry of dilute hydrochloric acid or a fatty meal into the duodenum. Emptying is also stimulated by hypertonic saline and magnesium sulphate. All of these agents stimulate the secretion of cholecystokinase, an enzyme which is formed in the duodenum and upper jejunum and which causes the gall bladder to contract. The various bile ducts contain little smooth muscle in their walls and act largely as passive conductors of bile. However, biliary colic is probably due to muscular contraction of the bile ducts as well as of the gall bladder.

Some authorities ascribe certain biliary symptoms to hypertonia of the bile ducts or the sphincter of Oddi. Manometric pressure readings and cholangiography at operation have been said to confirm these conditions. The greatest circumspection is however necessary in interpreting such findings, and the diagnosis of hypertonia of the bile ducts or sphincter must be accepted with considerable reserve.

**Tests of Function.** Radiological visualisation of the gall bladder following the ingestion of radio-opaque substances which are excreted by the liver and concentrated in the gall bladder has revolutionised the investigation of biliary disease. The opaque substance usually used is *sodium tetra-iodo-phenol-phthalein*, and prior to its introduction by Graham, in 1926, diagnosis was largely dependent on the history and clinical findings. Failure of the medium to outline the gall-bladder on X-ray is presumptive evidence of gall-bladder disease. But non-filling of the gall bladder may be due to the patient's failure to take or absorb the medium, or to liver failure. Sluggish emptying of the gall bladder

following a fatty meal is further evidence of disease. Many patients with non-opaque gall-stones show good concentration in the gall bladder and normal response to a fatty meal. Because sodium tetra-iodo-phenolphthalein is not sufficiently opaque to be seen on X-ray until concentrated by the gall bladder, the bile ducts cannot be demonstrated if the gall bladder has been removed, is non-functioning, or fails to fill due to a blocked cystic duct. The recent development of new agents which are given intravenously (e.g. "Biligrasin"), and secreted in more concentrated form by the liver itself, allow the duct system to be visualised even after removal of the gall bladder.

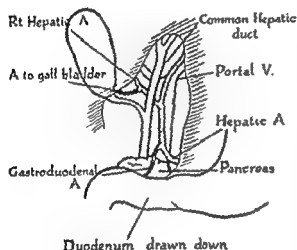


FIG. 25. The normal anatomy of the bile ducts and related vessels.

Successful visualisation of the bile ducts (cholangiography) does, however, depend upon reasonably good liver function.

**Anatomy.** The structure of the normal gall bladder does not need description here, but certain abnormalities are mentioned. Bilobed and double gall bladders, and diverticulum of the organ, are rare anomalies. Rudimentary gall bladder, sometimes no more than 1 cm. long, is occasionally seen. Sometimes the gall bladder has a mesentery and less often it is

buried in the liver. The variations in the anatomy of the bile ducts and neighbouring vessels are of considerable surgical importance (Figs. 25 and 26). The surgeon must know of the various unusual arrangements and remember that extreme vigilance at the time of operation is the best way of avoiding damage to ducts and vessels. The cystic duct is of very variable length and occasionally joins the back or left side of the common hepatic duct. Accessory hepatic ducts not infrequently join the common hepatic duct and even the cystic duct. An accessory duct draining direct into the gall bladder (as opposed to one situated superficially in the liver which may be opened during cholecystectomy) is undoubtedly very rare. Most of the vascular anomalies concern the right hepatic artery and the artery to the gall bladder (Fig. 26).

#### INFLAMMATION OF THE GALL BLADDER

*Cholecystitis* occurs in acute and chronic forms, but the latter is more common, and both forms are much more common in women. Although bowel organisms can gain access to the gall bladder and may be responsible for inflammation, it would seem that other factors too are of importance. Chemical irritation, obstruction, and the presence of stones are

causal factors in some cases. Indeed some authorities suggest that acute cholecystitis is more often due to irritation by excessive bile salt concentration than bacterial infection. In infected cases, *E. coli*, *Cl. welchii*, and non-haemolytic streptococci are usually responsible. Organisms reach the gall bladder via the biliary tract, blood vessels, lymphatics, and possibly direct from the liver.

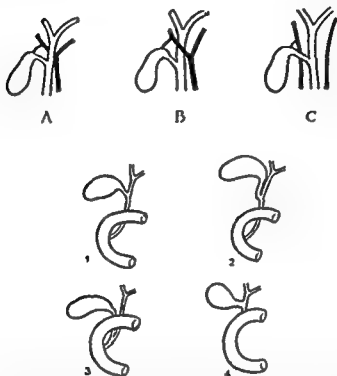


FIG. 26. Some variations in the anatomy of the vessels and ducts. A. Common arrangement with right hepatic artery behind the hepatic duct. B. Artery anterior to duct. C. Unusual origin of the right hepatic artery from the superior mesenteric. Variations in the cystic duct. (1) Normal. (2) Adherent to the common hepatic duct. (3) Long cystic duct passing behind duodenum. (4) Short duct.

### ACUTE CHOLECYSTITIS

This may occur in a previously normal gall bladder or be superimposed on chronic cholecystitis. Whether the inflammation be primarily bacterial or chemical in nature there is no doubt that obstruction of the cystic duct greatly aggravates the condition. Clear-cut examples of bacterial inflammation are seen in those cases complicating typhoid and paratyphoid fever.

**Pathology.** The gall bladder is usually thickened by congestion and oedema. Reddening or ulceration of the mucosa occurs and there may be a fibrinous exudate on the serous surface. Sometimes there is localised necrosis of the wall and occasionally the whole organ becomes gangrenous.



In the latter cases it is possible that the blood vessels around the neck of the gall bladder are obliterated by edema or thrombosis. When the cystic duct is obstructed, the gall bladder either fills up with pus or with a creamy exudate of cholesterol and calcium carbonate. On histological section there may be marked edema of the wall, although this appearance sometimes disappears in the course of fixation. Ulceration of the mucosa is occasionally seen, but neutrophil infiltration of the wall is sometimes slight.

**Clinical Features.** Usually the onset is sudden. Pain occurs in the epigastrium or right hypochondrium and sometimes radiates to the right shoulder or back. In some cases there is a history of dyspepsia and other symptoms due to pre-existent chronic cholecystitis. Marked pyrexia and even rigors may occur in the obstructed variety. Anorexia and nausea or vomiting are common; with obstructive cholecystitis there may be repeated vomiting. Sometimes the severity and catastrophic quality of the pain signify an associated pancreatitis.

On examination the temperature and the pulse rate are often raised and the tongue is frequently furred. There is tenderness below the right costal margin, local rigidity and sometimes a palpable gall bladder, though extreme tenderness and guarding may make it difficult to palpate. The gall bladder is certainly easier to feel if the examiner stands on the left of the patient and palpates with the left hand. In patients with pre-existent chronic cholecystitis the gall bladder is usually indistensible and therefore not palpable. Windy distension of the gut due to aerophagy is not uncommon and is sometimes so marked as to simulate gut obstruction. Jaundice may occur, but is usually mild. It can be due to swelling in the region of the neck of the gall bladder or cholangitis and is sometimes due to an associated stone in the common duct.

**Complications.** *Perforation* of the gall bladder is uncommon and occurs in about 1 per cent. of cases. *Acute pancreatitis* is sometimes seen. *Cholangitis* and hepatic abscess formation (usually multiple) are more likely if there is associated duct disease or pancreatitis. *Subphrenic abscess* is uncommon.

**Differential Diagnosis.** In the milder cases, cholecystitis must be differentiated from *appendicitis*, exacerbation of *duodenal ulcer*, and occasionally from *pyelitis*. More severe cases must be differentiated from *biliary colic* due to a stone in the common duct, *perforated peptic ulcer*, *acute pancreatitis*, and *intestinal obstruction*. A plain X-ray of the abdomen with the patient erect may establish the diagnosis of perforation or obstruction, and some inconsistent clinical features and a raised serum *amylase* facilitate the diagnosis of pancreatitis. Windy distension of the gut is sometimes so extreme that even after X-ray examination, it may be most tempting to diagnose intestinal obstruction. Occasionally the films show a sentinel loop of small gut or a few fluid levels in the small gut adjacent to the gall bladder. An uncommon diagnostic snag is the

patient with a distended gall bladder, felt low in the right iliac fossa, which is mistaken for an *appendix abscess* or even a *twisted ovarian cyst*.

**Treatment.** The great majority of cases of acute cholecystitis settle down without operation and, as perforation is uncommon, most surgeons prefer to treat the patients conservatively in the acute phase. Rest in bed and a light diet suffice for most cases. Gastric aspiration is of value if there is much vomiting or windy distension. Sometimes rectal or intravenous fluids may be needed for the first day or two. Antibiotics are of dubious value, although some surgeons use them routinely. Penicillin attains a low concentration in the gall bladder even when given in large doses. Streptomycin is favoured by some. In severe fulminating infections, or with associated cholangitis causing high temperature and rigors, *terramycin* (oxytetracycline) may be the most suitable agent. However, in the majority of cases, it is likely that antibiotics are both unhelpful and unnecessary.

Operation for acute cholecystitis is indicated in the following circumstances: (1) A tensely distended gall bladder, with increasing pain, pyrexia or rigors. (2) Perforation of the gall bladder. In the United States emergency cholecystectomy in the first forty-eight hours is quite widely practised. There is no doubt that at this early stage of inflammation the operation is not necessarily more difficult; indeed it is frequently easier. However, many of these patients have had previous chronic cholecystitis, and the superadded acute process then makes the operation more difficult. Immediate operation for acute cholecystitis may reveal that the diagnosis is incorrect and that an unnecessary laparotomy has been done for a flare-up of a duodenal ulcer. On the credit side, however, the operator may find that the patient has acute appendicitis and the operation will not have been in vain. Emergency cholecystectomy, especially if undertaken by the inexperienced surgeon, is liable to result in a higher incidence of injury to the bile ducts and vessels. When early operation is indicated, cholecystectomy is not necessarily the procedure of choice. Simple drainage of the gall bladder may be best in: (1) Old or debilitated patients; (2) excessively obese patients; (3) those with perforation of the gall bladder; (4) those with associated acute pancreatitis; (5) those who are so acutely ill as to necessitate the quickest, safest and simplest operation.

### CHRONIC CHOLECYSTITIS

① The exact cause of chronic cholecystitis is not known. Factors such as stasis, infection, chemical irritation and the presence of stones are important, but their exact causal relationship has not been established. Stones are present in the gall-bladder in about three out of four cases, and there are associated stones in more than 90 per cent. of long-standing cases.

**Pathology.** The appearances of the gall bladder are variable. In some

there are only slight changes, or the naked-eye appearance may be within normal limits. On the other hand there may be marked thickening of the wall with consequent reduction in the lumen of the gall bladder. Sometimes there is excessive subserous deposition of fat and adhesion to neighbouring organs such as the liver, duodenum and colon. The histological appearances are variable. Usually the thickened gall bladder wall

is infiltrated with lymphocytes and plasma cells and, if there has been superadded acute inflammation, polymorphonuclear cells may be present too. The congested and oedematous mucosa sometimes ulcerates and is replaced by granulation tissue. In many cases there is outpouching of the mucosa into and through the muscular layer (so-called Rokitansky-Aschoff sinuses). The latter feature may be so marked that it is referred to as cholecystitis glandularis proliferans. Occasionally the wall of the gall bladder is almost completely replaced by fibrous tissue (Fig. 27).

**Clinical Features.** Chronic cholecystitis is by no means confined to fair, fat, stultent, fertile females of forty, as one aphorism would have us believe. It is certainly more common in women and most often occurs in the fourth and fifth decades. Symptoms of indigestion are especially liable to occur after fried foods and pastries. There may be upper abdominal fullness and aching, usually in the epigastrium and right hypochondrium. The pain often radiates to the back and the right scapular region. Heartburn and shoulder pain may occur



FIG. 27. Chronic cholecystitis. One half of a gall bladder showing fibrous replacement of the wall. The small rectangular defect below is where a piece was removed for histology.

and the patient often feels blown up with wind. The appetite is usually unaffected, but occasionally nausea and vomiting occur during the attacks. Jaundice is rarely present with uncomplicated chronic cholecystitis, but is usually due to associated stones. The latter may also cause severe colicky pain.

On examination, pyrexia is usually slight or absent unless there is marked infection and poor drainage of the organ. Tenderness is present below the right costal margin near the fundus of the gall bladder which may be palpable in some cases. Pain, and a catch in the breath during inspiration, may be elicited below the ninth costal cartilage (Murphy's

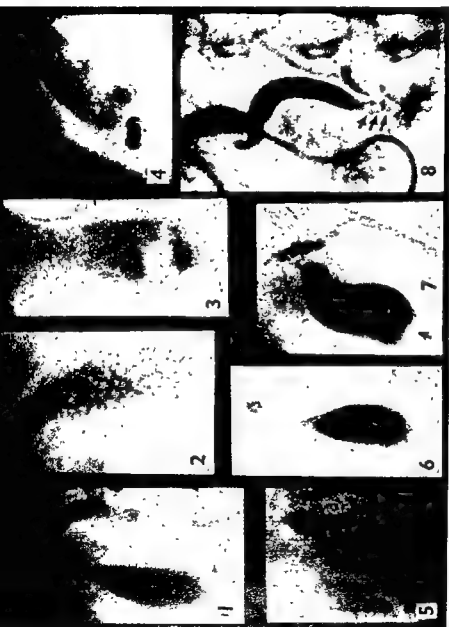


FIG. 28. Biliary tract X-rays. (1) Oral cholecystography. Gall bladder contracted after fatty meal and common duct thereby filled. (2) Multiple radio-opaque gallstones. (3) Non-opaque gall-stones floating in fundus visualised by cholecystography. (4) Laminated opaque gall-stones. (5) Calcification in gall-bladder wall. (6) "Limy" bile; the gall bladder is outlined by bile of high calcium content. (7) Polyp at fundus. (8) Post-operative cholangiogram through a T-tube. Three stones are present at lower end of common duct and there is reflux of medium into the pancreatic duct.  
(Courtesy of Dr. L. S. Carslaw.)

sign). Plain X-ray of the abdomen sometimes reveals opaque stones (25 per cent. of cases). A positive diagnosis can usually be made on cholecystography. Disease of the gall bladder is indicated by : (1) Non-filling of the organ ; (2) poor concentration of opaque medium in the gall bladder with delayed and incomplete emptying ; (3) filling defects in the opaque medium due to the presence of non-opaque stones (Fig. 28).

**Differential Diagnosis.** The symptoms may be confused with those caused by *peptic ulcer*, *hiatus hernia*, *subacute recurrent appendicitis*, *pancreatitis* and *cardiac disease*. Duodenal ulcer symptoms usually have

a characteristic periodicity, and tend to occur at night, features which are unusual in biliary disease. However, atypical peptic ulcer symptoms may easily be confused with those of chronic cholecystitis. Barium meal X-ray examination assists diagnosis ; peptic ulcer craters and hiatus hernia are readily demonstrated by an expert radiologist. A chronically inflamed gall bladder which is adherent to the duodenum may deform the latter, but deformity alone should never be accepted as evidence of active peptic ulcer. It is not uncommon for peptic ulcer or hiatus hernia to be coexistent with biliary disease ; the possibility of multiple pathology must therefore always be considered. Atheroma (atherosclerosis) of the coronary arteries, with resultant myocardial ischaemia, may cause symptoms very suggestive of biliary disease and, conversely, the latter may simulate heart disease.

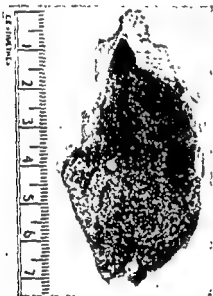


FIG. 29. Cholesterolosis. The numerous white specks are due to cholesterol deposits in the mucosa of the gall bladder.

#### CHOLESTEROLOSIS OF THE GALL BLADDER

Cholesterolosis is characterised by lipid deposits in the mucosa of the gall bladder (Fig. 29). When the mucosa is red and studded with little yellow flecks of cholesterol, the term "*strawberry gall bladder*" is appropriate. Such changes may be diffuse or localised and associated with cholesterol stones or chronic inflammation. It is possible that some cholesterol stones form as a result of detachment of these mucosal deposits. Cholesterolosis may cause similar symptoms to those of chronic cholecystitis.

#### STASIS GALL BLADDER

There are some patients who have symptoms distinctive of biliary trouble without definite radiological or operative evidence of disease.

The term *biliary dyskinesia* or *stasis gall-bladder* is sometimes applied to these cases in which the organ contains rather thick dark bile and empties sluggishly. There are certainly some patients in this group in whom both symptoms and X-ray evidence remain equivocal for several years, but in whom definite disease eventually occurs. Although some surgeons have advocated cholecystectomy for stasis gall bladder it is usually prudent to observe these cases for some time before being tempted to operate. Although symptoms are occasionally relieved, it is true to say that they are especially liable to persist post-operatively in those cases with minimum pathology. Experience in this latter type of case has given origin to the witticism that "the only cure for the post-cholecystectomy syndrome is to put the gall bladder back again!"

### GALL-STONES

There are three principal types of gall-stone :

(1) Pure cholesterol; (2) pure pigment; (3) mixed. The majority of stones form in the gall bladder itself, but primary stone formation in the bile ducts does occasionally occur. The incidence of gall-stones increases with age; they are about three times as common in women as in men and they quite commonly occur in adult diabetic patients. *Cholesterol Stones*.—Such stones may first appear in a normal gall bladder, but with subsequent infection calcium bilirubin may be deposited on them. When this sequence occurs the later product is called a combination

stone. Pure cholesterol stones are often large and sometimes solitary. The cut surface has a crystalline structure with radiating lines on it. Cholesterol stone formation is sometimes related to high serum cholesterol concentration, but alteration in the bile acids, which normally hold the cholesterol in solution, may also be a factor. *Pure Pigment Stones*.—Such stones are usually multiple, tiny, black and brittle. They result from increased red cell destruction, and are a common complication of familial hæmolytic anæmia. Both pure pigment and cholesterol stones are non-opaque to X-rays. *Mixed Stones*.—These are the commonest and constitute about four-fifths of all gall-stones. Usually multiple, they tend to be cuboid and faceted (Fig. 30). The cut surface shows concentric lamellæ of protein material with cholesterol, calcium carbonate and pigment incorporated in its structure. Various combinations of these three main varieties of gall-stone may occur.

**Mechanism of Stone Formation.** Whereas metabolic factors play a part in the formation of cholesterol and pigment stones, the mixed type is



FIG. 30. Chronic cholecystitis with multiple large and small stones.

essentially due to infection. Stasis of bile is a possible contributory factor and each of these factors may act either independently or together at different phases of development. Cholesterol and bile pigments are normally in solution in bile, but are precipitated in the presence of infection. A decrease in the bile salt concentration also favours precipitation of cholesterol. High blood cholesterol concentrations occur in pregnancy, diabetes, and in association with extensive atherosclerosis. In all of these conditions there is an increase in the bile cholesterol, and stone formation is common.

**Clinical Features.** Stones are quite often discovered accidentally and this is especially true of the metabolic variety. However, the patient with mixed stones has associated infection, and either the cholecystitis or the stones may be responsible for symptoms. It will be appreciated that cholecystitis may either precede or succeed stone formation, and the symptomatology of that condition needs no further discussion. Attempts to pass stones through the cystic duct and bile ducts give rise to sharp colicky pain. Large stones, in the absence of infection, are commonly symptomless, for it is impossible for them to enter the cystic duct. Jaundice can be due either to a stone in the common bile duct or to one in Hartmann's pouch which is pressing on the main ducts. Characteristically the jaundice due to a stone waxes and wanes, but sometimes there is steadily increasing jaundice which is suggestive of a neoplasm. In most of these cases there is associated cholecystitis and the gall bladder is thickened. For this reason it is rare for the gall bladder to be palpable, for it is relatively indistensible.

Gall-stones occasionally ulcerate through into neighbouring organs such as the duodenum or colon. In the former case, a large stone may impact in the lower part of the ileum and cause intestinal obstruction. Sometimes a relatively large stone in the gall bladder ulcerates into the common hepatic duct or common bile duct with much distortion of the normal anatomy, as seen at the time of operation. Apart from this type of *internal biliary fistula*, *external biliary fistula* may also occur if the inflamed gall bladder becomes adhered to the abdominal wall and then discharges its contents through the skin.

**Treatment of Chronic Cholecystitis and Stones.** There are many patients with mild symptoms from chronic cholecystitis who do not seek or require operative relief. The surgeon should confine his attentions to those with more severe symptoms many of whom have associated stones. The patient with repeated attacks of severe pain and vomiting requires little persuasion to have an operation. In those with rather less severe and less frequent symptoms it is wise to emphasise the possibility of increasing trouble and to advise operation, but never to press the patient unduly.

Although patients with long-standing chronic cholecystitis and stones are at risk for carcinoma, fear of this disease should not become the overriding consideration in all cases. For example, an elderly and unfit

patient in whom a silent and solitary stone in the gall bladder is accidentally discovered on X-ray examination is best left alone. However, with symptomless disease of this kind in a younger patient, and especially if there are multiple small stones, the possibilities of later malignancy may reasonably obtrude more in advocating operation.

Chronic cholecystitis and calculous disease confined to the gall bladder is dealt with by cholecystectomy. The indications for exploring the common duct at the time of cholecystectomy are discussed later. Stones in the common bile duct following cholecystectomy have generally been overlooked at the time of operation. Nevertheless, with persistent cholangitis, there is no doubt that fresh stone formation (often combinations of calcium salts and bilirubin) may occur. It is in the investigation of such cases that intravenous cholangiography is sometimes so useful. It should be emphasised that in cases of *overlooked stone* there may be recurrent colic over long periods of time without any jaundice. Those patients with *recurrent stone* formation in an infected duct more often have attacks of pain, pyrexia and jaundice. All such cases will require exploration of the common duct and removal of the stones. In patients with associated cholangitis causing pyrexia, rigors and jaundice, it is wise to give antibiotics. Quite apart from cholangitic abscess formation in the liver, infection and obstruction may lead to serious impairment of liver function after operation and even result in hepato-renal failure.

### TUMOURS OF THE BILIARY TRACT

**Simple Tumours.** These are not of much surgical importance and are generally discovered in the course of operation. *Adenomas* are usually small. *Simple papilloma* and *lipoid papilloma* (a stage of cholesterosis) are more common.

**Malignant Tumours.** *Carcinoma of the Gall bladder.*—This is not uncommon in cases of long-standing calculous cholecystitis. Indeed, stones are present in some 95 per cent. of cases of carcinoma of the gall bladder. Rarely carcinoma seems to develop from a simple adenoma. *Pathology.*—The growth is usually an adenocarcinoma, and may occur in invasive or papillary forms. Colloid degeneration is occasionally seen. Squamous celled carcinoma is rare. The tumour readily spreads into the neighbouring liver and bile ducts, and at operation it is not unusual to find a large and irremovable mass. *Clinical Features.*—Few cases occur under the age of fifty and the disease is three or four times more common in women. Often there is a long history of indigestion or biliary symptoms, but sometimes the onset is rapid. Flatulent dyspepsia, typical cholecystitis symptoms and jaundice may occur. In some cases there is well-marked upper abdominal "girdle" pain. There may be nothing specific on clinical examination, or a palpable mass and hepatomegaly may be present. Although the cholecystogram usually shows a non-functioning gall bladder there is generally no specific clue to diagnosis on X-ray.



essentially due to infection. Stasis of bile is a possible contributory factor and each of these factors may act either independently or together at different phases of development. Cholesterol and bile pigments are normally in solution in bile, but are precipitated in the presence of infection. A decrease in the bile salt concentration also favours precipitation of cholesterol. High blood cholesterol concentrations occur in pregnancy, diabetes, and in association with extensive atherosclerosis. In all of these conditions there is an increase in the bile cholesterol, and stone formation is common.

**Clinical Features.** Stones are quite often discovered accidentally and this is especially true of the metabolic variety. However, the patient with mixed stones has associated infection, and either the cholecystitis or the stones may be responsible for symptoms. It will be appreciated that cholecystitis may either precede or succeed stone formation, and the symptomatology of that condition needs no further discussion. Attempts to pass stones through the cystic duct and bile ducts give rise to sharp colicky pain. Large stones, in the absence of infection, are commonly symptomless, for it is impossible for them to enter the cystic duct. Jaundice can be due either to a stone in the common bile duct or to one in Hartmann's pouch which is pressing on the main ducts. Characteristically the jaundice due to a stone waxes and wanes, but sometimes there is steadily increasing jaundice which is suggestive of a neoplasm. In most of these cases there is associated cholecystitis and the gall bladder is thickened. For this reason it is rare for the gall bladder to be palpable, for it is relatively indistensible.

Gall-stones occasionally ulcerate through into neighbouring organs such as the duodenum or colon. In the former case, a large stone may impact in the lower part of the ileum and cause intestinal obstruction. Sometimes a relatively large stone in the gall bladder ulcerates into the common hepatic duct or common bile duct with much distortion of the normal anatomy, as seen at the time of operation. Apart from this type of *internal biliary fistula*, *external biliary fistula* may also occur if the inflamed gall bladder becomes adhered to the abdominal wall and then discharges its contents through the skin.

**Treatment of Chronic Cholecystitis and Stones.** There are many patients with mild symptoms from chronic cholecystitis who do not seek or require operative relief. The surgeon should confine his attentions to those with more severe symptoms many of whom have associated stones. The patient with repeated attacks of severe pain and vomiting requires little persuasion to have an operation. In those with rather less severe and less frequent symptoms it is wise to emphasise the possibility of increasing trouble and to advise operation, but never to press the patient unduly.

Although patients with long-standing chronic cholecystitis and stones are at risk for carcinoma, fear of this disease should not become the overriding consideration in all cases. For example, an elderly and unfit

liver. The gall bladder bed is reperitonised with fine catgut ; and the stump of the cystic duct is buried at the same time. Sometimes reperitonisation of the gall bladder bed is neither practicable nor necessary. A corrugated rubber drain is laid along the gall bladder bed down to the stump of the cystic duct. The drain is brought out through the lateral end of the subcostal incision or, if a vertical incision has been used, at any other suitable site, and is usually left in place for two to three days.

**Complications.** The most serious accidents to occur during operation involve damage to the hepatic arteries and main bile ducts. Extreme care and vigilance at the time of operation is the only way to avoid or minimise

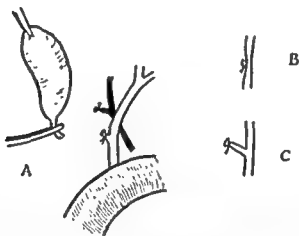


FIG. 31. Cholecystectomy. A. Correct technique. The right hepatic artery is identified before ligating the artery to the gall bladder. The cystic duct is tied flush with the common bile duct. B. Care is taken to avoid tying the cystic duct too close and thus narrowing the common duct. C. Too long a stump of cystic duct may result in stone formation in the residuum.

such damage. If damage to the ducts does occur it is vitally important that it shall be clearly recognised and remedied at the time. Duct damage can occur in even the most skilled hands, but it is usually the unskilled surgeon who fails to recognise that such damage has occurred. Post-operative complications include *paralytic ileus*, *deep venous thrombosis* and *pulmonary embolism*. If the first is suspected gastric aspiration should be carried out. *Persistent biliary fistula* is generally due to damage to the bile ducts. Pyrexia and jaundice may be due to the same cause or to an overlooked stone in the common bile duct. *Stricture of the common bile duct* is a late sequel, usually resulting from damage to the ducts, but occasionally due to extravasation of bile and subsequent periductal fibrosis.

**Cholecystostomy.** The indications for drainage of the gall bladder in cases of acute cholecystitis have already been referred to. The incision is the same as for cholecystectomy. If the gall bladder is tense and distended it may be evacuated through a needle and sucker. Contained

*Treatment.*—The prognosis in these cases is very bad; few are cured surgically. In the majority the disease is already advanced at the time of operation, and resection is impossible. Occasionally the tumour is sufficiently localised for removal to be practicable and, in a small number, the disease is unexpectedly discovered by the pathologist in a cholecystectomy specimen. It is in these latter cases of "accidental" removal of a carcinoma that the best results are achieved. Lately, attempts have been made to deal with these carcinomas by resection of a part, or even the whole, of the right lobe of the liver with the gall bladder. Provided the diagnosis is not in doubt such radical surgery is a reasonable proposition in suitable cases.

*Carcinoma of the Bile Ducts.* This is usually similar in character to carcinoma of the gall bladder. Excepting for those at the ampulla of Vater (see Pancreas), successful removal of these growths is quite exceptional. Jaundice, vague upper abdominal symptoms and pyrexia are frequent. The gall bladder is enlarged and palpable when the tumour obstructs the common bile duct, but enlargement of the liver is the only frequent abdominal sign when the common hepatic duct is involved.

#### OPERATIONS ON THE BILIARY TRACT

*Cholecystectomy.* This is one of the commonest upper abdominal operations and, although a cholecystectomy can be a most straightforward procedure, there are few operations which demand a greater degree of care and precision in difficult cases. Either a right paramedian or right subcostal (Kocher) incision may be used. Most surgeons prefer the latter, but a vertical incision is certainly wise when the exact diagnosis is in doubt, and other procedures may be necessary. In very fat patients, some prefer a more or less transverse incision. The abdomen is carefully explored; packs are placed below, medially and, if necessary, laterally, and the gall bladder and bile ducts are displayed by suitable retraction. The fundus of the gall bladder is held in a tissue forceps or haemostat and, after clearing any adhesions, a second forceps is applied nearer the neck of the gall bladder. The peritoneum overlying the junction of cystic, hepatic and common bile ducts is incised and the trinity of these ducts is clearly displayed. Then, and only then, is a ligature passed around the cystic duct and tied close to its junction with the common bile duct. The distal part of the cystic duct is clamped and divided, and the surgeon is then ready to carry the dissection of the gall-bladder forwards. It is usually at this stage that the cholecystic artery is identified, ligated and divided; especial care is taken to avoid damage to the right hepatic artery which is sometimes very close to the gall bladder (Fig. 31). When the cholecystic artery arises from some source other than the right hepatic artery it frequently lies in front of the ducts and is secured at an earlier stage of the operation. The peritoneum on either side of the gall bladder is, where possible, neatly incised before peeling the gall bladder off the

minimise the incidence of negative exploration of the bile ducts some surgeons have advocated the routine use of *cholangiography* on the operation table. As soon as the abdomen is opened and preliminary exploration completed a needle or fine catheter is inserted into the cystic duct and a suitable radiopaque medium (such as 30 per cent. diodone) is injected. However, although such X-rays are occasionally of help, it is



FIG. 33. Exploration of the common duct. Trinity of ducts displayed, duodenum held downward, stay sutures inserted and common duct incised.

not uncommon for a stone to be missed in the X-ray films, and this procedure does not overcome the need for very careful palpation, and, if necessary, incision and exploration of the bile ducts. A properly performed exploration of this type should do no damage and will certainly disclose stones which would otherwise be missed.

So far as the technique of exploration of the bile ducts is concerned, once the trinity of ducts is displayed, two fine stay sutures are inserted into the adventitia of the common duct at a suitable place between the cystic duct junction and the duodenum (Fig. 33). The wall of the duct is incised carefully between the stay sutures. The lumen is explored with

stones are removed and a drainage tube is inserted and fixed at the fundus with a purse-string suture of catgut (Fig. 32). To prevent leakage of bile or herniation of gut close to the stoma the drainage tube may be brought through the greater omentum.

**Exploration of the Bile Ducts.** Incision of the common duct prior to exploration is called *choledochotomy*, and if T-tube drainage is established afterwards this is referred to as *choledochostomy*. The bile ducts are most



FIG. 32. Cholecystostomy. Tube in fundus of gall-bladder. Purse-string suture inserted but not yet tied.

often explored at the time of cholecystectomy. Some surgeons explore the common bile duct much more frequently than others, and the indications for this procedure vary in different hands as well as in different circumstances. The only absolute indication for exploring the common duct is when stones can be felt there, or their presence is strongly suspected. However, apart from this, there are a number of other occasions on which exploration may be indicated. A greatly dilated common duct (especially if thickened and containing infected turbid bile), demands exploration, but sometimes the common duct is dilated simply because it is storing bile in the presence of an infected and non-functioning gall bladder. Exploration is also called for if there is a history of repeated jaundice, especially if sure palpation of the bile ducts is difficult. The patient who is jaundiced at the time of operation also needs duct exploration. In attempts to

by antibiotics so far as the local condition is concerned and the incidence of perforation and empyema is unchanged. However, when severe systemic symptoms are present as a result of infection these are often alleviated. The occasional case of non-obstructive cholecystitis may even be cured by antibiotics, but this type of infection is uncommon. Antibiotic therapy should therefore be reserved for the serious cases with high fever. The antibiotics are an adjunct to surgery and in no way alter the usual indications for operation. In obstructive jaundice the antibiotics are of no use *within the biliary tract* until obstruction is relieved. They may be of value in protecting the liver from ascending infection and do relieve severe systemic reactions. Preoperative antibiotic therapy is indicated in the latter cases both to facilitate preparation and to ensure safer conduct afterwards.

**Choice of Antibiotic.** Since sulphonamides are somewhat toxic to the liver, they are best avoided now that so many other preparations are available. Most biliary tract infections are due to *B. coli*, or to *streptococci*, *clostridia* and *staphylococci*. Due to the frequency of *B. coli* infection, a fair proportion of these cases should respond to streptomycin. However, in view of the impracticability of sensitivity tests in many cases, there is much to be said for the use of a broad spectrum antibiotic such as *oxytetracycline*. More specific preoperative antibiotic treatment may be possible if a specimen of bile is collected by duodenal intubation and a dose of magnesium sulphate is given. Smear and culture of the duodenal aspirate will provide information about the organisms in the bile in a high proportion of cases provided that obstruction to the duct is not complete.

### STRICTURE OF THE BILE DUCTS

Stricture of the bile ducts may be congenital or acquired, but the latter is infinitely more common. The causes may be simply classified as follows :

1. **Congenital.** *Atresia* or *stenosis*.
2. **Acquired.** (a) *Post-operative.* Usually cholecystectomy or duct exploration. Occasionally after gastrectomy, etc.  
(b) *Accidental Trauma.*  
(c) *Inflammation.* Cholangitis. Fibrosis around duodenal ulcer.  
(d) *Malignant Disease.*

Of the acquired strictures most are due to operative trauma, and the majority of these cases follow cholecystectomy. Less commonly stricture follows exploration of the common duct or partial gastrectomy (especially for removal of a massive or distal duodenal ulcer). Either blunt or penetrating accidental injuries to the abdomen may rarely damage the bile ducts and cause subsequent stricture. Inflammatory causes are less common than operative ones, but stricture occasionally follows stone in the duct, cholangitis, the organisation of a periductal collection of bile

special forceps or silk web bougies ; the greatest care and gentleness is exercised. Some prefer to use malleable metal probes and scoops, but these are liable to be more traumatic to the ducts. A silk web bougie can be passed down into the duodenum and, by feeling carefully along its course, stones are usually readily felt. After removal of stones from the duct (*choledocholithotomy*) it is usual to insert a soft latex rubber T-tube of suitable calibre, and to close the wound in the duct with fine catgut sutures (Fig. 34). A further corrugated drain is inserted down to the site of the wound in the common duct. Quite exceptionally a stone is impacted at the lower end of the common duct and cannot be "milked" upwards.

In these circumstances, the second part of the duodenum may have to be incised and the stone extracted from the ampulla of Vater (*transduodenal choledocholithotomy*). An alternative and even less common procedure is to mobilise the second part of the duodenum, locate the impacted stone by palpation and then incise the overlying pancreas and duct wall (*retroduodenal choledocholithotomy*). Following these last two procedures T-tube drainage of the supraduodenal part of the duct is established.

**Choledochoduodenostomy.** End-to-side anastomosis of the common

duct and duodenum is sometimes used in the relief of stricture of the common bile duct. Side-to-side anastomosis is an alternative procedure which is sometimes done in those patients with recurrent stone formation in an infected duct. Although, theoretically, the absence of an effective sphincter may lead to regurgitation from the duodenum and consequent cholangitis, in actual practice cholangitis is uncommon. Furthermore, it may be better to run the small risk of infection than expose the patient to the possibility of a subsequent biliary operation for fresh obstruction. Regurgitation is preferable to the accumulation of infected debris in a poorly drained common duct.

#### ANTIBIOTIC THERAPY IN BILIARY DISEASE

It is of some importance to consider the place of antibiotic therapy in the surgery of the biliary tract. All soluble sulphonamides and antibiotics are excreted in the bile in high concentration, but a patent cystic duct is essential to their entry into the gall bladder. With an obstructed common bile duct or impaired liver function little or no excretion of these substances takes place. Acute obstructive cholecystitis is uninfluenced

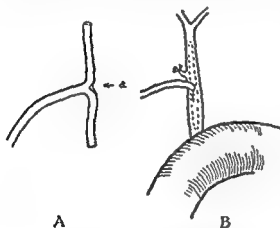


FIG. 34. Drainage of the common duct. A. T-tube ready for insertion with notch cut out at (a) to facilitate removal. B. T-tube in situ.

## CHAPTER 4

### THE PANCREAS

From the surgical viewpoint it should be remembered that the pancreas is a retroperitoneal organ which is closely applied to the structures behind it and intimately related to most of the duodenum (Fig. 36). These factors greatly favour the ready spread of malignant disease, and render the eradication of such disease extremely difficult. The accessory pancreatic duct is occasionally damaged when the duodenum is cleared in the course of gastrectomy, and the consequences of such injury are likely to be more serious when, as is sometimes the case, that duct drains

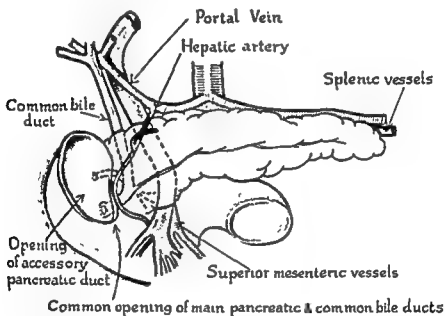


FIG. 36. Anatomy of pancreas.

a large part of the gland. The main pancreatic and common bile ducts usually drain through a common opening and the anatomical arrangement may sometimes be of significance in the production of acute pancreatitis (Fig. 37).

Apart from variations in the duct system of the pancreas, there are other congenital abnormalities. *Aberrant pancreatic tissue* is not uncommon, and is most often related to the duodenum, stomach or a Meckel's diverticulum. It is rarely seen in a wide variety of other situations in the abdomen and usually consists of a flattened yellowish plaque



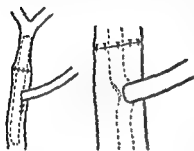
or inflammation and fibrosis around a duodenal ulcer. Malignant causes have already been discussed (see Carcinoma of Bile-ducts).

**Clinical Features.** In post-operative cases, there is often biliary discharge from the wound, persistent pyrexia and steadily increasing jaundice. The obstruction is sometimes partial and the symptoms are intermittent; thus there are phases of increasing jaundice and pyrexia which are succeeded by discharge from a biliary fistula and partial relief. In total

obstruction the jaundice becomes steadily deeper and, in the absence of a biliary fistula, the back pressure results in progressive liver failure and biliary cirrhosis. The latter may on occasion give rise to portal hypertension. If the condition is unrelieved the patient with duct obstruction rarely survives more than one or two years and it is surprising that even this is possible.

**Treatment.** Operations for the relief of biliary stricture are amongst the most difficult in the whole field of abdominal surgery and, even after successful repair, there is still the distinct possibility of further trouble.

FIG. 35. Repair of the common duct. End-to-end anastomosis with T-tube inserted through separate opening.



Prevention of most of these surgical disasters is best ensured by the exercise of extreme care at the time of cholecystectomy and other operations in the region of the ducts. If obstruction is present at the lower end of the common bile duct, an end-to-side or side-to-side *choledochoduodenostomy* may be done. As an alternative the duct can be joined to the jejunum. If a stump of duct is present below (and this is usually so), an end-to-end repair of the common duct can be effected (Fig. 35). In many of the post-operative cases, a length of duct is missing and the ends must be suitably mobilised to close the gap.

When the common hepatic duct has been damaged high up, it is sometimes necessary to anastomose the right and left hepatic ducts to the single distal duct. Alternatively in such cases, when much of the duct is missing, the hepatic ducts can be anastomosed to the duodenum (*hepatoduodenostomy*). Various prostheses such as tantalum, polythene and other plastic materials have been used to bridge big gaps. None of these materials is entirely satisfactory as there may be subsequent blockage by biliary debris or scar formation. The details of these complicated procedures are not discussed here. The large variety of operations used is partly due to the diversity of lesions treated, and partly to the fact that many have not proved entirely satisfactory.

complicate operation for their relief. Whereas that variety of pancreatitis complicating *mumps* is due to a blood-stream infection there is little evidence that other types are acquired in this fashion.

**Pathology.** It was common to subdivide acute pancreatitis into suppurative, gangrenous and hæmorrhagic varieties, but these are merely stages in the more extreme forms of the disease or manifestations of different degrees of severity. In many cases the inflammation is quite mild and characterised by swelling, congestion and induration of the organ. Sections of the pancreas show only slight necrosis in these cases. On the other hand, there may be massive necrosis of tissue with sloughing of large fragments of the gland. Extensive hæmorrhage into the pancreas, retroperitoneal tissues and mesentery is seen in some cases, and there may be blood-stained exudate into the greater and lesser sacs. The liberated pancreatic lipase may cause quite extensive fat necrosis in the form of whitish firm patches in the great omentum, subperitoneal fat and mesentery. These changes occur mainly around the pancreas but are sometimes seen outside the abdominal cavity, e.g. in the mediastinum. It is likely that the lipase is carried to these more distant sites by the blood and lymph vessels. The patches of fat necrosis tend to organise and calcify and the serum calcium is often lowered; indeed the depletion of the serum calcium may be so marked as to cause *tetany*.

**Clinical Features.** The sex incidence of this disease varies in different countries; in Great Britain it is rather more common in women. It is more frequent in obese and alcoholic subjects, and there is commonly a history of gall-bladder disease. The fulminating type of disease causes a dramatic clinical picture with sudden onset of catastrophic upper abdominal pain which frequently radiates to the back and to both shoulders. Profound collapse, low blood pressure, sweating and pallor or cyanosis are only occasionally seen, though they feature large in many accounts of this disease. However, there is sometimes rapid death from shock. Vomiting is common at the onset and is frequently persistent. A little blood is sometimes present in the vomitus or may be aspirated from the stomach.

The pulse may be slow at the onset, but usually becomes more rapid with the development of peritonitis and associated toxæmia. The profound toxæmia occasionally seen is believed to be due to fluid and electrolyte depletion and to the absorption of protein breakdown products and bacterial infection. Characteristically, the abdominal signs are frequently much less marked than the symptoms. There is usually upper abdominal tenderness and rigidity, but the latter is rarely so marked as in cases of perforated ulcer. Bowel sounds are usually present, but some degree of paralytic ileus is not uncommon after twenty-four hours. Plain X-ray of the abdomen sometimes shows one or two distended loops of upper jejunum in the early stages, and this may be succeeded by more marked distension later.

of tissue. Once seen such tissue is usually readily recognised on subsequent occasions. *Annular pancreas* is a rare condition, believed to be due to failure of rotation of the anterior pancreatic bud. It occasionally produces obstruction to the second part of the duodenum. *Congenital fibrocystic disease* is referred to in Chap. 19 in connection with *meconium ileus*.

### PANCREATIC INJURIES

Damage to the pancreas is uncommon and may be due to accidental or operative trauma. Accidental damage more often occurs in association

with injury to other abdominal viscera. There may be extravasation of blood and pancreatic ferments and formation of a pancreatic cyst, abscess or fistula. Operative injury sometimes occurs in the course of gastrectomy, and may be followed by a fistula or pancreatic necrosis and pancreatitis. In many of these cases it is difficult to recall any specific damage during the operation, though in some it may be caused by injury to the accessory duct as indicated above.

### ACUTE PANCREATITIS

Severe types of pancreatitis are much less common than mild ones, and it is likely that many of the latter are overlooked owing to the diagnosis not being considered. The importance of severe pancreatitis lies in the dangerous nature of the disease and in differentiating it from other acute abdominal conditions.

**Aetiology.** The condition is due to necrosis and enzymatic auto-digestion of the pancreas. A number of theories have been advanced to explain this phenomenon but none of them provides a satisfactory explanation of all cases. The trypsinogen in the pancreas may be activated by intestinal juice or infected bile, and it has been suggested that these may regurgitate into the pancreatic duct. Stone in the ampulla of Vater is a possible cause of biliary regurgitation. Spasm of the sphincter of Oddi may be of importance, although some say that it is commonly unsuitably placed to favour the passage of bile into the pancreatic duct. It is possible that simple blockage of the pancreatic ducts by stones, inspissated secretion and shed epithelium may cause rupture of acini with consequent extravasation of enzymes. Pancreatitis is sometimes due to a penetrating gastric or duodenal ulcer, or may

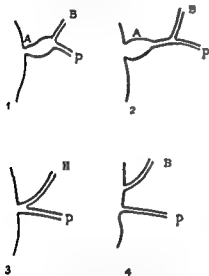


FIG. 37. Termination of common bile and pancreatic ducts. (1) Common arrangement with both ducts entering ampulla. (2) Ducts unite proximal to ampulla. (3) Ducts join at duodenum. (4) Ducts join duodenum separately.

imbalance; (4) gastric aspiration; (5) control of infection; (6) control of any associated diabetes or tetany. *Pain*, if not too severe, can be relieved with simple analgesics, or atropine and amyl nitrite. Although morphine and pethidine, on account of their effect on the sphincter of Oddi, may seem theoretically unsuitable there is every justification for their use when the pain is severe. *Shock* may be relieved by intravenous transfusion of blood or plasma substitutes. In fulminating cases of haemorrhagic pancreatitis, the degree of shock is profound and the mortality rate was formerly very high. These patients, however, often respond dramatically to cortisone. In gravely ill patients a rapid effect can be produced with intravenous hydrocortisone hemisuccinate, but for normal therapy prednisone 25 mg. daily may be given orally for five to ten days during the acute phase. It is wise to continue with gradually decreasing dosage for about an equal period of time.

*Fluid and electrolyte loss* from vomiting, paralytic ileus and peritoneal exudation may be considerable. The consequent dehydration and electrolyte disturbance must be carefully corrected after daily or more frequent estimation of the blood chemistry. Intravenous therapy is essential for all severe cases and a detailed fluid balance chart must be maintained. *Gastric aspiration* not only prevents vomiting but also reduces the hormonal stimulus to pancreatic secretion. It is wise to maintain aspiration during the really acute phase and to withhold oral fluids or reduce them to an absolute minimum during this period. The neurogenic stimulus to pancreatic secretion can be checked by the use of banthine or atropine. *Control of infection* by antibiotics is important. A broad spectrum antibiotic such as oxytetracycline is probably best and, although there are theoretical advantages in giving it by mouth, this is sometimes impracticable in the first day or two. A dose of 500 mg. six-hourly is given by intravenous injection and oral therapy is begun as soon as practicable. *Diabetes and tetany* are both uncommon although, as already indicated, the serum calcium is often low in the first few days. In the event of hyperglycaemia and acidosis the surgeon should seek the collaboration of a physician, as the skilful control of any associated diabetes is important. If tetany occurs it may be relieved with calcium gluconate; approximately 15 ml. of a 10 per cent. solution should be given for each 1 mg. per cent. depression of the serum calcium level.

### CHRONIC PANCREATITIS

All degrees of chronic pancreatitis from mild fibrosis to massive scarring may occur. The diagnosis of chronic pancreatitis can be most difficult on clinical grounds. Chronic cholecystitis and stones are present in a fair proportion of cases and it is possible that some cases result from previous acute pancreatitis.

**Pathology.** The disease may be diffuse or localised and, if the head of

Other features may develop after a few days. *Bluish discoloration of the skin* in the left flank or around the umbilicus is a manifestation of the hæmorrhage and extravasation around the pancreas. The umbilical discoloration is only possible in the small proportion of people who, having no periumbilical fascial layer, allow extraperitoneal blood to track in this direction. *Jaundice* is sometimes present, but usually mild. *Glycosuria* is present in a few. Increase in the urinary diastatic index above 200 units per ml. is suggestive of pancreatitis. *Raised serum amylase and lipase* levels may also help in the diagnosis, but it must be remembered that these can be increased in cases of perforated ulcer, peritonitis, obstruction, and after partial gastrectomy and other upper abdominal operations. Serum amylase above 200 mg. per ml. is suggestive and a higher level than 500 mg. is almost certainly due to pancreatitis. Normal levels are not uncommon after forty-eight hours, but at this stage the serum lipase is raised and remains elevated for a few days. *Lowered serum calcium* values are not uncommon during the first week and are to some extent related to the severity of the disease. Frank tetany is uncommon.

**Differential Diagnosis.** It is particularly important to exclude *perforated peptic ulcer, intestinal obstruction, biliary colic and cholecystitis, and myocardial infarction*. Occasionally there is some similarity to the symptoms of a *leaking or dissecting aneurysm, or mesenteric thrombosis*. Presence of bowel sounds, more localised tenderness and rigidity, and absence of free gas under the diaphragm help to exclude perforated ulcer. Obstruction is especially likely to be considered in those cases with repeated vomiting, but the absence of increased and high-pitched bowel sounds, and failure to demonstrate obstruction in the plain X-ray film, usually rules out this alternative. Severe biliary colic is difficult to exclude at the onset and pancreatitis may succeed acute cholecystitis. Myocardial infarction may be similar at the onset, but the electrocardiogram establishes this diagnosis. Dissecting or leaking aneurysm, may give rise to very similar pain, but shock is commonly more severe and more persistent.

**Treatment.** Formerly it was the rule to advocate early operation and to make a rather aggressive attack on the pancreas. The gland was drained, sloughs were removed and, in the event of associated biliary disease, the gall bladder was sometimes drained. The consensus of opinion now is that surgery is unnecessary in the mild cases and certainly harmful in the more severe examples of acute pancreatitis. Nevertheless, operation may be undertaken because some other condition is diagnosed or because the diagnosis is in doubt. Later on in the illness a secondary abscess may have to be drained, or a subsequent cholecystectomy may be indicated for associated biliary disease.

During the active phase of acute pancreatitis non-operative treatment is the rule. The principles of such treatment are: (1) Relief of pain; (2) treatment of shock; (3) correction of any fluid or electrolyte

survives partial pancreatectomy it is likely that no harm and possibly some good will have been done. *Cases with Intractable Pain.*—These cases seem to be altogether more common in the United States. Dilatation of the sphincter of Oddi or cutting thereof is said to be of value in the chronic relapsing variety with pain. Some surgeons have tried bilateral resection of the splanchnic nerves but proper assessment of these operations is most difficult. Rarely, when all other methods of relieving pain have failed, it is justifiable to consider pancreatectomy.

### CYSTS OF THE PANCREAS

Cysts arising in, or in connection with the pancreas are not common. The following varieties occur: (1) *Pseudocysts*. (2) *True cysts*.—(a) Retention cyst; (b) polycystic disease; (c) cystadenoma; (d) fibrocystic disease; (e) miscellaneous—hydatid, dermoid, degeneration in neoplasm.

**Pseudocyst.** This is due to a fluid collection in the lesser sac and is by far the commonest variety. It may occur at all periods of life. Such pseudocysts usually follow pancreatitis or trauma, or occasionally they are due to perforation of a gastric ulcer into the lesser sac. Sometimes they attain huge dimensions and may be so large as to simulate ascites or a large ovarian cyst. The lining is smooth and the contents clear, greenish or blood-stained. Sometimes the fluid contains all three pancreatic ferments. Rarely such a cyst ruptures into the peritoneal cavity, and infection is another occasional complication.

**Treatment.** The simplest method of treatment is by *marsupialisation*. This is done by exposing the cyst, evacuating its contents and then exteriorising the empty sac, preferably through a small separate wound. The redundant part of the cyst wall is cut off and its cut edge is sutured to the skin. There is a tendency for digestion of the surrounding skin to occur, and it is important to protect it from the time of operation by the application of aluminium paint, or zinc oxide and aluminium cream. Alternative to marsupialisation is *anastomosis* of the cyst to stomach, duodenum or jejunum, but this is really a less safe and certainly no more satisfactory procedure.

**True Cysts.** These are very much less common and do not justify detailed discussion. *Retention cysts* may follow pancreatitis or be due to stones. *Polycystic disease* is sometimes associated with similar changes in the liver and kidneys. *Cystadenoma* may attain quite large dimensions and thereby cause symptoms (Fig. 38). *Dermoid cysts* and *hydatid cysts*, and cystic degeneration in a *tumour* may also occur. *Fibrocystic disease* is a special condition which is mentioned in connection with *meconium ileus* in Chap. 19.

### TUMOURS OF THE PANCREAS

Tumours of the pancreas may arise from the acini and ducts, the islet-cells or the stroma. They include the following: (1) *Benign*.—

the organ is affected, there may be pressure on the common bile duct. The gland is either coarsely lobular or diffusely infiltrated with scar tissue. Stone formation and calcification occasionally occur in the pancreas, and the islet tissue is often damaged.

**Clinical Features.** *Indigestion* often occurs due to interference with the external secretions of the pancreas. Sometimes there is severe impairment of protein and fat digestion with *steatorrhœa* and *loss of weight*. *Pain* is not uncommon, but this may sometimes be due to associated biliary disease. Sometimes there are recurring attacks of abdominal pain which may be quite severe, and this condition is referred to as *chronic relapsing pancreatitis*. Exceptionally the pain is quite intractable. *Jaundice* may occur if the head of the gland is involved, and even at operation it may be impossible to differentiate the condition from carcinoma. *Low sugar tolerance* and *diabetes* are quite common.

Investigations may assist in the diagnosis. It is unusual for the serum amylase or lipase levels to be raised unless there has been some recent exacerbation of symptoms. Examination of the stools or aspiration of duodenal contents demonstrate deficiency of the external secretions in some cases. Plain X-ray may show stones or calcification, but it is quite exceptional for the barium meal X-ray to demonstrate any marked deformity or displacement of the duodenum.

It should be noted that pathological changes consistent with chronic pancreatitis are not uncommonly observed by the surgeon at the time of laparotomy. It is often difficult to assess the significance of such changes or to decide whether they represent an active inflammatory process. Moreover, even biopsy of the pancreas can be unhelpful in elucidating the diagnosis of carcinoma. Fortunately only a very small proportion of patients with chronic pancreatitis require operation. One of the chief dangers is that of undertaking an unnecessary resection for what is wrongly presumed to be a carcinoma.

**Treatment.** Operation is likely to be undertaken in three main sets of circumstances: For the relief of obstructive jaundice; for the removal of what is thought to be a carcinoma; or for the relief of pain. *Cases with Obstructive Jaundice.*—If the gall bladder is healthy it may be anastomosed to the jejunum, stomach or duodenum. However, if the gall bladder is diseased or has already been removed, the common bile duct must be used for the by-pass. Sometimes such an operation is done for what is believed to be an inoperable carcinoma and the surgeon is delighted to see his diagnosis confounded by the patient's long survival. *Cases Simulating Carcinoma.*—It is most difficult to exclude the diagnosis of carcinoma by biopsy, and frozen sections of the pancreas are notoriously difficult to interpret. It is not uncommon for resection of the head of the pancreas to be done for what is believed to be a carcinoma, and for the subsequent histological examination to show unequivocal evidence of pancreatitis. It is calamitous if such a patient dies after operation, but if the patient

survives partial pancreatectomy it is likely that no harm and possibly some good will have been done. *Cases with Intractable Pain.*—These cases seem to be altogether more common in the United States. Dilatation of the sphincter of Oddi or cutting thereof is said to be of value in the chronic relapsing variety with pain. Some surgeons have tried bilateral resection of the splanchnic nerves but proper assessment of these operations is most difficult. Rarely, when all other methods of relieving pain have failed, it is justifiable to consider pancreatectomy.

### CYSTS OF THE PANCREAS

Cysts arising in, or in connection with the pancreas are not common. The following varieties occur: (1) *Pseudocysts*. (2) *True cysts*.—(a) Retention cyst; (b) polycystic disease; (c) cystadenoma; (d) fibrocystic disease; (e) miscellaneous—hydatid, dermoid, degeneration in neoplasm.

**Pseudocyst.** This is due to a fluid collection in the lesser sac and is by far the commonest variety. It may occur at all periods of life. Such pseudocysts usually follow pancreatitis or trauma, or occasionally they are due to perforation of a gastric ulcer into the lesser sac. Sometimes they attain huge dimensions and may be so large as to simulate ascites or a large ovarian cyst. The lining is smooth and the contents clear, greenish or blood-stained. Sometimes the fluid contains all three pancreatic ferments. Rarely such a cyst ruptures into the peritoneal cavity, and infection is another occasional complication.

**Treatment.** The simplest method of treatment is by *marsupialisation*. This is done by exposing the cyst, evacuating its contents and then exteriorising the empty sac, preferably through a small separate wound. The redundant part of the cyst wall is cut off and its cut edge is sutured to the skin. There is a tendency for digestion of the surrounding skin to occur, and it is important to protect it from the time of operation by the application of aluminium paint, or zinc oxide and aluminium cream. Alternative to marsupialisation is *anastomosis* of the cyst to stomach, duodenum or jejunum, but this is really a less safe and certainly no more satisfactory procedure.

**True Cysts.** These are very much less common and do not justify detailed discussion. *Retention cysts* may follow pancreatitis or be due to stones. *Polycystic disease* is sometimes associated with similar changes in the liver and kidneys. *Cystadenoma* may attain quite large dimensions and thereby cause symptoms (Fig. 38). *Dermoid cysts* and *hydatid cysts*, and cystic degeneration in a *tumour* may also occur. *Fibrocystic disease* is a special condition which is mentioned in connection with *meconium ileus* in Chap. 19.

### TUMOURS OF THE PANCREAS

Tumours of the pancreas may arise from the acini and ducts, the islet-cells or the stroma. They include the following: (1) *Benign*.—



(a) *Adenoma* and *cystadenoma* from the acini; (b) *islet-cell tumour*; (c) *fibroma* and other very rare stromal tumours. (2) *Malignant*.—(a) *Carcinoma*; (b) *malignant islet-cell tumour*; (c) *sarcoma*. The most important of these are islet-cell tumour and carcinoma; the others are not discussed here.

**Islet-cell Tumour.** These may be situated anywhere in the pancreas, but are most common in the tail. They are usually about 1 to 2 cm. in diameter, but occasionally much larger. They are greyish-pink, slightly



FIG. 38. Cyst of pancreas. Barium X-ray showing great widening of duodenal loop. (X-ray by Dr. J. M. Stewart.)

darker than the surrounding pancreas and well encapsulated. On histological section the constituent cells resemble the  $\beta$ -cells of the normal islets and are variously arranged in sheets or columns. In 10 per cent. of cases there are multiple tumours.

**Clinical Features.** Some of these cases remain undiagnosed or incorrectly diagnosed for years. The symptoms are those of recurrent hyperinsulinæmia, and include sudden weakness, shivering, fine tremor, nausea, vomiting, drowsiness, loss of consciousness and convulsions. On account of these symptoms, the sufferer is not infrequently labelled as drunk, epileptic, an hysteric or psychotic. Symptoms most commonly occur in the early morning or after a period of fasting, and they can be relieved by the administration of sugar. On physical examination there are no features

specific to this tumour, and only detailed investigation will establish the diagnosis. The blood-sugar is low during the attacks (often 50 mg./100 ml. or lower), but frequently quite normal in between. Fasting usually causes a profound fall in blood-sugar and may precipitate an attack.

**Diagnosis.** In the more severe cases the diagnosis is fairly readily established, but it is essential for these patients to be carefully investigated by the physician before exploration is advised for a presumed tumour. Hypoglycaemic symptoms may also be due to: (1) Low carbohydrate intake; (2) liver disease; (3) hypopituitarism (Chap. 53); (4) rare cases of toxic goitre; and (5) some cases of Addison's disease. In some patients hypoglycaemia defies explanation.

**Treatment.** *Preoperative Preparation.*—Food is allowed up to four hours before operation, and 1,000 ml. of 5 per cent. glucose is slowly given intravenously. During operation a drip transfusion of similar fluid is maintained. *The Operation.*—An upper abdominal transverse incision gives good exposure to all parts of the pancreas. It is wise to search firstly for aberrant pancreatic tissue on the stomach, duodenum, small intestine and in the area between the spleen and tail of pancreas, for about 2 per cent. of tumours occur at an aberrant site. The gastrocolic omentum is divided, and the entire length of the pancreas is exposed and explored. If no tumour is evident the body and tail are mobilised by incising the peritoneum above and below. If, after further careful search there is still no tumour found, the peritoneum is incised lateral to the second part of the duodenum and the head of the pancreas is carefully mobilised. In the event of no tumour being found, it is probably best to excise the body and tail of the pancreas as this cures 50 per cent. of such cases. Total pancreatectomy is probably ill-advised.

During the first few days after successful removal of an islet-cell tumour the patient is sometimes temporarily diabetic and may need insulin.

### CARCINOMA OF THE PANCREAS

The certain identification of tumours arising from the parenchyma of the pancreas as opposed to those of the bile ducts, pancreatic ducts and ampulla is frequently impossible. However, those arising at the latter site are the most favourable.

**Pathology.** About 75 per cent. of carcinomas of the pancreas occur in the head of the viscus (Fig. 39). There is considerable variation in the macroscopic appearances and in the rate of growth. Direct spread occurs through the organ and into the duodenum and other surrounding structures. The regional lymph glands are usually early involved and hepatic deposits are frequently found at operation. Adenocarcinoma is most common and the majority are solid growths, though mucoid and cystic change is not uncommon. Spheroidal-cell and anaplastic types are much less often seen.

**Clinical Features.** The disease is rare under the age of thirty years,

and 75 per cent. of cases occur between the ages of forty and seventy. Men are affected about twice as often as women. Those growths which do not arise in the region of the common bile duct are notoriously silent, and most are quite inoperable at the time of exploration. A growth arising near the ampulla or common bile duct may rapidly cause jaundice and so be diagnosed whilst it is still small and circumscribed. In other cases pain is often the first symptom and consists of either a dull ache or a colic. Anorexia is common, but vomiting is generally a feature of

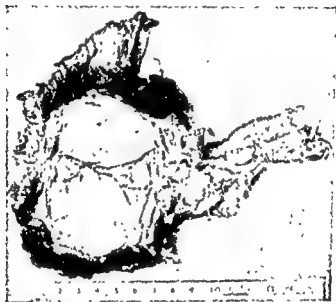


FIG. 39. Carcinoma of head of pancreas (necropsy specimen).

advanced cases with duodenal invasion or obstruction. Some patients have vague *dyspepsia*, *diarrhoea* or bulky and *fatty stools*. On occasions, a curiously *silver stool* is passed due to admixture of blood with a fatty stool lacking in bile pigment. On examination the tumour is occasionally felt, especially in thin people. A distended gall bladder is palpable in the majority of patients who are jaundiced. An enlarged liver may be due to biliary obstruction or secondary deposits.

**Investigations.** Early diagnosis of these tumours is infrequent and largely fortuitous if they do not arise close to the common bile duct. X-ray examination by a *barium meal* is frequently valueless, although many diagnostic features are cited such as widening of the duodenal loop, displacement of the first part of the duodenum, or ulceration of the mucosa. Unfortunately, these signs are generally evidence of advanced disease and in the majority of cases no X-ray evidence is forthcoming. The presence of *occult blood* in the stools helps to focus attention on the pancreas when other sites of origin have been excluded. Blood is more likely to be present with ampullary tumours or with advanced growths which ulcerate the duodenum.

**Treatment.** At operation, the diagnosis of malignant disease of the pancreas is perfectly straightforward when direct spread or metastasis has occurred. When disease is localised to the pancreas it may be exceedingly difficult to be sure that the patient has a carcinoma. Biopsy is sometimes misleading unless a fairly large specimen is taken, and even then mistakes are made. Assuming the diagnosis to be established or reasonably certain, preparations can be made to go ahead to resection or a palliative operation. Prior to operation it is essential to relieve anæmia, hypoproteinaemia, vitamin depletion and low prothrombin level. Every effort must be made to get the patient into the best possible condition.

**Palliative Operations.** In the presence of distant deposits or advanced and fixed local disease a palliative by-pass is indicated. The gall bladder is anastomosed to stomach or jejunum and, though surgeons are wont to argue their relative merits, there is little to choose between these two procedures. Occasionally a gastrojejunostomy is also necessary to by-pass an obstructed duodenum. Most of these patients survive for only a few months and there is a fairly high post-operative mortality. In a small number of patients with an apparently irremovable tumour the satisfactory progress after operation leaves no doubt that the mass was due to pancreatitis. The surgeon may then be thankful that the disease was not sufficiently well localised to have tempted him into an unnecessary resection.

**Resection Operations.** These include : (1) Removal of the body and/or tail ; (2) removal of the head ; (3) total pancreatectomy. *Removal of the body and tail* by partial pancreatectomy is only practicable for those infrequent cases in which disease is confined to the left half of the pancreas. The pancreas is divided at the neck and the left half is then removed with the spleen and its vessels. *Removal of the head* of the pancreas is quite commonly done, although the technique is by no means standardised (Fig. 40). It is an operation which should only be undertaken in suitably selected cases, since, even in the best hands, there is still a fairly high morbidity and mortality, and early recurrence of the disease is common. Before embarking on such an operation it is salutary to recall the aphorism that more people have died following resection of an inflammatory mass mistaken for carcinoma than have been cured of malignant disease by this operation. For this reason every effort must be made to establish a definite diagnosis before resection, and this can best be achieved by doing the operation in two stages. At the first stage a

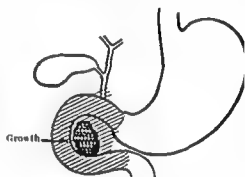


FIG. 40. Carcinoma of pancreas (marked with an arrow). To show extent of tissue removal when resecting carcinoma in the head of the pancreas.

biopsy is performed as well as choledochojejunostomy and gastrojejunostomy. If carcinoma is confirmed the resection can be effected two or three weeks later. Some prefer to do the operation in one stage and accept the risk of a possibly unnecessary resection. Whichever method is used the resection includes the head of pancreas, lower part of common bile duct and the first to third parts of the duodenum, which are

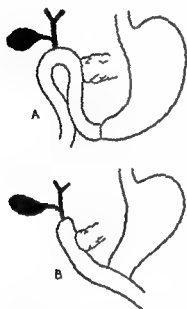


FIG. 41. Resection of the head of the pancreas. Showing two methods of reconstruction: A. End-to-side anastomosis of common duct and pancreas to the jejunum, end-to-end gastrojejunostomy and side-to-side jejunojejunostomy. B. End-to-end anastomosis of common duct, side-to-side anastomosis of pancreas and stomach.

most carefully cleared from the portal vein and mesenteric vessels. The continuity of the gastro-intestinal and biliary tracts is restored and the pancreatic duct is anastomosed to the jejunum (Fig. 41).

**Total Pancreatectomy.** So great is the digestive upset which follows this operation that few surgeons consider it justifiable in the treatment of a disease which itself carries such a poor prognosis. Steatorrhea, severe loss of weight, anorexia, anaemia and diabetes are common and many of the resulting symptoms are exceedingly distressing. To inflict intolerable suffering on a patient in exchange for a remote chance of curing the cancer may be both bad philosophy and bad surgery.

**Results of Surgery.** As is to be expected, the best results are obtained in cases of carcinoma of the ampulla of Vater, and operations for this tumour carry the lowest mortality. The next most favourable cases are those in which there is a localised growth in the head of the pancreas situated close to the common duct. In the very best hands a mortality as low as 5 per cent. has been reported for the former cases, and about 17

per cent. for the latter. The five-year-survival rate in these two groups does not exceed 30 and 10 per cent. respectively. These results, which represent the very best surgery under the most favourable conditions, are far better than those achieved in the majority of hospitals. Moreover, such results are only seen in proper perspective if it be remembered that resection of the growth is only practicable in a minority of cases. The over-all results of treatment of carcinoma of the pancreas are extremely disappointing.

## CHAPTER 5

### THE LIVER

**DESIGN:** the wide variety of conditions which may affect the liver, operations upon this organ are not a common feature of the surgeon's repertoire. Although the resection of tumours of the liver and operations for the attempted relief of portal hypertension are now done more frequently, these are still uncommon procedures. Recent injection studies have given a much clearer idea of the vascular pattern in the liver, and this may facilitate resection operations when these are necessary. About 75 per cent. of the blood entering the liver comes by the portal vein and the rest by the hepatic artery, but the higher oxygen tension of the arterial as compared with the portal vein blood means that at least half the oxygen required by the liver is derived from the arterial source.

**Liver Function Tests in Surgery.** Liver function tests are of importance in two connections: (1) Assessing the fitness and suitability of a patient for operation; (2) differentiating between medical and surgical causes of jaundice. It must be emphasised that innumerable tests have been described, that many of these are of limited value, and that intelligent interpretation of the results is essential to proper assessment of a case. Properly used, these tests are valuable ancillaries, but they rarely supplant mature clinical judgement. Details of the various tests are not given here, but the following are amongst those most commonly used in surgical practice.

**Serum Bilirubin.** Although the serum bilirubin level (normal 0.1 to 0.5 mg. per 100 ml.) reflects the intensity of jaundice, neither it nor the Van den Bergh reaction permit confident differentiation of medical and surgical jaundice. In the obstructive phase of infective hepatitis there may be a high serum bilirubin level and a direct Van den Bergh reaction.

**Plasma Prothrombin.** The plasma prothrombin may be low both in obstructive and medical jaundice, in obstructive cases due to failure to absorb vitamin K, and in medical cases due to impairment of prothrombin synthesis by the liver cells. In obstructive jaundice the prothrombin percentage usually returns fairly rapidly to normal when synthetic vitamin K analogue is given. In medical cases the response is usually slower, although vitamin K<sub>1</sub> may act rapidly. The rapidity of response to vitamin K analogue is not a reliable method of differentiating between medical and surgical jaundice, since parenchymatous liver damage may be associated with obstruction, especially if there is infection in the biliary tract. The prothrombin level and its response to vitamin K administra-

tion do, however, give the surgeon useful information about the fitness of the patient for operation.

*Serum Proteins.* Depletion of the serum proteins is a feature of advanced liver disease. Marked lowering of the albumin level is often found in severe cirrhosis and also occurs in long-standing cases of surgical jaundice. The serum proteins may also be low as a result of malnutrition and loss of protein in ascitic fluid. Low total serum protein and reversal of the albumin/globulin ratio may not only be of serious prognostic importance, but should also warn the surgeon that his patient needs careful preparation before operation.

*Alkaline Phosphatase.* Although the serum alkaline phosphatase is raised in obstructive jaundice, this is not a certain method of differentiating medical and surgical jaundice. There are occasional cases of hepatitis in which the serum bilirubin and the alkaline phosphatase levels are so high as to suggest an obstructive cause.

*Flocculation Tests.* Of the various flocculation tests in common use, estimation of the thymol turbidity and flocculation is one of the most popular. Increased flocculation is commonly found in hepatitis and cirrhosis. It can be said that, in the presence of jaundice, a raised thymol turbidity and low alkaline phosphatase favours a medical rather than a surgical cause. On the other hand, a surgical cause is suggested by high alkaline phosphatase and low thymol turbidity.

*Liver Biopsy.* Needle-puncture biopsy of the liver is a well-established and valuable diagnostic procedure in skilled hands. Although it is only necessary in a minority of cases, it may be the only method of differentiating medical from surgical jaundice. When there is doubt in the diagnosis, a needle biopsy is far safer than laparotomy, but it should only be done when the prothrombin level has been restored to at least 80 per cent., and full preparation has been made for blood transfusion and exploration. Leakage of bile into the peritoneum is an occasional complication of biopsy in cases of obstructive jaundice; but, if the biopsy is done after full preparation and the surgeon is ready to proceed to laparotomy and relieve the obstruction, there should be no serious danger. Apart from percutaneous puncture biopsy, a specimen may be taken during laparotomy; for example, to confirm the diagnosis of carcinoma or to assess the severity of parenchymatous disease. In the latter cases it is important to take the specimen as soon as the abdomen is opened, for the liver tends to become infiltrated with neutrophil cells in the course of operation.

### RUPTURE OF THE LIVER

Due to its size, position and vulnerability, the liver is damaged more often than any other solid abdominal viscus. Blunt injuries are commonly due to road accidents, and there is often fracture of one or more lower ribs. Open wounds are rare in civilian practice in Great Britain.

*Clinical Features.* The symptoms and signs are those of blood loss

together with pain in the upper abdomen, chest or right shoulder. Upper abdominal tenderness and guarding are usually present, but sometimes the patient is so severely injured that satisfactory physical examination is difficult (Fig. 42). Occasionally a hollow viscus is also damaged. If so, gut sounds are usually absent and air may be seen beneath the diaphragm on X-ray.

**Treatment.** Relief of shock, replacement of blood loss, exclusion of damage to other abdominal viscera, and proper assessment of associated extra-abdominal injuries are most important before operation is undertaken. Minor tears may need no treatment and may not even be suspected, but patients with major lacerations may quickly perish. There is



FIG. 42. Rupture of liver. Extensive laceration of back of liver at junction of right and left lobes.

no doubt that early intervention is desirable, but the exact extent of the damage may be impossible to assess before operation.

Sometimes, when the patient is relaxed under the anæsthetic and the abdomen opened, torrential bleeding occurs. It is often wise to have two intravenous transfusions running and occasionally an arterial transfusion apparatus is of value. Exposure is usually best with an adequate upper abdominal vertical incision. Clean-cut and accessible tears can be quite easily repaired by suture. Bleeding vessels can be ligated first and sutures are then inserted wide of the wound and loosely tied. Various types of suture are used for this purpose (Fig. 43). Packing may be the only practicable method of dealing with bigger irregular tears or those involving the back of the liver; usually absorbent gauze is used. For some wounds a part of the great omentum or one of the proprietary brands of oxidised cellulose or gelatin sponge may be used. Drainage of the peritoneum is usually advisable and, if a gauze pack has been used, its end is brought out alongside the drain. Such a pack is gradually withdrawn during the ensuing two or three days.



tion do, however, give the surgeon useful information about the fitness of the patient for operation.

*Serum Proteins.* Depletion of the serum proteins is a feature of advanced liver disease. Marked lowering of the albumin level is often found in severe cirrhosis and also occurs in long-standing cases of surgical jaundice. The serum proteins may also be low as a result of malnutrition and loss of protein in ascitic fluid. Low total serum protein and reversal of the albumin/globulin ratio may not only be of serious prognostic importance, but should also warn the surgeon that his patient needs careful preparation before operation.

*Alkaline Phosphatase.* Although the serum alkaline phosphatase is raised in obstructive jaundice, this is not a certain method of differentiating medical and surgical jaundice. There are occasional cases of hepatitis in which the serum bilirubin and the alkaline phosphatase levels are so high as to suggest an obstructive cause.

*Flocculation Tests.* Of the various flocculation tests in common use, estimation of the thymol turbidity and flocculation is one of the most popular. Increased flocculation is commonly found in hepatitis and cirrhosis. It can be said that, in the presence of jaundice, a raised thymol turbidity and low alkaline phosphatase favours a medical rather than a surgical cause. On the other hand, a surgical cause is suggested by high alkaline phosphatase and low thymol turbidity.

*Liver Biopsy.* Needle-puncture biopsy of the liver is a well-established and valuable diagnostic procedure in skilled hands. Although it is only necessary in a minority of cases, it may be the only method of differentiating medical from surgical jaundice. When there is doubt in the diagnosis, a needle biopsy is far safer than laparotomy, but it should only be done when the prothrombin level has been restored to at least 80 per cent., and full preparation has been made for blood transfusion and exploration. Leakage of bile into the peritoneum is an occasional complication of biopsy in cases of obstructive jaundice; but, if the biopsy is done after full preparation and the surgeon is ready to proceed to laparotomy and relieve the obstruction, there should be no serious danger. Apart from percutaneous puncture biopsy, a specimen may be taken during laparotomy; for example, to confirm the diagnosis of carcinoma or to assess the severity of parenchymatous disease. In the latter cases it is important to take the specimen as soon as the abdomen is opened, for the liver tends to become infiltrated with neutrophil cells in the course of operation.

#### RUPTURE OF THE LIVER

Due to its size, position and vulnerability, the liver is damaged more often than any other solid abdominal viscus. Blunt injuries are commonly due to road accidents, and there is often fracture of one or more lower ribs. Open wounds are rare in civilian practice in Great Britain.

*Clinical Features.* The symptoms and signs are those of blood loss

confirmed on X-ray, and the latter occasionally reveals a gas bubble in the abscess cavity. The differential diagnosis is sometimes difficult, and the gas bubble may be the only unequivocal way of diagnosing subphrenic abscess rather than portal thrombophlebitis.

**Acute Suppurative Cholangitis.** Suppurative cholangitis may complicate all forms of biliary obstruction and is most often due to stone in the



FIG. 44. Suppurative cholangitis due to malignant obstruction of common duct. Multiple abscesses and related hepatitis surrounding bile ducts on the left. Secondary carcinoma on extreme right of specimen.

common bile duct (Fig. 44). It may be associated with cholecystitis or pancreatitis.

**Treatment.** Both the above conditions are considered together. With *portal thrombophlebitis*, if no abscess is present, treatment is by : (1) relief of the primary condition ; (2) antibiotic therapy. Prophylactic ligation of the ileocolic vein, formerly advocated in cases of appendicitis with high temperature and rigors, is probably of no value. Massive dosage of penicillin (2 mega units daily) is effective in many cases ; but, if there is a poor response, or the infection seems very severe, it would seem wise to use tetracycline (2 gm. daily). Antibiotics should not be given less than six-hourly in such cases. Although it is unusual for a large solitary abscess

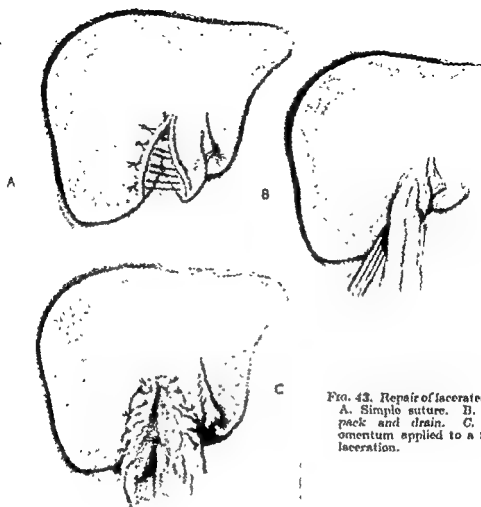


FIG. 43. Repair of lacerated liver. A. Simple suture. B. Gauze pack and drain. C. Great omentum applied to a ragged laceration.

### PYOGENIC INFECTION OF THE LIVER

Pyogenic organisms most often reach the liver via the portal vein or bile ducts ; less often infection enters by the hepatic artery or by direct spread. In many of these cases no primary source of infection is detected. In the event of pyæmic infection via the hepatic artery the general infection usually overshadows the local condition, which is rarely of surgical importance.

**Portal Pyæmia and Thrombophlebitis.** These are most often due to appendicitis, but this complication occurs in less than one in 300 cases of acute appendicitis. *Diverticulitis coli* and other infective conditions in the portal area are less common causes. Multiple small abscesses may occur in the liver. Clinically, there is continued pyrexia and sometimes rigors occur. The liver may be enlarged and tender, but jaundice, if present, is often slight. Subphrenic abscess can give rise to similar clinical features, but in these cases the diaphragm is more often raised. This may be

or upper part of small gut, and is carried to the liver by the portal vein.

The established cyst in the liver is surrounded by a condensation of adventitious tissue called the ectocyst (Fig. 46). Within this layer is a laminated hyaline membrane resembling bacon rind and lined by a germinal layer of cells from which brood capsules develop. The cyst is filled with a clear fluid which may contain daughter cysts and grand-daughter cysts which look like grape skins (Figs. 46 and 47). In long-standing cases there is often calcification in the adventitia. As the cyst increases in size it may rupture into the biliary tract, gut, pleural cavity or peritoneal cavity. Sometimes the bile ducts are obstructed by the cyst

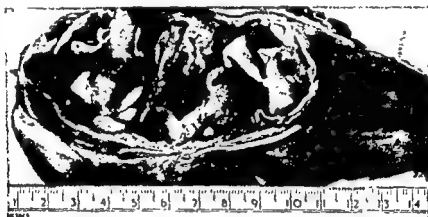


FIG. 46. Hydatid cyst of liver. Note thick ectocyst and daughter cysts. The younger daughter cysts have the thinnest walls.

contents. In the event of rupture into the peritoneum there may be several possible consequences: (1) Anaphylactic shock and possible death due to toxic substances in the cyst fluid; (2) secondary cyst formation in the peritoneum; (3) peritonitis, if the cyst is infected.

**Clinical Features.** There are usually no symptoms until the cyst ruptures or is secondarily infected. Biliary colic may follow rupture into a duct. Occasionally an abdominal swelling is accidentally noted or calcification in the cyst wall is seen on X-ray.

**Diagnostic Tests.** *Eosinophilia* is present in about a third of cases. *Casoni Test.*—This is positive in between 90 and 95 per cent. of cases. The test consists in the intradermal injection of 0.3 ml. of cyst fluid and a positive result is indicated by a large wheal. *Complement Fixation Test.*—This is similar to the Wassermann reaction for syphilis. It is positive in about 80 per cent. of cases and a persistently positive result more than a year after operation means that another cyst is present. *Stool Examination.*—This sometimes reveals daughter cysts after rupture of the primary cyst into the gut or bile ducts.

to form, drainage of this or a complicating subphrenic abscess is occasionally necessary. Careful inspection, palpation and exploratory needling of the liver may be necessary in these cases. The pus is then drained by

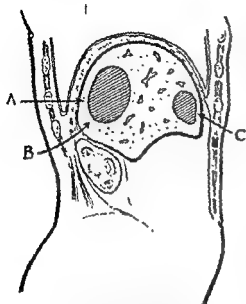


FIG. 45. Drainage of liver abscess. A. Posterior route through bed of eleventh rib. B. Below twelfth rib. C. Anterior subcostal route.

the most direct and accessible route (Fig. 45). Those cases in which hepatic infection is secondary to suppurative cholangitis are usually easier to treat. In the majority of these cases antibiotic therapy, removal of stones from the biliary tract, and drainage of the ducts suffices.

### OTHER INFECTIONS

**Amoebic hepatitis and abscess** are discussed in Chap. 11.

**Actinomycosis** is rare. It may be caused by either aerobic or anaerobic strains of *Streptothrix actinomyces*. Infection is usually from the caecum and appendix, or occasionally from the lung via the diaphragm. This condition was formerly almost invariably fatal. Initially the liver is

enlarged, firm and contains granulomatous lesions. Later it tends to be honeycombed with abscess cavities. Abscesses may point into the gut and through the abdominal wall or diaphragm. Usually there are symptoms due to the primary site of infection. A high swinging temperature is common and there is often a leucocytosis. The liver is generally palpable and sometimes much enlarged, but jaundice is usually not marked. *Treatment* is by massive doses of antibiotics. Penicillin (2 mega units daily) is suitable for some strains of streptothrix, but normally the antibiotic used depends upon sensitivity tests. Antibiotic treatment should continue for two to three months.

### CYSTS AND TUMOURS OF THE LIVER

**Hydatid Disease.** Hydatid cyst of the liver occurs most commonly in Australia, New Zealand and Argentina. It is uncommon in Great Britain. The liver is by far the most common site for hydatid cyst, being involved in 80 per cent. of all cases. The cyst is usually solitary and is four times as frequent in the right lobe as in the left (Fig. 46). *Tenia echinococcus*, the responsible parasite, lives in the intestine of the dog, which is the main definitive host, and discharges its eggs in the faeces. Man is most commonly infected by handling the dog, or by ingesting contaminated water or vegetables. The parasite penetrates the mucosa of the stomach

In infected or ruptured cysts the mortality may be as high as 20 per cent.

**Other Cysts and Benign Tumours.** *Non-parasitic Cysts.*—These may vary from 1 to 15 mm. in diameter. Their chief importance is that they may be mistaken for secondary deposits of carcinoma if they are merely palpated and not inspected during laparotomy. *Polycystic* disease is very rare, it is occasionally associated with polycystic disease of the kidneys and does not impair liver function. *Benign cystadenoma* and *dermoid cyst* are also rare.

*Hæmangioma.* This is certainly the commonest benign lesion of the

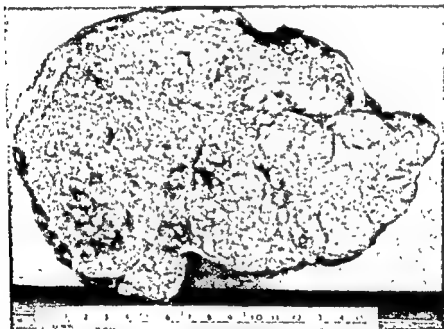


FIG. 48. Cirrhotic ("hobnail") liver with extensive carcinomatous transformation on right.

liver. It is often multiple, small and superficial, and requires no treatment. Like simple cyst, it is chiefly important because on palpation it may be mistaken for metastasis at laparotomy. *Riedel's Lobe.*—This accessory lobe arising from the right side of the liver, if palpated per abdomen, may be mistaken for a tumour.

#### MALIGNANT TUMOURS OF LIVER

**Carcinoma.** *Primary Carcinoma.*—Although rare in most races, this is the commonest of all carcinomas in the Bantus of South Africa and may be associated with cirrhosis (Fig. 48). The more common type arises from the liver cells (*hepatoma*), and less often the tumour starts from the bile ducts (*cholangioma*). Occasionally it is feasible to resect a primary growth

**Treatment.** *Calcified cysts* are left alone unless they are giving rise to severe symptoms. *Uncomplicated Cysts.*—These should be localised as accurately as possible by clinical and X-ray examination. Arrangements are then made for drainage. According to the site of the cyst, the incision will be paramedian, subcostal, or through the bed of the resected eleventh and twelfth ribs. The cyst is rarely deeply situated and usually visible on

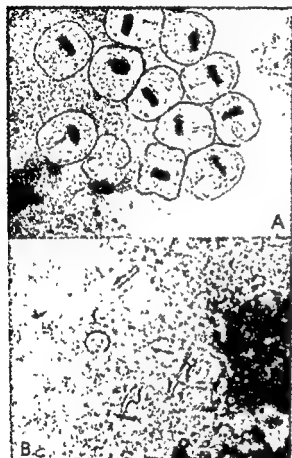


FIG. 47. Hydatid disease. A. Scolices and B. hooklets from cyst fluid.

exposing the liver. Other cysts should be searched for before proceeding. To avoid possible contamination of the wound the whole field is protected with a double layer of drapes; the more superficial layer is black so as to show up any spilled hydatid particles. The cyst is next aspirated as completely as possible through a wide-bore needle. Pure commercial formalin is then injected into the cavity. After allowing five minutes for the formalin to act traction sutures are inserted into the cyst wall and the contents evacuated with a powerful sucker. To ensure that all daughter cysts and the germinal layer are removed the resultant cavity is swabbed out with 2 per cent. formalin. In straightforward cases the cavity can be filled with normal saline solution containing sodium penicillin (1 mega unit) and the capsule firmly sutured. With bile-con-

taining or infected cysts the adventitia is closed snugly around a wide-bore drainage tube. As an alternative, the cavity can be marsupialised by suturing the adventitia to skin.

**Complications.** (1) Rupture may take place into the biliary tract, peritoneal cavity or pleural cavity. In such cases there may be biliary colic, jaundice, anaphylactic shock, or signs of peritonitis or pleurisy. Occasionally the cyst contents are coughed up following perforation into a bronchus. (2) Suppuration is usually due to infection from the biliary passages. The clinical features are variable and may simulate liver abscess, cholecystitis or subphrenic abscess.

**Results.** In uncomplicated cases the operative mortality is negligible.

are told that the operative findings are unfavourable, but that they should hope for the best, the patient is sometimes spared that over-anxious indulgence of his relatives which can be so damaging to his own morale. Moreover, in the event of unexpectedly long survival, regression of the deposits, or a wrong initial diagnosis, the doctor is regarded as a poor prophet rather than a fool.

### CIRRHOSIS HEPATIS AND PORTAL HYPERTENSION

Cirrhosis hepatitis is by no means regularly accompanied by portal hypertension, and the latter is not always due to cirrhosis, but these conditions are conveniently discussed together. It is still too early to assess the true value of a number of operations which have been introduced during the last few years.

**Portal Hypertension.** The normal portal venous pressure is between 8 and 14 cm. of water, but in portal hypertension it may rise to 40 cm. and pressures as high as 60 cm. have been recorded. Portal hypertension may be due to pre-hepatic, intrahepatic or post-hepatic venous obstruction (Fig. 40). The latter includes *congestive heart failure*, *hepatic vein thrombosis* and *constrictive pericarditis* (see Chap. 46). This discussion is not concerned with this type of case though it is important to remember such possibilities: misdiagnosis has been known to result



FIG. 50. Cirrhosis hepatitis. Showing "hob-nail" liver, enlarged spleen due to portal hypertension, and well-marked oesophageal varices. (N.B. The varices tend to collapse after death and are not so evident at necropsy, cf. Fig. 53.)

in a totally unjustifiable porta-caval venous shunt operation on a patient with constrictive pericarditis. Intra-hepatic causes include thrombosis of the portal vein within the liver and all forms of cirrhosis (Fig. 50). For a full discussion of the latter conditions the reader is referred to authoritative medical texts. Cirrhosis is most commonly due to: (1) Infective hepatitis and toxic agents; (2) bacterial and protozoal infections; (3) biliary obstruction; (4) congestive heart failure; (5) unknown causes.

Prehepatic causes of portal hypertension include congenital abnormalities and thrombosis of the portal vein. Sometimes thrombosis is



of the liver, but the prognosis is usually bad. *Secondary Carcinoma.*—Secondary carcinoma is the most common type of malignant hepatic tumour in Great Britain, and the liver is the commonest site for visceral metastasis. The primary tumour is most often in the stomach, colon, breast or lung. Spread to the liver occurs via the portal vein, the lymphatics, the hepatic artery, or by direct spread.

It is impossible to predict how fast hepatic secondary deposits will grow. Very rarely indeed spontaneous regression of growth occurs. In occasional cases it is justifiable to resect part of the liver when a secondary tumour is solitary or strictly localised. Sometimes, when the liver is

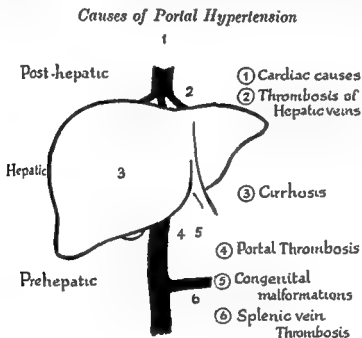


FIG. 49. Causes of portal hypertension.

involved by direct spread, the affected part can be resected in continuity with the primary growth. Though metastasis tends to be superficial, it may be difficult to detect. Therefore the greatest caution is necessary before subjecting a patient to hepatic resection for a secondary tumour. Deposits of *secondary carcinoid tumour* from the small gut are occasionally suitable for resection, but it is noteworthy that even without treatment some of these cases survive for many years.

The doctor is often called upon to predict the likely period of survival of a patient with hepatic deposits. It can be exceedingly difficult to give a prognosis in these cases, and for this reason it is sometimes wiser and kinder for the doctor not to refer specifically to hepatic deposits when he talks to a relative. After a fruitless laparotomy, at which hepatic deposits are found, the surgeon is tempted to tell the next of kin that the condition is quite hopeless. Such truthfulness does not always pay. If the relatives

are told that the operative findings are unfavourable, but that they should hope for the best, the patient is sometimes spared that over-anxious indulgence of his relatives which can be so damaging to his own morale. Moreover, in the event of unexpectedly long survival, regression of the deposits, or a wrong initial diagnosis, the doctor is regarded as a poor prophet rather than a fool.

### CIRRHOSIS HEPATIS AND PORTAL HYPERTENSION

Cirrhosis hepatis is by no means regularly accompanied by portal hypertension, and the latter is not always due to cirrhosis, but these conditions are conveniently discussed together. It is still too early to assess the true value of a number of operations which have been introduced during the last few years.

**Portal Hypertension.** The normal portal venous pressure is between 8 and 14 cm. of water, but in portal hypertension it may rise to 40 cm. and pressures as high as 60 cm. have been recorded. Portal hypertension may be due to pre-hepatic, intrahepatic or post-hepatic venous obstruction (Fig. 40). The latter includes *congestive heart failure*, *hepatic vein thrombosis* and *constrictive pericarditis* (see Chap. 46). This discussion is not concerned with this type of case though it is important to remember such possibilities: misdiagnosis has been known to result



FIG. 50. Cirrhosis hepatis. Showing "hob-nail" liver, enlarged spleen due to portal hypertension, and well-marked oesophageal varices. (N.B. The varices tend to collapse after death and are not so evident at necropsy, cf. Fig. 53.)

in a totally unjustifiable porta-caval venous shunt operation on a patient with constrictive pericarditis. Intra-hepatic causes include thrombosis of the portal vein within the liver and all forms of cirrhosis (Fig. 50). For a full discussion of the latter conditions the reader is referred to authoritative medical texts. Cirrhosis is most commonly due to: (1) Infective hepatitis and toxic agents; (2) bacterial and protozoal infections; (3) biliary obstruction; (4) congestive heart failure; (5) unknown causes.

Prehepatic causes of portal hypertension include congenital abnormalities and thrombosis of the portal vein. Sometimes thrombosis is

confined to the splenic vein with resultant localised congestion and splenomegaly. Whatever the cause of portal obstruction, collateral channels tend to open up at various sites and a natural shunt is thus formed between portal and systemic circulations (Figs. 51 and 52). From

the surgical viewpoint, the collateral site of most importance is at the cardiac end of the stomach and lower end of the oesophagus. It is interesting to speculate why severe bleeding should occur from varicosities at this site, and yet they rarely cause serious trouble in the rectum in such cases.

*Ascites.* The precise cause of ascites is not known, though lowering of the plasma albumin may be the most important single factor. Sometimes the plasma albumin is quite normal. Raised portal venous pressure may be a contributory factor in some patients. Disturbances of sodium metabolism may perpetuate the condition and increased intra-abdominal pressure and decreased renal blood flow may also be of importance.

*Splenomegaly.* The cause of the splenomegaly is not entirely clear, since the degree of splenic enlargement is not directly related to the degree of portal hypertension.

Thrombosis in the splenic vein is sometimes a contributory factor and it has been suggested that the liver produces some agent which induces splenic hyperplasia.

*Diagnosis.* The detailed investigation of cirrhosis is a matter for the physician and is not appropriate to this discussion. In about 50 per cent. of cases the only certain method of diagnosing uncomplicated cirrhosis is by needle biopsy of the liver. When oesophageal varices are present they can more easily be demonstrated by a barium swallow X-ray than by

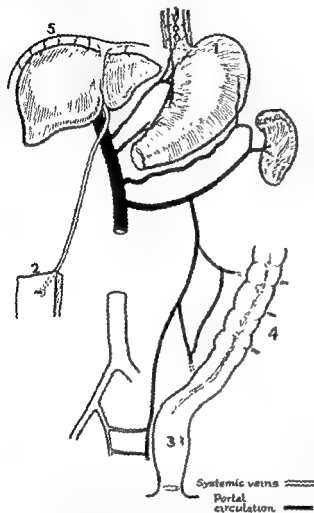


FIG. 51. Sites of natural anastomosis between portal and systemic veins. (1) Gastric with azygos. (2) Portal with para-umbilical. (3) Superior with middle and inferior hemorrhoidal. (4) Colic with lumbar. (5) Hepatic with diaphragmatic.

oesophagoscopy (Fig. 53). In some of these patients the first sign of trouble is massive bleeding, which must be differentiated from that due to a peptic ulcer. Such cases may present with both hæmatemesis and melæna, and strangely enough melæna is sometimes the only sign of bleeding from oesophageal varices. The state of liver function is assessed by the various tests already described; their significance in relation to porta-caval venous anastomosis is referred to later.

**Treatment.** Cirrhosis is primarily a therapeutic problem for the

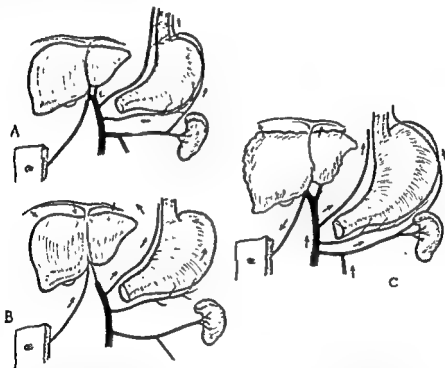


FIG. 53. Portal obstruction. To show some of the natural by-passes established in: A. Splenic vein block. B. Extrahepatic portal vein block. C. Intrahepatic portal vein block due to cirrhosis.

physician and in only a very small proportion of cases are there complications which justify surgical intervention. The chief of these is bleeding from oesophageal varices, but reference must also be made to congestive splenomegaly with hypersplenism, and to ascites. Views on this subject are likely to change considerably in the course of the next few years, and a number of procedures which are fashionable to-day may be discredited in the future.

The problem of bleeding oesophageal varices can be tackled either by direct treatment of the offending veins or by reducing the portal venous pressure.

**Direct Treatment.** *Injection* of oesophageal varices with sclerosant solutions has proved generally unsatisfactory. The injection is made

confined to the splenic vein with resultant localised congestion and splenomegaly. Whatever the cause of portal obstruction, collateral channels tend to open up at various sites and a natural shunt is thus formed between portal and systemic circulations (Figs. 51 and 52). From

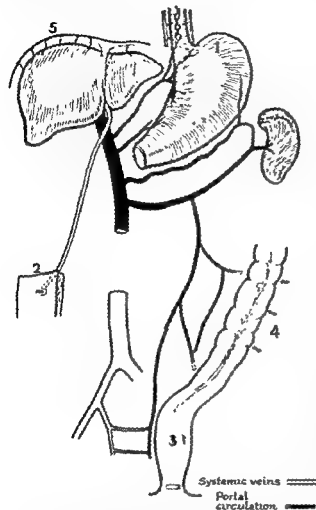


FIG. 51. Sites of natural anastomosis between portal and systemic veins. (1) Gastric with azygos. (2) Portal with para-umbilical. (3) Superior with middle and inferior hemorrhoidal. (4) Colic with lumbar. (5) Hepatic with diaphragmatic.

the surgical viewpoint, the collateral site of most importance is at the cardiac end of the stomach and lower end of the oesophagus. It is interesting to speculate why severe bleeding should occur from varicosities at this site, and yet they rarely cause serious trouble in the rectum in such cases.

**Ascites.** The precise cause of ascites is not known, though lowering of the plasma albumin may be the most important single factor. Sometimes the plasma albumin is quite normal. Raised portal venous pressure may be a contributory factor in some patients. Disturbances of sodium metabolism may perpetuate the condition and increased intra-abdominal pressure and decreased renal blood flow may also be of importance.

**Splenomegaly.** The cause of the splenomegaly is not entirely clear, since the degree of splenic enlargement is not directly related to the degree of portal hypertension.

Thrombosis in the splenic vein is sometimes a contributory factor and it has been suggested that the liver produces some agent which induces splenic hyperplasia.

**Diagnosis.** The detailed investigation of cirrhosis is a matter for the physician and is not appropriate to this discussion. In about 50 per cent. of cases the only certain method of diagnosing uncomplicated cirrhosis is by needle biopsy of the liver. When oesophageal varices are present they can more easily be demonstrated by a barium swallow X-ray than by

is not an indication for such an operation, and portal-systemic anastomosis is quite unjustifiable in the treatment of uncomplicated cirrhosis. There are some who would deny that these operations are ever justified, but the evidence so far available encourages neither unstinting enthusiasm nor condemnation.

In assessing the suitability of a patient for this type of procedure the general health and liver function tests are of great importance. A patient with earthy complexion, frank jaundice, marked anorexia and lethargy is almost certainly a poor risk. The hazards of operation are great if the

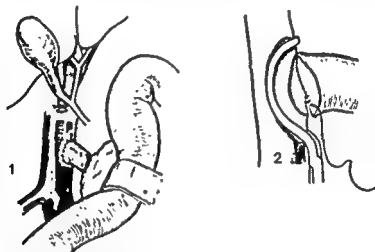


FIG. 54. Porta-caval shunt. (1) Showing end-to-side anastomosis of divided portal vein and vena cava. (2) Everting suture being inserted, with clamp on side of vena cava.

plasma albumin is below 3 gm. per 100 ml., the albumin-globulin ratio reversed, alkaline phosphatase and flocculation tests high, and if the plasma prothrombin level cannot be fully restored with vitamin K. Even assuming the patient's general fitness, it is sometimes a most difficult task to decide whether operation is really necessary. The natural history of the disease is variable and it is therefore difficult to predict the future course of any individual case. Generally, operation should only be considered when repeated severe hæmorrhage has occurred. Some, it is true, would advocate shunting operations on lesser grounds, but the spontaneous development of a good collateral circulation probably justifies a conservative attitude in milder cases.

*Porta-caval Shunt.* Anastomosis of the portal vein to the inferior vena cava offers the best prospect of a good shunt with least risk of subsequent blockage by thrombus (Fig. 54). The abdomen is usually explored through a long upper vertical incision, as this permits exposure of either side of the abdomen. However, when a porta-caval shunt is specifically planned beforehand, many prefer a right thoraco-abdominal incision along the line of the ninth or tenth rib. The liver is examined first, and a biopsy, if

through an œsophagoscope with special long needles, but this is only done between bouts of bleeding. Although a few still advocate this method, it is certainly of limited value and there is always risk of severe bleeding, or perforation and sloughing of the œsophagus. *Balloon tubes* have been specially designed to control hæmorrhage from œsophageal varices. Close to the distal end of the tube is a spherical balloon which is inflated



FIG. 53. Barium swallow X-ray showing large œsophageal varices. (X-ray by Dr. J. M. Stewart.)

within the stomach and then withdrawn to the cardia. Just proximal to this balloon is a stronger elongated one which can be inflated in the lower end of the œsophagus to compress the offending varices. With adequate sedation most patients tolerate such a tube for three or four days, and they can be fed through the main tube. Although a frankly palliative measure, it is occasionally life-saving.

*Direct Operations on Varices.* Various operations have been devised in an attempt to deal with the varices directly. *Resection* of the lower part of the œsophagus and upper end of the stomach is a radical method which is more risky and no more successful than simpler measures. After a short period in vogue it went out of fashion. *Ligation* of all the vessels passing in and out of the lower part of the œsophagus has been tried, but there is some danger of sloughing afterwards. *Ligation of the varices* themselves has been attended with some success, and does not seem to carry too great a risk. The œsophagus is exposed through the left pleural cavity and, after isolating the lower part, an incision is made in its wall. Each of the varices is then ligated and obliterated so far as possible with a continuous catgut suture.

*Disconnection of the varices* is yet another method. This portal-azygos disconnection is effected by dividing the left gastric vein and vasa brevia. The stomach is then transected high up and finally repaired with catgut. The object of this operation is to deprive the varices of their systemic and portal venous connections. The variety of operations which have been tried indicates that none is really satisfactory, and it would be imprudent to presume that any of the above procedures provides a proper answer to this most difficult problem.

*Portal-systemic Venous Shunt.* The diversion of portal blood into the systemic veins with the object of reducing portal venous pressure is commonly referred to as a "shunt." *Portal-systemic venous-shunt operations* should only be considered in those cirrhotics who have suffered severe hæmorrhage from œsophageal varices. Ascites complicating cirrhosis

is not an indication for such an operation, and portal-systemic anastomosis is quite unjustifiable in the treatment of uncomplicated cirrhosis. There are some who would deny that these operations are ever justified, but the evidence so far available encourages neither unstinting enthusiasm nor condemnation.

In assessing the suitability of a patient for this type of procedure the general health and liver function tests are of great importance. A patient with earthy complexion, frank jaundice, marked anorexia and lethargy is almost certainly a poor risk. The hazards of operation are great if the

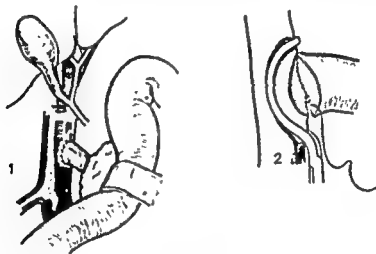


FIG. 54. Porta-caval shunt. (1) Showing end-to-side anastomosis of divided portal vein and vena cava. (2) Everting suture being inserted, with clamp on side of vena cava.

plasma albumin is below 3 gm. per 100 ml., the albumin-globulin ratio reversed, alkaline phosphatase and flocculation tests high, and if the plasma prothrombin level cannot be fully restored with vitamin K. Even assuming the patient's general fitness, it is sometimes a most difficult task to decide whether operation is really necessary. The natural history of the disease is variable and it is therefore difficult to predict the future course of any individual case. Generally, operation should only be considered when repeated severe hæmorrhage has occurred. Some, it is true, would advocate shunting operations on lesser grounds, but the spontaneous development of a good collateral circulation probably justifies a conservative attitude in milder cases.

*Porta-caval Shunt.* Anastomosis of the portal vein to the inferior vena cava offers the best prospect of a good shunt with least risk of subsequent blockage by thrombus (Fig. 54). The abdomen is usually explored through a long upper vertical incision, as this permits exposure of either side of the abdomen. However, when a porta-caval shunt is specifically planned beforehand, many prefer a right thoraco-abdominal incision along the line of the ninth or tenth rib. The liver is examined first, and a biopsy, if



needed, is taken at this stage. The portal vein is then carefully examined and the portal pressure is measured, usually by inserting a suitable needle or fine polythene tube into a jejunal vein. If the portal pressure is raised and the state of the vessels is suitable, a broad porta-caval anastomosis can be made. In some cases there is a form of cavernous transformation of the portal vein, or the latter may be obliterated by thrombosis. In these circumstances, a porta-caval shunt is impossible, but if the splenic and left renal veins are of adequate size, an end-to-side splenorenal anastomosis can be done after removal of the spleen (Fig. 55). If the large vessels

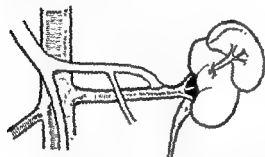


FIG. 55. Splenorenal shunt. Spleen removed and end-to-side splenorenal venous anastomosis.

are blocked or unsuitable, and small vessels only are available, a shunt operation is often ill-advised, as more useful collateral vessels may be divided than can be provided by the surgeon. The anastomosis of the portal vein to the vena cava may be either end-to-side or side-to-side; the latter is preferred by many surgeons. Junction of the two vessels is effected by meticulously careful

insertion of a fine silk suture which everts the edges and apposes the intima of the two vessels.

*Congestive Splenomegaly with Hyperaplenism.* In some of these cases one or other variety of hæmatocytopenia is associated with splenomegaly. Removal of the spleen will relieve the blood dyscrasia and also effect considerable reduction in the portal blood flow, but it is usually agreed that a splenorenal venous shunt should be done at the same time when these veins are most easily utilised (Fig. 55). In such cases, if splenectomy alone is done and there is subsequent need for a portal-systemic shunt, it may be found that none of the available veins is suitable for anastomosis. Most authorities are now agreed that only when congestive splenomegaly is due to thrombosis in the distal part of the splenic vein is splenectomy alone justified.

*Results of Portal-systemic Venous Shunts.* The available figures on this subject are still somewhat confusing and he would be a bold person who tried to draw too firm conclusions from them. The intrinsic and relative merits of the various operations cannot be easily assessed. The fact that a number of venous anastomoses become blocked by subsequent thrombosis, and that complications are not uncommon, should impel the surgeon to avoid such operations unless the available vessels are large and other circumstances are favourable. The routine use of anticoagulants, in an attempt to prevent blockage of the anastomosis by thrombus, is generally inadvisable in the early post-operative period, since hæmorrhage can be a more serious hazard than thrombosis.

**Operations for Relief of Ascites.** With an adequate protein diet and low salt intake, control of ascites is obtained in most cases. Occasionally this is not so, and the necessity for frequent paracentesis is inconvenient and deprives the patient of valuable protein. In such cases various attempts have been made to drain the ascitic fluid away into the subcutaneous tissues. One of the simplest measures consists in inserting a special metal or plastic "button" between the peritoneal cavity and the subcutaneous tissues. Sometimes the channel through the "button" becomes blocked by omentum and a fresh one has to be inserted. Alternatively the great omentum has been implanted in the rectus sheath, but this is not so satisfactory and may cause an incisional hernia.

## CHAPTER 6

### THE SPLEEN

by

A. A. G. LEWIS and R. S. MURLEY

THE surgery of the spleen itself is limited to removal of the organ for a variety of conditions. Splenorenal venous anastomosis, which is sometimes indicated in cases of congestive splenomegaly, is discussed with disease of the liver (Chap. 5).

The first elective splenectomy was done on 1826 for a patient with cirrhosis hepatis who survived only a few hours. In the latter part of the nineteenth century the mortality rate of splenectomy was very high, chiefly due to selection of unsuitable cases for operation. At the present time, frequently with the help and guidance of the physician, the surgeon is able to select patients for operation much more precisely and with virtual certainty of benefit.

Accessory spleens, usually from 1 to 2 cm. in diameter, are sometimes found in the region of the hilum or pedicle of the spleen. They are usually single; they are commoner in children than in adults; and, if overlooked, may cause a relapse when splenectomy has been performed for a blood dyscrasia.

*Indications for Splenectomy.* The indications for splenectomy are included in the following groups:

1. *Rupture.* Due to accident or operation.
2. *In the course of an upper abdominal resection*, e.g. for cancer of the stomach or pancreas.
3. *Certain blood diseases*, notably idiopathic thrombocytopenic purpura and hæmolytic anæmia.
4. *Congestive splenomegaly.*
5. *Miscellaneous conditions.* Hypersplenism, lymphosarcoma and Hodgkin's disease of the spleen, cysts of the spleen.

**Rupture of the Spleen.** Rupture of the spleen is not uncommonly due to road accidents. In about one case in five there is damage to the left kidney; occasionally the liver too is ruptured. Associated damage to other viscera is uncommon, but fracture of the ninth, tenth or eleventh ribs occurs in about a third of cases. Sometimes bleeding from a small tear is slow or is delayed for a few hours or even days. Delayed bleeding rarely occurs more than a fortnight after injury, and usually starts within a week.

Damage to the spleen during an abdominal operation may be due to injury by an instrument, but more often is the result of traction on the

omentum causing a tear near the lower pole or hilum. The surgeon learns to handle the spleen with the greatest care, for it is certainly the most friable organ in the abdomen.

*Clinical Features.* There may be a history of a blow on the left flank or lower ribs. Pain generally occurs in the left upper quadrant, but is variable. Referred pain in the left shoulder is fairly constant and occurs in the great majority of cases, and vomiting occurs in about 50 per cent. The physical signs are those of internal hæmorrhage, and the degree of shock varies with the volume and rate of blood loss. The pulse rate, though often normal in cases of immediate rupture, is commonly raised in those with delayed bleeding. The blood pressure is generally low. Upper abdominal tenderness and rigidity are usually, but not invariably, present. Auscultation of the abdomen is of no positive value as bowel sounds are variable. Accumulation of blood and clot in the left flank may cause dullness to percussion, but this and occasional shifting dullness are unreliable signs. A plain X-ray film of the abdomen, apart from showing fracture of the lower left ribs, occasionally reveals an opacity in the left upper quadrant and displacement of the shadows of stomach and splenic flexure due to collection of blood clot.

*Treatment.* Splenectomy is always indicated. No attempt should be made to suture even a small tear, as secondary hæmorrhage is liable to occur. Blood transfusion is commonly needed. Prior to operation the urine is examined for blood and a gastric aspiration tube is passed.

*Splenectomy during other Operations.* Although it is frequently desirable to remove the spleen in the course of resection of an upper abdominal cancer (e.g. of stomach or pancreas), the tendency for some surgeons to regard the spleen as a freely "expendable" organ is to be deprecated. Damage to the spleen during an upper abdominal operation occasionally necessitates its removal, as in the course of a partial gastrectomy for peptic ulcer.

### BLOOD DISEASES

Splenectomy may be indicated in two main groups of disorder: idiopathic thrombocytopenic purpura and hæmolytic anæmia.

*Idiopathic Thrombocytopenic Purpura.* This disease is characterised by spontaneous purpura and bleeding from mucous membranes, with increased capillary fragility and bleeding time, and reduced platelet count. The spleen is not usually palpable. Acute episodes may occur, especially in young women, often with severe bleeding from mucous membranes. A chronic form, sometimes with acute exacerbations, is commoner in older patients. Purpura occurring in other blood diseases, in severe infections, or due to drugs, and the form known as the Schönlein-Henoch syndrome (often with rheumatic manifestations and renal damage) must be excluded. Spontaneous recovery from acute episodes is common. This cannot be predicted, however, and severe hæmorrhages may make transfusion

## CHAPTER II

### THE SPLEEN

by

A. A. G. LEWIS and R. S. MURLEY

THE surgery of the spleen itself is limited to removal of the organ for a variety of conditions. Splenorenal venous anastomosis, which is sometimes indicated in cases of congestive splenomegaly, is discussed with disease of the liver (Chap. 5).

The first elective splenectomy was done on 1826 for a patient with cirrhosis hepatis who survived only a few hours. In the latter part of the nineteenth century the mortality rate of splenectomy was very high, chiefly due to selection of unsuitable cases for operation. At the present time, frequently with the help and guidance of the physician, the surgeon is able to select patients for operation much more precisely and with virtual certainty of benefit.

Accessory spleens, usually from 1 to 2 cm. in diameter, are sometimes found in the region of the hilum or pedicle of the spleen. They are usually single; they are commoner in children than in adults; and, if overlooked, may cause a relapse when splenectomy has been performed for a blood dyscrasia.

*Indications for Splenectomy.* The indications for splenectomy are included in the following groups:

1. *Rupture.* Due to accident or operation.
2. *In the course of an upper abdominal resection,* e.g. for cancer of the stomach or pancreas.
3. *Certain blood diseases,* notably idiopathic thrombocytopenic purpura and hæmolytic anæmia.
4. *Congestive splenomegaly.*
5. *Miscellaneous conditions.* Hypersplenism, lymphosarcoma and Hodgkin's disease of the spleen, cysts of the spleen.

**Rupture of the Spleen.** Rupture of the spleen is not uncommonly due to road accidents. In about one case in five there is damage to the left kidney; occasionally the liver too is ruptured. Associated damage to other viscera is uncommon, but fracture of the ninth, tenth or eleventh ribs occurs in about a third of cases. Sometimes bleeding from a small tear is slow or is delayed for a few hours or even days. Delayed bleeding rarely occurs more than a fortnight after injury, and usually starts within a week.

Damage to the spleen during an abdominal operation may be due to injury by an instrument, but more often is the result of traction on the

omentum causing a tear near the lower pole or hilum. The surgeon learns to handle the spleen with the greatest care, for it is certainly the most friable organ in the abdomen.

*Clinical Features.* There may be a history of a blow on the left flank or lower ribs. Pain generally occurs in the left upper quadrant, but is variable. Referred pain in the left shoulder is fairly constant and occurs in the great majority of cases, and vomiting occurs in about 50 per cent. The physical signs are those of internal hæmorrhage, and the degree of shock varies with the volume and rate of blood loss. The pulse rate, though often normal in cases of immediate rupture, is commonly raised in those with delayed bleeding. The blood pressure is generally low. Upper abdominal tenderness and rigidity are usually, but not invariably, present. Auscultation of the abdomen is of no positive value as bowel sounds are variable. Accumulation of blood and clot in the left flank may cause dullness to percussion, but this and occasional shifting dullness are unreliable signs. A plain X-ray film of the abdomen, apart from showing fracture of the lower left ribs, occasionally reveals an opacity in the left upper quadrant and displacement of the shadows of stomach and splenic flexure due to collection of blood clot.

*Treatment.* Splenectomy is always indicated. No attempt should be made to suture even a small tear, as secondary hæmorrhage is liable to occur. Blood transfusion is commonly needed. Prior to operation the urine is examined for blood and a gastric aspiration tube is passed.

*Splenectomy during other Operations.* Although it is frequently desirable to remove the spleen in the course of resection of an upper abdominal cancer (e.g. of stomach or pancreas), the tendency for some surgeons to regard the spleen as a freely "expendable" organ is to be deprecated. Damage to the spleen during an upper abdominal operation occasionally necessitates its removal, as in the course of a partial gastrectomy for peptic ulcer.

### BLOOD DISEASES

Splenectomy may be indicated in two main groups of disorder: idiopathic thrombocytopenic purpura and hæmolytic anæmia.

*Idiopathic Thrombocytopenic Purpura.* This disease is characterised by spontaneous purpura and bleeding from mucous membranes, with increased capillary fragility and bleeding time, and reduced platelet count. The spleen is not usually palpable. Acute episodes may occur, especially in young women, often with severe bleeding from mucous membranes. A chronic form, sometimes with acute exacerbations, is commoner in older patients. Purpura occurring in other blood diseases, in severe infections, or due to drugs, and the form known as the Schönlein-Henoch syndrome (often with rheumatic manifestations and renal damage) must be excluded. Spontaneous recovery from acute episodes is common. This cannot be predicted, however, and severe hæmorrhages may make transfusion

necessary. *Cortisone* (up to 300 mg. daily) or *ACTH* (20 to 80 i.u. of gel daily) frequently produce a rapid remission which may be permanent, especially if the disease is of recent onset. Often there is an improvement without any change in the platelet count. Splenectomy should be considered in the more chronic cases in which steroid therapy has had little effect. It may benefit particularly those patients in whom sternal puncture shows the presence of numerous megakaryocytes in the bone marrow. It is very rarely necessary in the acute forms in younger patients.

**Hæmolytic Anæmia.** This disorder is characterised by increased destruction of circulating red cells, with an increase in the level of *serum bilirubin* (often sufficient to cause jaundice), an absence of this pigment from the urine (unless biliary obstruction occurs from the pigment stones which are a common complication), and an increased *fecal urobilinogen* (more than the normal excretion of 300 mg. per day, estimated in a three-day sample of faeces). The latter is the most specific indication available of increased hæmolysis, as survival studies of transfused cells are rarely practicable. Increased formation of red cells is shown in the marrow and by the high reticulocyte count. Hæmolytic anæmia may be a manifestation of a collagen disease, of a reticulosis or a leukaemia (particularly the chronic lymphatic form), or it may be due to drugs or bacterial toxins, or to congenital abnormalities of the erythron. Hæmolysis may be due to the circulation of abnormal antibodies, usually for no known reason. Splenectomy is only indicated in certain forms of the last two varieties, namely *hereditary spherocytosis* and "*acquired hæmolytic anæmia*" (auto-immune hæmolytic anæmia).

*Hereditary spherocytosis* (congenital hæmolytic anæmia) is inherited as a Mendelian dominant; the severity of the disease varies from a barely detectable increase in the osmotic fragility of the red cells (which hæmolyse more readily than normal when incubated with saline) to a form in which anæmia and jaundice may be clinically obvious. Sudden relapses are common, especially after infections. The diagnosis depends on: (1) the family history of anæmia and jaundice, and (2) evidence of hæmolysis (see above) and of increased red cell fragility. The occurrence of spherocytes (red cells of decreased diameter and increased thickness) is not peculiar to this form of hæmolytic anæmia. The spleen is commonly palpable and it is in this organ that the majority of the abnormal cells are destroyed. Splenectomy therefore cures the great majority of patients and greatly improves the condition of the remainder. It should be advised in all but the very mildest forms of the disease for there is always a risk of more severe episodes and of pigment stone formation in the biliary tract.

*Acquired hæmolytic anæmia* is characterised by the formation of abnormal auto-antibodies which are adsorbed by the red cells which therefore invariably give a positive direct Coombs's reaction (agglutina-

tion *in vitro* in the presence of anti-human globulin). The cells are probably agglutinated *in vivo* and destroyed by local tissue haemolysins in the reticulo-endothelial system. The disease is commonly seen in older patients, presenting with anaemia, jaundice and splenomegaly. Acute exacerbations may occur, with fever, rapidly falling haemoglobin level, deepening jaundice and marked reticulocytosis and leucocytosis. Every effort must be made to exclude known causes of the condition, including diseases such as virus pneumonia and syphilis, in which auto-agglutinins may be formed which are active in the cold. A positive W.R. may, however, occur in any form of the disease and be misleading.

In acute episodes transfusion is usually necessary, but cross matching may be made extremely difficult by the presence of the abnormal antibodies. It should be performed at the transfusion depot where many bottles of blood can be tested for compatibility by experts. Transfusion reactions are common however carefully this is done, even when the blood is given very slowly.

When no cause for the condition can be found, two forms of therapy should be considered: the use of steroid hormones or splenectomy. A large number of patients may be tided over to a spontaneous remission by cortisone or ACTH. Large doses may be necessary (300 mg. or more of the former or up to 160 i.u. of the latter daily). Occasionally a patient who fails to respond to one of these drugs may do so to the other. In other patients, however, splenectomy may have to be undertaken, for this removes one of the major sites of red cell destruction. The disease can be controlled by this operation, or by steroid hormone therapy afterwards, in a considerable proportion of cases. There are some, however, in whom haemolysis continues at a rapid rate and in whom repeated transfusion at decreasing intervals becomes impossible.

**Congestive Splenomegaly.** This title is probably the most appropriate one for certain cases in which splenomegaly, anaemia, bleeding from the gastro-intestinal tract and ascites are present either separately or in combination. Formerly called "*Banti's disease*," no single factor is responsible for this condition. Obstruction may occur in the pre-hepatic, intra-hepatic or post-hepatic veins and some of the possible causes are shown in Fig. 49. This is discussed more fully elsewhere (Chap. 5). The association of this condition with *haematocytopenia* constitutes a further indication for splenectomy. Occasionally a similar state of affairs is produced by thrombosis in the splenic vein.

Splenectomy can be very difficult in some of these cases due to troublesome vascular adhesions between spleen and diaphragm. When there is associated portal hypertension splenectomy may be a preliminary step to end-to-side spleno-renal venous anastomosis (Chap. 5).

**Miscellaneous Conditions.** Increased destruction of the formed elements of the blood may occasionally occur in a spleen enlarged in a number of conditions (e.g. in congestive splenomegaly or in lipid storage disorders



such as Gaucher's disease). This is usually known as *hypersplenism*, and, rarely, may necessitate splenectomy. *Hodgkin's disease* and *lymphosarcoma* occasionally produce very large spleens with no evidence of the disease elsewhere, at any rate in the early stages. Splenectomy should then be performed. *Cysts of the spleen* are rare but should be remembered as a cause of splenomegaly, especially in a young patient. The organ

should be removed: frequently the nature of the condition can only be diagnosed in this way.

**The Operation of Splenectomy.** Left paramedian, transverse or subcostal incisions may be used according to the circumstances of the case and surgical preference. An adherent spleen in congestive splenomegaly may be best exposed through a thoraco-abdominal incision. Whatever exposure is used the first step is to divide the *spleno-colic ligament*. The *spleno-renal fold* of peritoneum is then incised and the organ lifted gently forward. The gastro-splenic ligament is ligated and divided in segments (Fig. 50); the spleen can now be turned to the right and its pedicle carefully

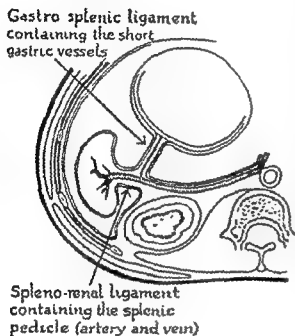


FIG. 56. The splenic ligaments.

freed from the pancreas. The vessels are doubly ligated and divided and the spleen is removed. With a very large and vascular spleen it is often wise to expose the splenic artery and ligate it at an early stage. The application of a faradic current to the pedicle or injection of adrenaline will cause the organ to contract before removal.

**Complications.** These include: (1) left lower lobe pulmonary collapse, (2) pancreatic fistula (sometimes encouraging wound dehiscence), (3) paralytic ileus, (4) deep venous thrombosis. Haemorrhage from the pedicle, formerly not uncommon, is not often seen to-day. Other troublesome complications include hiccup, which is probably due to diaphragmatic irritation, and slight haematemesis, which is probably due to congestion of the gastric mucosa following ligation of the short gastric vessels. An unusual but interesting complication is splenosis in which nodules of splenic tissue are transplanted on to the peritoneum by injury or operation.

## CHAPTER 7

# THE PERITONEUM, OMENTUM AND MESENTERY

## PERITONITIS

INFLAMMATION of the peritoneum is a complication of many intra-abdominal conditions. Peritonitis, although usually bacterial in origin, is occasionally due to irritation of the peritoneum by bile, urine, blood or the contents of a ruptured intra-abdominal cyst. The initial features of perforated peptic ulcer are caused by chemical irritation of the peritoneum and this is shortly followed by bacterial peritonitis.

**Causes.** The common causes of peritonitis are classified as follows :—

1. *Perforation or Necrosis of Gut.*
  - (a) Perforation of appendix, peptic ulcer, diverticulum or growth.
  - (b) Gangrenous appendix, strangulation of gut.
  - (c) Post-operative leakage from gut.
2. *Inflamed Lymph Glands.*
  - (a) A not uncommon source of tuberculous peritonitis.
  - (b) A rare source of pyogenic peritonitis.
3. *Genital Tract Infection.*
  - (a) Salpingitis and criminal abortion.
  - (b) Pneumococcal peritonitis in little girls.
4. *External Wounds.*
5. *Blood Stream Infection.*

In actual practice the commonest causes of peritonitis are appendicitis, perforated peptic ulcer and complications of operations on the gut.

**Pathology.** The bacterial flora of the peritoneum can be most difficult to assess in cases of peritonitis. Stained smears of the exudate, rather than cultures, may give the most accurate information about the predominant organism. The pathogenicity of the organisms may be unrelated to their frequency in smear or culture, and it will be appreciated, therefore, that the bacteriological assessment of peritoneal exudate is no easy task. The commonest organisms present are : *E. coli* ; hæmolytic streptococci ; various types of clostridia and staphylococci. Attention has recently been drawn to the possible importance of Gram-negative non-sporeing anaerobic bacilli of the genus *Bacteroides*, which are inhabitants of the mouth and lower bowel. The course of the disease depends upon the source of origin, the type and severity of infection, the defences of the host and the treatment adopted.

The spread of infection in the peritoneum is influenced partly by the disposition of the viscera which form natural barriers and watersheds, and partly by the position of the patient, since exudate and pus naturally

tend to gravitate to the dependent parts. The body may attempt to limit spread of infection in several ways ; first, by limiting movement of the abdominal muscles and diaphragm ; secondly, by inhibiting peristalsis ; and, lastly, by formation of adhesions between the viscera, omentum and abdominal wall. Diminished peristalsis may result in paralytic ileus, and the subsequent formation of adhesions may cause mechanical obstruction to the gut. When the infection is successfully localised, abscess formation may occur at the site of the original disease, or in any dependent part to which pus has tracked.

**Clinical Features.** The clinical picture of peritonitis is related to the primary disease, to the degree of toxæmia and to the effects of paralytic or mechanical ileus. The variability of these factors is responsible for the wide variation in the symptoms and signs of peritonitis. Initially the symptoms are those of the causative condition, and only later is there any common pattern of symptoms. Pain may firstly be due to the cause and later becomes more generalised. The associated paralytic ileus may result in nausea and vomiting. Usually no flatus is passed and occasionally there is absolute constipation. On the other hand diarrhœa is not uncommon with pelvic peritonitis or abscess formation. The abdomen tends to become distended, and distension may eventually be marked. In the more acute types of infection, localised and then more general rigidity is common, but in elderly patients and post-operative cases, rigidity is often conspicuous by its absence. Indeed, in these last two groups of cases, the diagnosis of peritonitis is sometimes exceedingly difficult. In the severe and more advanced cases of peritonitis, the patient tends to lie motionless, with drawn and anxious face, subnormal temperature and a rapid pulse of poor volume.

**Treatment.** Many of the details of treatment are discussed with the individual diseases, but the principles of proper management include : (1) treatment of the primary disease ; (2) proper positioning of the patient ; (3) rest to the bowel ; (4) attention to fluid and electrolyte balance ; (5) appropriate antibiotic therapy.

The primary disease necessitates appropriate treatment. The best position for the patient is generally the one in which he is most comfortable. A semi-recumbent posture with the head of bed raised or with the trunk half propped up is usually best. The formerly popular high Fowler position is unsatisfactory for two reasons : firstly, because it is uncomfortable to maintain, and, secondly, because the hips and knees are bent with consequent impairment of venous return from the legs. Rest to the bowel is achieved by withholding all fluids and food by mouth, by gastric aspiration and by suitable parenteral fluid therapy. The regular subcutaneous injection of Omnopon or morphine, in small doses, not only relieves pain but also helps to rest the bowel. Caution must be observed to avoid unnecessarily large dosage with morphine, and, in very ill patients, the action of such drugs can be more precisely gauged if they are given

intravenously. *Parenteral fluids* are usually best given intravenously; the fluid and electrolyte balance is intelligently controlled by careful clinical observation, well-kept fluid balance charts, and close observation of the blood chemistry. *Antibiotic and chemotherapy* are largely empirical since it is rarely practicable to wait for detailed bacteriological investigation before starting treatment. Furthermore, as already stressed, the investigation of the bacterial flora can be difficult. General knowledge and experience of the different types of peritonitis has shown that penicillin and sulphonamide are commonly effective. It is usual to give an adult patient crystalline penicillin (0.5 mega unit, six-hourly intramuscularly), together with a soluble sulphonamide preparation, such as sulphadimidine (1.5 gm., six-hourly). Although streptomycin is commonly prescribed for cases of peritonitis it would seem that it is of limited value. Reference was earlier made to the importance of the genus *Bacteroides* of which there are four main groups (*fusiformis*, *necrophorus*, *melaninogenicum* and *fragilis*) all of which are resistant to streptomycin. On the other hand the first three groups are all sensitive to penicillin, whereas the last group (*Bacteroides fragilis*) is resistant to penicillin but sensitive to oxytetracycline. Since the *fragilis* group is closely associated with the large bowel it would seem rational to give oxytetracycline in cases of severe peritonitis arising from this source, but to use penicillin and sulphadimidine in the other cases.

### SPECIAL TYPES OF PERITONITIS

Certain special types of peritonitis deserve further mention. *Gonococcal peritonitis* is uncommon and confined to females, being secondary to salpingitis in the adult or vulvo-vaginitis in the child. It tends usually to be pelvic in distribution and dense adhesions are commonly formed. *Pneumococcal peritonitis* occurs in a so-called *primary* variety which is seen in little girls under seven years of age, particularly in poorer districts. This type of peritonitis, which is not so common now as formerly, is believed to be due to ascending infection via the genital tract. Since in this and the gonococcal type of peritonitis the infection is primarily pelvic there is often diarrhoea and painful frequency of micturition. There is a *secondary* type of pneumococcal peritonitis which rarely follows pneumonia or otitis media.

Clinically, pneumococcal peritonitis may simulate appendicitis on the one hand or, on the other hand, be so chronic as to resemble tuberculous peritonitis. If the abdomen is opened the exudate is seen to be sticky, greenish, and sometimes blood-stained and purulent. Most cases respond to large doses of penicillin. *Streptococcal peritonitis*, due to  $\beta$  haemolytic streptococci, is an unusual variety which may follow puerperal infection, and more rarely scarlet fever. It usually responds to treatment with penicillin or sulphonamides.

**Tuberculous Peritonitis.** Tuberculous peritonitis is very much less

common than it was and this is chiefly due to the marked reduction in all forms of bovine infection. It is commonest in children and young adults, in whom it is usually secondary to an abdominal focus of infection. The commonest primary sources of infection are the bowel, mesenteric glands and Fallopian tubes. Occasionally, it is secondary to extra-peritoneal infection, e.g. in the urinary tract, and sometimes it spreads from the chest, or from bones, joints and distant lymph glands. The disease may present in acute or chronic forms.

*Acute tuberculous peritonitis* is often a manifestation of generalised miliary tuberculosis, but occasionally the peritoneum is the only obvious site of disease. Multiple tiny tubercles are scattered over the parietal and visceral peritoneum; the latter may be thickened and œdematous, and free fluid is often present. Treatment is by strict rest and antibiotic therapy.

*Chronic tuberculous peritonitis* occurs in two main varieties: the *ascitic* and the *adhesive*, although combinations of the two may give rise to encysted collections of fluid. (1) *Ascitic Type*. A considerable volume of fluid may accumulate with resultant gross abdominal distension. The patient is often severely wasted and prominent veins are sometimes seen on the abdominal wall. The peritoneum may be studded with miliary tubercles as well as bigger collections of tuberculous granulation tissue. The fluid is thin, yellow or greenish in colour, and usually low in fibrin content. The great omentum often forms a stiff pancake-like mass or a rigid roll which can be palpated after paracentesis. (2) *Adhesive Type*. In this variety there are dense inflammatory adhesions with little free fluid. Sometimes coils of gut become inextricably bound together. Caseous masses are common, and the peritoneum and subserous layers may be greatly thickened. Faecal fistula is not uncommon and usually occurs between the small gut and the abdominal wall.

*Diagnosis*. In young patients there are very few other causes of ascites, but *polyserositis* is a possibility. Large *pseudo-pancreatic* and *ovarian cysts* are occasionally seen, even in children, but wasting is not present in these cases. In adult patients, *cirrhosis* and *malignant ascites* must be excluded. Once the abdomen is opened, the diagnosis is usually straightforward, but sometimes biopsy of the peritoneum is the only certain way of differentiating tuberculous and malignant ascites. The exudate in tuberculous peritonitis occasionally contains many tubercle bacilli.

*Treatment*. The ascitic type sometimes benefits from laparotomy. Apart from this, surgical treatment is of limited value and will only be needed in a few cases, either to establish a doubtful diagnosis or to deal with a complication such as faecal fistula or intestinal obstruction. Some of these patients are already under sanatorium care for tuberculosis when the abdominal condition appears. Full hospital care is imperative, and this entails strict bed rest and antibiotic therapy; streptomycin (1 gm.

daily) is combined with either Isoniazid (100 mg. b.d.) or para-aminosalicylic acid (20 gm. daily). Isoniazid is usually more suitable than para-aminosalicylic acid as it causes fewer side effects.

### LOCALISED INTRAPERITONEAL ABSCESS

Localised intraperitoneal abscess most often occurs in relation to the primary disease, and in the pelvis or subphrenic region. Perforated peptic ulcer and acute appendicitis are the commonest causes. Many such abscesses resolve; some discharge spontaneously (either to the surface or into the bowel), and some require drainage. Whenever possible such collections are drained by an extraperitoneal approach to avoid undue contamination of the peritoneum. Sometimes these abscesses remain dormant for long periods of time and may later flare up again.

### SUBPHRENIC ABSCESS

Subphrenic abscess is usually pyogenic in origin, but sometimes it is due to other types of infection such as tuberculosis or actinomycosis. The pus may collect in several possible positions in relation to the main lobes of the liver: (1) The right subhepatic space (hepato-renal space of Morison) which lies below and partly behind the right lobe of the liver. It is most often infected from the appendix, duodenum and right colon. (2) The right subdiaphragmatic space which is above the liver and anterior to the triangular ligament. It is most often infected following a perforated peptic ulcer. (3) The right extraperitoneal space which is behind the bare area of the liver and may be infected from the liver, kidney or retrocaecal appendix. (4) The left posterior space is the lesser sac which is usually infected from the stomach or pancreas. (5) The left subdiaphragmatic and anterior subhepatic spaces which are both small and most usually contaminated following a gastric perforation.

**Pathology.** At least 50 per cent. of cases are due to gastric, duodenal or appendicular disease. The others follow infections from the colon, liver, biliary tract, pelvic and other sources. Rarely, an empyema tracks through the diaphragm, but more commonly empyema is a sequel to a subphrenic abscess. The abscess is most often due to the same pyogenic organisms responsible for the peritonitis. In some cases anaerobic bacteria cause large quantities of gas to collect in the abscess. The pus varies from very thin to excessively thick with a marked tendency to loculate. Spontaneous resolution occasionally occurs, but an untreated abscess tends to discharge through the skin or to rupture into the gut, pleura or pericardium.

**Clinical Features.** Characteristically, following appendicitis or repair of a perforated ulcer, there is failure of resolution or recrudescence of pyrexia. Sometimes the patient is very toxic and ill with hectic fever and marked leucocytosis. There may be shoulder tip pain and pain in

relationship to the abscess and, if it is presenting near the parietes, there may be subcostal or renal angle tenderness. The liver is often readily palpable if the abscess is above it and there may be obvious signs of a raised diaphragm. Only rarely is it possible on percussion to elicit the classical four zones :—resonance over normal lung ; dullness over the collapsed lower margin of lung or basal effusion ; resonance due to gas in the abscess and, finally, dullness over the liver and pus.

Plain X-ray films reveal elevation of the diaphragm and frequently a pleural effusion. Subdiaphragmatic gas is not uncommon, and is best shown with the patient erect, or lying on the side with the abscess uppermost. A fluid level may be clearly discerned below the gas. The gas in an abscess may be partly due to the pneumoperitoneum of a perforation and partly due to generation by anaerobic organisms. Subhepatic abscesses are sometimes best demonstrated on X-ray with the aid of a barium meal, which shows displacement of the stomach. The diagnosis of subphrenic abscess is not difficult, providing it is thought of. A hectic fever and marked leucocytosis following appendicitis or a perforation should, in the absence of any other obvious cause, be assumed to be due to a subphrenic abscess until proved otherwise. In the absence of gas and a fluid level on X-ray, the diagnosis of liver abscess and pylephlebitis must equally well be considered. Occasionally, a certain diagnosis is only established by careful exploratory needling in the theatre.

**Treatment.** Once the diagnosis is made early drainage is indicated, and no reliance should be placed on antibiotic therapy, excepting as an adjuvant measure. The pus should be evacuated by the simplest and most accessible route. On the right side resection of the twelfth rib and incision of the diaphragm can provide good access and dependent drainage whether the abscess lies above, behind or below the liver. Abscess at the latter site (i.e. right subhepatic), and also in the left posterior space, can be drained by an incision below the appropriate last rib. Anterior abscesses on either side sometimes present at the costal margin and are best drained by an anterior subcostal incision. In all cases an adequate drain of corrugated rubber or a soft tube is inserted and is kept in until it is clear that there is complete drainage and progressive healing without pocketing.

### PELVIC ABSCESS

Pelvic abscess may result from any type of pyogenic infection and also from tuberculosis of the pelvic organs. Pus may also gravitate to the pelvis from elsewhere in the abdomen. The commonest causes are appendicitis, diverticulitis coli and genital tract infections in the female. The general features are similar to those of subphrenic abscess, but there are usually characteristic localising signs. Irritation of the bowel results in diarrhoea, and sometimes the passage of much mucus. Painful frequency of micturition may occur too. Rectal examination reveals a

boggy and somewhat tender swelling anteriorly, and occasionally a large abscess is palpable per abdomen.

Resolution or spontaneous discharge of pus per rectum or per vaginam may occur. When a mass is clearly presenting per rectum it is easiest to drain the abscess there, but sometimes the abdominal route is preferable.

### OTHER DISEASES OF THE PERITONEUM

**Ascites.** The collection of ascitic fluid in the peritoneum may be due to general or local causes. The former include cardiac failure, renal failure and depletion of the plasma proteins. The local causes are portal vein obstruction by cirrhosis of the liver, or thrombosis, blockage of lymphatic vessels by inflammation or neoplasm, and tumours of the ovary. The character of the fluid varies a good deal with the underlying cause. Generally speaking, the specific gravity and protein content are higher when there is associated inflammation, and bloodstaining is common in cases of neoplasm.

**Peritoneal Tumours.** Primary tumours of the peritoneum are exceedingly rare whereas secondary deposits are common. Carcinoma of the stomach or ovary is the most frequent primary site, and other common abdominal sources of such tumours are the colon and pancreas. Spread of the disease may be by direct involvement, transperitoneal seeding and by blood or lymphatic vessels. *Direct spread* to the peritoneum is liable to occur with all gut tumours in the course of time. *Transperitoneal spread* is a feature of some gastric carcinomas. Thus, direct implantation on the ovaries is not uncommon, and it is likely that the so-called Krukenberg tumours occur in this way. *Blood and lymph vessel spread* are common. Both may bring tumour cells from outside the abdomen, and lymph-vessel spread into the peritoneum is common when the mesenteric and retroperitoneal nodes are extensively involved. If ascites is present it may be possible to identify malignant cells in the centrifuged deposit.

**Pseudomyxoma Peritonei.** Localised or diffuse collections of gelatinous material (pseudomucin) are seen in connection with certain affections of the ovary and appendix. The commonest ovarian source is a pseudomucinous cystadenoma, but the peritoneal involvement is rarely due to rupture of the tumour. It would seem most likely that the pseudomucin is formed on the peritoneum by cellular metastasis from the primary tumour even though the latter may otherwise have no malignant characteristics. Pseudomucin evokes a peritoneal reaction which may result in the formation of fibrous adhesions. Of the appendicular causes, the condition is most often due to rupture of a *mucocoele*, and occasionally to a ruptured diverticulum or adenocarcinoma of the appendix.

**Retroperitoneal and Mesenteric Tumours.** Strictly speaking retroperitoneal tumours arise only from the fat, connective tissue, nerves or lymph nodes. Those arising from the pancreas, kidneys or other retro-



relationship to the abscess and, if it is presenting near the parietes, there may be subcostal or renal angle tenderness. The liver is often readily palpable if the abscess is above it and there may be obvious signs of a raised diaphragm. Only rarely is it possible on percussion to elicit the classical four zones :—resonance over normal lung ; dullness over the collapsed lower margin of lung or basal effusion ; resonance due to gas in the abscess and, finally, dullness over the liver and pus.

Plain X-ray films reveal elevation of the diaphragm and frequently a pleural effusion. Subdiaphragmatic gas is not uncommon, and is best shown with the patient erect, or lying on the side with the abscess uppermost. A fluid level may be clearly discerned below the gas. The gas in an abscess may be partly due to the pneumoperitoneum of a perforation and partly due to generation by anaerobic organisms. Subhepatic abscesses are sometimes best demonstrated on X-ray with the aid of a barium meal, which shows displacement of the stomach. The diagnosis of subphrenic abscess is not difficult, providing it is thought of. A hectic fever and marked leucocytosis following appendicitis or a perforation should, in the absence of any other obvious cause, be assumed to be due to a subphrenic abscess until proved otherwise. In the absence of gas and a fluid level on X-ray, the diagnosis of liver abscess and pylephlebitis must equally well be considered. Occasionally, a certain diagnosis is only established by careful exploratory needling in the theatre.

**Treatment.** Once the diagnosis is made early drainage is indicated, and no reliance should be placed on antibiotic therapy, excepting as an adjuvant measure. The pus should be evacuated by the simplest and most accessible route. On the right side resection of the twelfth rib and incision of the diaphragm can provide good access and dependent drainage whether the abscess lies above, behind or below the liver. Abscess at the latter site (i.e. right subhepatic), and also in the left posterior space, can be drained by an incision below the appropriate last rib. Anterior abscesses on either side sometimes present at the costal margin and are best drained by an anterior subcostal incision. In all cases an adequate drain of corrugated rubber or a soft tube is inserted and is kept in until it is clear that there is complete drainage and progressive healing without pocketing.

### PELVIC ABSCESS

Pelvic abscess may result from any type of pyogenic infection and also from tuberculosis of the pelvic organs. Pus may also gravitate to the pelvis from elsewhere in the abdomen. The commonest causes are appendicitis, diverticulitis coli and genital tract infections in the female. The general features are similar to those of subphrenic abscess, but there are usually characteristic localising signs. Irritation of the bowel results in diarrhoea, and sometimes the passage of much mucus. Painful frequency of micturition may occur too. Rectal examination reveals a

commoner in fat people and occurs in two forms. The cause of *primary torsion* is unknown, but the twist is always clockwise, and strangulation of the omentum tends to occur. *Secondary torsion* occurs when the omentum is adherent to the inside of a hernial sac or other structure. The rest of the omentum rotates about the axis of attachment. The symptoms of omental torsion may resemble those of appendicitis or cholecystitis. A lump is sometimes palpable, but the correct diagnosis is not often made before operation. The treatment is removal of the damaged part of the omentum.

*Peritoneal Loose Bodies.* Smooth pearl-like bodies of variable size are occasionally seen in the peritoneum. Most of these are necrotic pieces of fat which have become detached from the omentum, appendices epiploicæ and other fatty fringes. On section they often resemble hard-boiled eggs since they have an outer layer of white fibrous tissue and a small centre of bright yellow necrotic fat. Such loose bodies rarely give rise to any symptoms and they are sometimes items of interest at laparotomy.

### THE MESENTERY

The connective tissues of the mesentery are subject to similar diseases to the retroperitoneal structures and no further description of these is needed. *Mesenteric Lymphadenitis.*—Enlargement of the lymphatic glands of the mesentery may be secondary to a variety of inflammatory conditions. Primary mesenteric lymphadenitis occurs, however, in non-specific and tuberculous forms. The former condition is almost entirely confined to children and adolescents and is described in Chapter 22. Tuberculous lymphadenitis, generally due to a bovine type of infection, is very much less common than it was. In the majority of cases there is no obvious primary lesion in the gut although the infection must presumably have originated there. Abscess formation may occur and it is not uncommon for the caseous material and the glands to become calcified. Adhesion to neighbouring structures may cause obstruction or, more rarely, traction diverticulum.

*Mesenteric Vascular Occlusion.* Occlusion of the mesenteric veins is invariably due to thrombosis, but arterial occlusion may be due to embolism or thrombosis. Neither condition is commonly seen by the surgeon.

*Pathology.* *Arterial occlusion* is due to an embolus which may arise from the heart (e.g. vegetation from the mitral or aortic valve, or a mural thrombus from the auricular appendix or ventricle). An embolus may also arise from an atheromatous plaque in the aorta. Due to its size, the superior mesenteric artery is much more often involved than the inferior mesenteric. The arterial flow is arrested, vascular spasm is common and the associated veins become highly engorged. Infarction of the gut usually ensues in a few hours. If a smaller embolus impacts in one of the more distal vessels the initial effects are much less dramatic. However,

peritoneal organs should not be included. *Lipomas* in this situation sometimes grow to huge dimensions when they are commonly sarcomatous. *Fibrosarcoma* also occurs, and both these tumours tend to spread widely behind the peritoneum and between the layers of the mesentery. Tumours of nerve tissue include *neurofibroma*, *neuroblastoma*, *ganglioneuroma* and *chromaffinoma*.

Tumours of the lymphatic glands include *secondary deposits*. Notable amongst these are metastasis from *seminoma* or *teratoma of the testis* which may be mistaken for a primary retroperitoneal sarcoma. Other tumours of lymphoid tissue, such as *Hodgkin's disease*, *lymphosarcoma* and *reticulosarcoma* may also occur.

**Peritoneal Adhesions.** The formation of adhesions is fundamental both to the healing of gut anastomoses and to the localisation of infection in the abdomen. Only when the process becomes perverted or excessive does it constitute a troublesome or potentially dangerous state of affairs. Adhesion formation may be due to *chemical irritation*, as by gastric contents; *infection*, especially if mild, or *mechanical trauma*, as in the course of operation. Although most adhesions are temporary and harmless, persistent ones may give rise to trouble. Gonococcal infection causes the most dense adhesions, whereas virulent streptococcal infections cause no adhesions at all. The persistent adhesions are converted into fibrous tissue which may form broad sheets or narrow bands. There is no doubt that some people are especially prone to adhesion formation at the slightest provocation. Others, despite repeated and extensive surgical operations, may show no pathological adhesions at all.

**Talc Granuloma.** In this connection it is important to mention that *magnesium trisilicate* (formerly used as a glove dusting powder) can evoke a granulomatous reaction and cause dense inflammatory masses and adhesions. Starch dusting powder gives rise to much less reaction, but whatever dusting powder is used in gloves it is essential to ensure that the amount introduced is cut down to a minimum.

### THE GREAT OMENTUM

The great omentum can be both a help and a hindrance. An exceedingly fat-laden omentum can add to the difficulties of working in the abdomen of an obese patient. Fortunately, these difficult occasions are far outnumbered by those on which the omentum exercises a useful function. Thus, by becoming adherent to inflamed structures it can help to localise infection and limit its spread in the peritoneum. It also is capable of absorbing organisms from the peritoneum, and in cases of peritonitis, the omentum may be loaded with bacteria which are quite sparse in the exudate. Most of the pathological conditions seen in the omentum do not call for special description. They include cyst, pyogenic abscess, tuberculous infection, secondary carcinomatosis, and torsion.

**Torsion of the Omentum.** This is an interesting condition which is

## CHAPTER 8

### THE SMALL INTESTINE

#### DIVERTICULA OF THE SMALL GUT

**DIVERTICULA** of the small gut may be either congenital or acquired. The congenital variety includes Meckel's diverticulum and a proportion of the diverticula found elsewhere in the gut. Acquired diverticula are usually of the *pulsion* type, but sometimes pouches are formed as a result of *traction* on the wall of the gut following its adhesion to a neighbouring structure.

**Meckel's Diverticulum.** Meckel's diverticulum is found in roughly 1 per cent. of people and is situated on the antimesenteric border of the ileum. It may be anywhere from 2 inches to 3 feet from the ileocaecal valve, but is usually about 2 feet away. The lining of the diverticulum may consist of ileal, gastric or even colonic mucosa and aberrant pancreatic tissue is sometimes present too.

**Clinical Features.** The great majority of Meckel's diverticula give rise to no trouble and are discovered in the course of laparotomy or necropsy. A variety of symptoms may occur as a result of certain complications which include (Fig. 57):—(1) *Peptic ulcer*, causing pain, bleeding or even perforation; (2) *diverticulitis*, occasionally followed by perforation; (3) *intestinal obstruction* due to associated bands or adhesions following inflammation; (4) *fistula* at the umbilicus; (5) *intussusception*; (6) other rare complications such as *cyst*, *neoplasm* or *torsion*. In the majority of cases needing surgical relief the patient is a child or young adult. One of the most alarming symptoms is massive bleeding. Meckel's diverticulitis

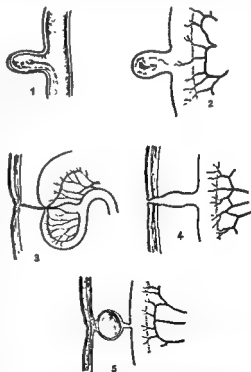


FIG. 57. Complications of Meckel's diverticulum. (1) Peptic ulcer and bleeding. (2) Inflammation and perforation. (3) Obstruction by a band. (4) Fistula. (5) Cyst formation.

retrograde thrombosis in the artery usually follows and, if this is not arrested, infarction of gut will then occur. *Primary arterial thrombosis* is very rare. It may occur in *thromboangiitis obliterans* and occasionally in patients who are especially thrombosis-prone, for example, those with advanced malignant disease or leukaemia. *Venous occlusion* may be due to portal obstruction by a cirrhotic liver, or a tumour, and it can also be secondary to inflammatory conditions such as appendicitis, diverticulitis coli and thrombosed haemorrhoids. Unless the mesenteric thrombosis is confined to the smaller peripheral veins, infarction of gut usually occurs.

*Clinical Features.* Whatever the cause the general and local effects of mesenteric vascular occlusion are similar to those of strangulation of the gut. The condition is uncommon before middle age and the majority of cases occur in old and unfit people. The severity of the symptoms depends on how sudden and extensive the occlusion has been. There is generally sudden and very severe abdominal pain, often accompanied by shock and sometimes by haematemesis or melaena. Untreated cases generally perish from obstruction or peritonitis.

*Treatment.* In cardiac cases the patients are sometimes extremely ill before the embolism occurs. On the other hand, mesenteric arterial occlusion may occur in a relatively fit and well-compensated case of mitral stenosis. In these and other fit patients the treatment of choice is by operation. After suitable preparation and resuscitation (which should not be prolonged), the abdomen is explored and the infarcted area of gut is resected. The mesentery is handled with great care to avoid aggravating the thrombosis. In a very few cases of arterial embolism, the embolus can be successfully removed. In certain very ill and poor risk cases, or in those where lesser involvement of smaller vessels is suspected, there is a good case for anticoagulant therapy. Anticoagulant drugs can also be given after operative treatment, but it is wise to withhold them for twelve hours or so, and then to exercise extreme care lest bleeding should occur (see Chap. 75).

operation. Operation is also indicated occasionally because of inflammation and obstruction of the duodenum.

In the rare cases for which operation is indicated the diverticulum is removed. Extreme care is exercised in freeing it to avoid damage to the common bile or pancreatic ducts, and this is best done by dissecting the diverticulum with a finger inside it.

*Jejunal and Ileal Diverticulum.* Diverticula of this region occur in about 0.5 per cent. of people. They are most common in the upper jejunum and almost invariably on the mesenteric border of the gut (Fig. 58). It is possible that the protrusion occurs at the site of one of the small penetrating vessels. In 50 per cent. of cases they are multiple and in about 20 per cent. there are diverticula in the duodenum or colon too. Vague abdominal pain, flatulence and vomiting occasionally occur. Acute inflammation, bleeding, strangulation and tumour formation are rare complications which may demand operation. When operation is done this usually entails resection of the affected length of gut followed by end-to-end anastomosis.

*Traction Diverticulum.* Acquired lesions of this type are rare but may occur with the scarring of a duodenal ulcer or when the gut becomes adherent to an inflamed gland. The condition causes no symptoms and does not itself call for treatment.

### NEOPLASM OF THE SMALL GUT

All tumours of the small intestine are rare. *Simple tumours* include adenoma, leiomyoma, hæmangioma, fibroma and lipoma, the first three of which are liable to bleed. Such tumours rarely cause intestinal obstruction unless complicated by intussusception. Many are accidentally discovered at operation or necropsy. Accessory pancreatic tissue is most often found around the duodenum and occasionally it is the site of an islet-cell tumour. *Malignant tumours*, also rare, are of greater importance and include carcinoid, adenocarcinoma and sarcoma.

*Carcinoid Tumour.* Carcinoid tumours are often called argentaffin or Kulschitzky cell carcinomas and, even in the absence of metastasis, they are best regarded as malignant in all cases. The fact that they sometimes secrete a hormone which may cause characteristic symptoms has only recently been appreciated. These tumours occur anywhere in the duodenum or small intestine, or even in a Meckel's diverticulum. Sometimes there are multiple tiny tumours and occasionally there is diffuse infiltration of the small vessels in the gut and mesentery. Most carcinoids are small and whitish or yellow. They are most common after middle life. On section they consist of strands or clumps of close-packed polyhedral cells, and, unlike the usually benign carcinoids of the appendix, they commonly metastasise to the liver and lymph glands.

*Clinical Features.* There are three main types of clinical presentation : (1) Bleeding ; (2) obstruction ; and (3) symptoms due to secretion of

gives rise to symptoms which are virtually identical with those of appendicitis, and it is rarely diagnosed before operation unless the appendix has previously been removed.

*Treatment.* A Meckel's diverticulum which is causing trouble should be excised. If the proximal end is narrow it may be ligated and inverted like the base of the appendix, but if it is broad the defect in the ileum is closed with two layers of sutures. Occasionally the adjacent intestine is so inflamed or infarcted that resection of a short length of gut is necessary. Meckel's diverticulum is also discussed in Chap. 19.

*Other Diverticula.* Other diverticula of the small intestine are uncommon and are situated in the duodenum, jejunum or ileum in that



FIG. 58. Multiple diverticula of the jejunum arising mainly at mesenteric border.

order of frequency. *Duodenal Diverticulum.*—About 80 per cent. of all duodenal diverticula are in the *second* part of the duodenum; the remainder occur in the third or fourth parts; true diverticulum of the first part is exceptionally rare. These diverticula may include the whole gut wall or consist of mucosal protrusion through the muscle coats. They are usually in the concavity of the duodenum and more than one is occasionally present.

There are no specific clinical features and the majority of such diverticula are discovered accidentally on barium meal X-ray or, less commonly, at operation. The greatest caution is necessary in attributing symptoms to a diverticulum and, if pain, indigestion or other symptoms occur, it is essential to exclude other causes. Bleeding from such a diverticulum or tumour formation therein are exceedingly rare, but may be indications for

diffuse malignant lymphoma, such as Hodgkin's disease or lymphatic leukaemia. Treatment of a *localised* tumour of this group is by resection when the prognosis is commonly better than for adenocarcinoma. In the diffuse lymphoma group surgery is only justifiable for relief of complications such as intestinal obstruction.

### INFLAMMATION OF THE SMALL GUT

**Crohn's Disease (Regional Ileitis).** Although Crohn's disease occurs in other parts of the gastro-intestinal tract it is so much more common in the small intestine that it is described here. In at least 80 per cent. of cases the terminal ileum is the main or only site of the disease. The colon is involved in about a third of all cases, but nearly always in combination with the terminal ileum and on the right side. Isolated disease in the large gut is most common in the transverse colon. It is uncommon in the rectum and rare in the stomach.

**Ætiology.** The ætiology is obscure, and there seems to be no relationship whatever to tuberculosis. The close similarity between the early lesions and those of Boeck's sarcoid has led some to suppose that they have a common ætiology, but the two diseases are rarely associated in the same patient. Other ætiological factors have been suggested, such as lymphatic obstruction due to primary disease in the mesenteric glands; this would seem improbable; a virus infection, of which there is no positive evidence, has been suggested, and, of course, there are the inevitable psychosomatic theories.

**Pathology.** Since there is rarely any opportunity for studying the initial lesion it is difficult to say how it begins. The established lesions are granulomatous; the bowel wall is rigid, swollen and congested. The neighbouring mesentery is usually œdematous and the mesenteric lymph nodes are pink, fleshy and large. Tiny whitish "tubercles," resembling those of tuberculosis, may be seen on the serous aspect of the gut. Fluid is sometimes present in the peritoneal cavity. There is frequently a fairly clear-cut line of demarcation between diseased and normal bowel; but a characteristic feature in some cases is the presence of multiple lesions with intervening healthy bowel (so-called "skip" areas).

The mucosa usually shows some shallow ulceration, more often on the mesenteric border and axial in disposition. Alternatively, there may be thickening of the mucosa with a coarse cobblestone appearance. Thickening of the submucosa and fibrosis in the muscle layers causes narrowing of the lumen with consequent obstruction and hypertrophy of the bowel wall above. Adhesion to neighbouring viscera may occur, and fistulæ sometimes form between loops of gut and other hollow viscera. External fistulæ, however, are nearly always the result of operation.

**Microscopical examination** shows enlargement of the lymph follicles which are replaced by proliferating endothelial cells. There is infiltration with neutrophils, plasma cells and lymphocytes, and giant cells appear



*serotonin* (5-hydroxytryptamine). The patient occasionally presents with hæmatemesis or melæna and the hæmorrhage may be severe. Attacks of colicky pain, due to obstruction, are often caused by recurrent intussusception, and sometimes acute intestinal obstruction occurs. Symptoms due to serotonin secretion seem to occur only in those patients who have hepatic metastasis. The clinical features include sudden flushing and an unusual type of patchy and constantly changing cyanosis of the skin; bronchial spasm causing asthma-like attacks and respiratory stridor; abdominal pain and frequent watery stools. Some patients develop pulmonary valve stenosis and occasionally there is œdema and ascites. The 5-hydroxytryptamine content of the serum is substantially increased

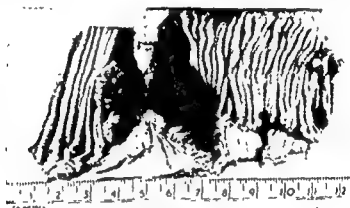


FIG. 59. Primary adenocarcinoma of the small gut.

and can be estimated by paper chromatography. The deamination of serotonin results in increased output of 5-hydroxyindole acetic acid in the urine.

**Treatment.** Localised carcinoid tumour of the small intestine can be treated by resection. Sometimes there is solitary or localised metastasis to the liver which may justify partial hepatectomy. The growth of liver secondary deposits is frequently very slow indeed and patients have been known to survive for many years. In one or two reported cases, the metastatic tumours have apparently remained unchanged in size over a period of several years.

**Adenocarcinoma.** Although adenocarcinoma of the colon is so common it is curious that this type of growth is exceptionally rare in the small intestine (Fig. 59). There is usually a solid whitish tumour with marked narrowing of the lumen of the bowel, and metastasis to the glands and liver is common. Symptoms consist of abdominal pain, and sometimes there is obstruction due to simple occlusion or intussusception. Treatment is by resection, but the prognosis is usually rather poor.

**Sarcoma.** Sarcoma of the small gut, another rare tumour, may occur as a localised lymphosarcoma, leiomyosarcoma, or as a manifestation of

diffuse malignant lymphoma, such as Hodgkin's disease or lymphatic leukaemia. Treatment of a *localised* tumour of this group is by resection when the prognosis is commonly better than for adenocarcinoma. In the diffuse lymphoma group surgery is only justifiable for relief of complications such as intestinal obstruction.

### INFLAMMATION OF THE SMALL GUT

**Crohn's Disease (Regional Ileitis).** Although Crohn's disease occurs in other parts of the gastro-intestinal tract it is so much more common in the small intestine that it is described here. In at least 80 per cent. of cases the terminal ileum is the main or only site of the disease. The colon is involved in about a third of all cases, but nearly always in combination with the terminal ileum and on the right side. Isolated disease in the large gut is most common in the transverse colon. It is uncommon in the rectum and rare in the stomach.

**Ætiology.** The ætiology is obscure, and there seems to be no relationship whatever to tuberculosis. The close similarity between the early lesions and those of Boeck's sarcoid has led some to suppose that they have a common ætiology, but the two diseases are rarely associated in the same patient. Other ætiological factors have been suggested, such as lymphatic obstruction due to primary disease in the mesenteric glands; this would seem improbable; a virus infection, of which there is no positive evidence, has been suggested, and, of course, there are the inevitable psychosomatic theories.

**Pathology.** Since there is rarely any opportunity for studying the initial lesion it is difficult to say how it begins. The established lesions are granulomatous; the bowel wall is rigid, swollen and congested. The neighbouring mesentery is usually œdematous and the mesenteric lymph nodes are pink, fleshy and large. Tiny whitish "tubercles," resembling those of tuberculosis, may be seen on the serous aspect of the gut. Fluid is sometimes present in the peritoneal cavity. There is frequently a fairly clear-cut line of demarcation between diseased and normal bowel; but a characteristic feature in some cases is the presence of multiple lesions with intervening healthy bowel (so-called "skip" areas).

The mucosa usually shows some shallow ulceration, more often on the mesenteric border and axial in disposition. Alternatively, there may be thickening of the mucosa with a coarse cobblestone appearance. Thickening of the submucosa and fibrosis in the muscle layers causes narrowing of the lumen with consequent obstruction and hypertrophy of the bowel wall above. Adhesion to neighbouring viscera may occur, and fistulæ sometimes form between loops of gut and other hollow viscera. External fistulæ, however, are nearly always the result of operation.

**Microscopical examination** shows enlargement of the lymph follicles which are replaced by proliferating endothelial cells. There is infiltration with neutrophils, plasma cells and lymphocytes, and giant cells appear

relatively early. There is no tendency to caseation, and acid-fast bacilli are never found. Similar changes in the lymph glands can produce lymphatic vessel obstruction. Once ulceration of the mucosa occurs, there is secondary infection and infiltration of the tissues.

*Clinical Features.* The incidence of the disease in both sexes is about equal. It is commonest in young adults, but any age group can be afflicted. Patients may present with : (1) Acute abdominal symptoms ; (2) ulcerative enteritis ; (3) chronic small gut obstruction ; (4) persistent fistulæ.

*Acute Cases.* In these cases a diagnosis of appendicitis is often made due to the pain, vomiting, low pyrexia and right iliac fossa signs. However, there is often a history of attacks of diarrhœa in the past.

*Ulcerative Enteritis.* Diarrhœa is a prominent feature and pain is common. Loss of weight, anæmia and low-grade fever also occur. Obvious blood in the stools is unusual, but occult blood is often present. Some of these cases may be difficult to differentiate from ulcerative colitis ; internal fistulæ are liable to form.

*Obstructive Type.* Intermittent abdominal colic, borborygmi, nausea and occasionally vomiting occur. The symptoms are often not particularly severe and may continue for a long time before the diagnosis is made.

*Fistula Formation.* As already stated, internal fistulæ are a feature of the ulcerative type of disease whereas external fistulæ commonly result from operation, such as appendicectomy in acute cases. Anal fistula is a not uncommon complication, and the possibility of Crohn's disease should always be considered in these cases.

Naturally these four groups are not distinct and separate entities, and any sequence or combination of symptoms may occur. On physical examination there is a palpable mass in the abdomen in at least half the chronic cases. Anæmia is rarely severe.

*Diagnosis.* The only certain method of diagnosis before operation is by means of X-ray, and this yields a high percentage of positive results. Some favour barium *enema* X-ray, since, if the terminal ileum fills, the affected zone is less obscured by the rest of the small gut. However, the characteristic lesions in the small gut are not always convincingly demonstrated without a barium follow-through.

The changes seen in Crohn's disease closely resemble those occurring in *hyperplastic tuberculosis*. Although it has lately become fashionable to deny its existence, there is no doubt that *hyperplastic tuberculosis* is a definite entity. However, the relative frequency of tuberculosis and Crohn's disease is a matter for some conjecture. *Ulcerative tuberculosis* of the gut is a somewhat different entity (*vide infra*). *Actinomycosis* is another cause of persistent fistula after an appendicectomy and may be diagnosed by identification of the *Streptothrix actinomyces* in the discharge. *Ulcerative colitis* is sometimes localised in distribution and may involve the terminal ileum. Although such cases are uncommon, it may be

impossible to differentiate them from Crohn's disease either by X-ray or even at operation. *Malignant tumours* of the bowel will usually be differentiated on X-ray, and rarely mistaken at operation. *Talc granuloma* is another possible cause of pathological changes which are sometimes suggestive of Crohn's disease.

*Treatment.* Although spontaneous cure of this condition does occur many cases require operation which may entail:—(1) Resection, either in one or two stages (Fig. 60). (2) Exclusion. At first it was believed that resection was the method of choice, and one would certainly have expected less likelihood of recurrence. A two-stage resection is generally safer for the patient, *but an exclusion operation is safer still, and is, more-*

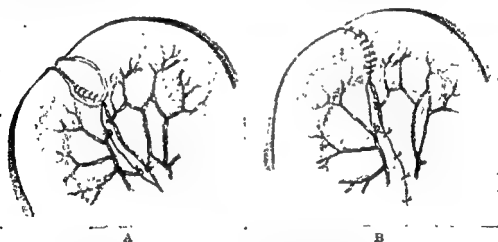


FIG. 60. Resection of small gut. A. All-coats suture partly inserted. B. Sero-muscular sutures inserted and mesentery partly repaired.

over, less often followed by recurrent disease. Nowadays, therefore, the exclusion operations are preferred. The diseased area is by-passed, either by end-to-side or side-to-side ileotransverse colostomy, and the distal end of the ileum is closed. If, however, there is obstruction to the distal ileum, its end is brought up to the skin as a mucous fistula to minimise the possibility of the blind end subsequently bursting.

Follow-up of these cases shows that in about a third of those treated by resection there is either proven recurrence, or persistent symptoms which are suggestive of this. Experience to date suggests that recurrent disease is less common after exclusion, probably of the order of 10 to 15 per cent. of cases.

**Acute Terminal Ileitis.** There seems to be every reason for regarding this as a separate entity from Crohn's disease. Occasionally, at operation for supposed appendicitis, an acute terminal ileitis is found. Up to a foot or more of the ileum is reddened, oedematous and covered with scattered

petechiæ. The mesenteric glands are often enlarged and inflamed and sometimes fluid is present. The appendix and cæcum may also be involved. Appendicectomy in these cases is never followed by fistula formation, and the condition follows an uncomplicated course without recurrent disease. A similar condition is occasionally seen in the jejunum.

**Pseudomembranous Enterocolitis.** A number of unusual types of inflammation in the small gut have been described under this title. These have been variously related to recent gastric or intestinal operations, the use of wide spectrum antibiotics, or infection by staphylococci and *Clostridium welchii*. The colon is involved in some of these cases. The mucosa of the gut is intensely inflamed and extensive patches of yellowish-white slough are usually present. The affected bowel is often dilated, red and thickened. In staphylococcal enteritis masses of organisms may be present in the mucosa. It is important to remember that these cases occurred in pre-antibiotic days, but they certainly appear to be more frequent now. It may be that the use of antibiotics such as *terramycin*, *aureomycin* and *chloromycetin* eliminates the normal gut flora and allows the antibiotic-resistant organisms (e.g. some staphylococci) to flourish.

**Clinical Features.** The symptoms and signs are often very dramatic with profound toxæmia and circulatory collapse. Profuse watery diarrhœa may occur and produce a state comparable to that seen in cases of cholera.

**Treatment.** It is an unfortunate fact that many of these patients die despite vigorous treatment. Replacement of any drastic fluid and electrolyte loss should give the best chance of recovery. Cortisone and ACTH have also been given with benefit. In view of the possibility of clostridial and resistant staphylococcal infections it may be wise to give erythromycin and anti gas gangrene serum. The published results do not permit any precise and definite recommendations for all cases apart from vigorous attention to fluid and electrolyte depletion.

### TUBERCULOSIS

There are two main types of gut tuberculosis, and, in Great Britain, both varieties are much less common nowadays.

**Hyperplastic Tuberculosis.** This variety occurs chiefly in young adults and, as already stated, some go so far as to deny its existence and to attribute the pathology to Crohn's disease (*vide supra*). However, most authorities are agreed that authentic examples of hyperplastic tuberculosis are still seen. Indeed, it may be prudent to suggest that some cases of so-called Crohn's disease are really due to tuberculosis. The infection in hyperplastic tuberculosis probably starts in the lymphoid tissue. Shallow ulcers with undermined edges tend to form; these are usually situated in the lower ileum, and sometimes in the right colon. The wall of the gut becomes oedematous and congested, and small tubercles may be seen in the subserosa. Enlargement of mesenteric lymph

nodes is common, and there may be caseation and abscess formation in the mesentery to which loops of gut may become adherent.

**Ulcerative Tuberculosis.** The other variety of intestinal tuberculosis is usually secondary to open pulmonary disease. Ulceration is commonest in the lower ileum, but may occur throughout the small and large gut. The ulcers tend to *encircle* the bowel, and ensuing fibrosis may result in the formation of *multiple strictures*. Sometimes there is associated *tuberculous peritonitis*. It should be emphasised that ulcerative intestinal tuberculosis is rarely seen to-day, and it is very uncommon even in sanatoria. This greatly decreased incidence is probably due to the more effective control of pulmonary tuberculosis.

**Treatment.** The hyperplastic variety of disease is not usually associated with pulmonary tuberculosis and can therefore be treated on its own merits. There is no evidence that long term antibiotic and chemotherapy has any significant effect on the gut lesions. In most cases, right hemicolectomy is the best treatment. The rare cases of ulcerative intestinal tuberculosis should be treated on general lines with full sanatorium regime; streptomycin, 1 gm. daily, and isoniazid (B.P.), 200 mg. daily. Para-aminosalicylic acid, owing to its tendency to cause nausea, is less well suited to these cases. Operative treatment is only needed for certain complications, such as abscess formation and intestinal obstruction.

### INTESTINAL FISTULÆ

Since the general effects of fistulæ of the small intestine are the most profound and serious it is appropriate to refer to fistulæ of the gut in this chapter. Fistula-in-ano is dealt with in Chap. 10. Fistula of the gut may be external or internal, and can arise at any level in the gastro-intestinal tract, being due to a wide variety of causes. The commonest varieties of internal fistula are gastro-jejuno-colic due to anastomotic ulcer; ileo-ileal or ileocolic fistula due to Crohn's disease or intestinal tuberculosis, and vesicocolic fistula due to diverticulitis coli. External fistulæ of the gut are very much more common than internal ones. This section is concerned with those external fistulæ due to trauma and disease rather than those, such as ileostomy and colostomy, which are deliberately planned by the surgeon.

#### Causes of External Fistulæ.

A. *Congenital*. Patent omphalomesenteric duct—rare.

B. *Acquired*. (1) *Planned*. Gastrostomy, jejunostomy, ileostomy and colostomy.

(2) *Accidental injury*. (a) Abdominal wounds; (b) perforation by foreign body.

(3) *Post-operative*. (a) Burst duodenal stump following gastrectomy; (b) leaking intestinal anastomosis; (c) operative damage to gut or its blood supply.

(4) *Inflammatory*. (a) Appendicitis and diverticulitis; (b) following

strangulation of gut ; (c) chronic inflammation due to Crohn's disease, actinomycosis or tuberculosis.

(5) Neoplastic.

**Effects of Fistulæ.** The effects of a high intestinal fistula are much more serious than those of a low one. In the former, the loss of fluid and electrolytes may be so severe as to threaten life itself within a few days. On the other hand, a fistula of the large bowel causes the patient no more than the local and psychological upset due to its presence. Apart from the general effects the local consequences of a high intestinal fistula are also more serious. Thus, digestion and excoriation of the skin may be extreme with a duodenal fistula but negligible with a colostomy.

**Diagnosis.** In many cases the diagnosis and the cause of a fistula is perfectly obvious. Sometimes, however, it may be difficult to decide whether or not there is a communication with the gut. In such cases the clinician has to decide if, in fact, the patient has a sinus or a fistula. An abdominal sinus may be due to an appendix abscess or pericolic abscess ; it may also be due to Crohn's disease or actinomycosis. The character of the discharge may give some clue to its origin. Small gut discharge is strongly alkaline, and that from the upper part of the small gut contains bile and pancreatic ferments. Sometimes the foul-smelling brownish discharge from a pyogenic abscess may be suggestive of intestinal contents. In cases of doubt, a soluble capsule of gentian violet or carmine may be ingested to see whether the dye appears at the external opening. As an alternative to this, Lipiodol or some other radio-opaque medium may be injected up the track from the external opening. Further information may be obtained from barium follow-through or enema X-rays. Barium will not, however, often pass down a fistula from the gut unless the track is short and broad.

**Treatment.** The treatment of an individual case depends, amongst other things, on the following points about the fistula : (1) The cause ; (2) the presence of associated disease or obstruction ; (3) the level of communication with the gut ; (4) the length of the fistula and nature of its lining. *Uncomplicated cases.*—The majority of fistulæ following operation for appendicitis close spontaneously in a week or two and require no special treatment. Many of them are due to damage to the terminal ileum or cæcum at the time of operation, or their involvement in the wall of an abscess. *Complicated Cases.*—If there is some specific underlying trouble, such as actinomycosis or Crohn's disease, specific treatment will be required. Actinomycosis necessitates suitable antibiotic treatment, and in Crohn's disease it will be necessary to by-pass or resect the affected bowel.

If there is obstruction to the bowel beyond the fistula, the latter will remain open. With incomplete or intermittent obstruction, the fistula may heal but repeatedly break down. In all such cases, relief of the obstruction cures the fistula. The detailed management of intestinal

fistula may be considered under two main headings: (1) General measures; (2) local treatment.

*General Measures.* These include: (1) Correction of fluid and electrolyte losses; (2) relief of avitaminosis, malnutrition and anaemia; (3) measures designed to decrease the intestinal fluid loss.

*Fluid and Electrolyte Loss.* In high gut fistula (e.g. burst duodenal stump), vast quantities of water, sodium and potassium may be lost. Such losses can only be corrected by intravenous therapy. The detailed control of this treatment depends upon accurate fluid balance charts and estimation of the plasma electrolytes daily or more often. So great is the depletion in some cases that the equivalent of 5 litres or more of normal saline solution may be required in twenty-four hours, and the actual amount required can be calculated from the plasma electrolyte values.

*Nutritional Problem.* If fluid loss from a high fistula continues for more than a few days there is serious loss of nourishment as well as fluids and electrolytes. Properly treated, most of these cases can be kept in a good state of nutrition. However, in occasional cases of severe hypoproteinaemia, avitaminosis and anaemia may develop, and each of these requires correction before operation. If adequate oral feeding is impossible with a high intestinal fistula (e.g. duodenal), a jejunostomy can be made. Generally speaking it is not practicable to correct any severe nutritional disturbance by parenteral therapy although the giving of whole blood, protein hydrolysates and vitamin injections may all be of some value.

*Measures designed to decrease intestinal fluid loss.* Gastric or gastro-duodenal suction, and strict limitation or the complete withholding of oral feeding greatly decreases the loss from a high intestinal fistula. Gastric suction removes fluid and electrolytes which must therefore be replaced intravenously. The withholding of oral feeding is only possible for short periods. If a fistula is not showing signs of closing after a few days it is usually impracticable to continue on this regime. Jejunostomy, already referred to above, not only facilitates proper nourishment but also enables the patient to continue for much longer without oral feeds. Furthermore, the fluid from the fistula can often be collected *in toto* and put back into the gut, thus greatly simplifying the problem of electrolyte replacement.

*Local Measures.* Local treatment is of great importance in those high intestinal fistulae which discharge large quantities of fluid and digestive enzymes. The following measures are of value: (1) Suction at the fistula site; (2) protection of the skin; (3) the use of suitable collecting bags; (4) operative closure of the fistula.

*Suction.* It is usually best to use a small catheter with a number of side holes and to connect it to a suitable suction pump (e.g. Roberts' pump). Efficient suction keeps the surrounding skin dry, or at least greatly limits skin damage by intestinal juices.



*Skin Protection.* Aluminium and zinc cream, aluminium paint or one of the barrier creams help to protect the skin. It is essential to start protecting the skin as early as possible and not to wait until the surface has been severely breached by the digestive juices. Some have advocated the application of dried milk powder to the surrounding skin with the object of saving it from damage, but this often causes a good deal of local stinging pain.

*Collecting Bags.* When a catheter cannot be inserted into the fistula, or when discharge occurs despite local suction, it is useful to apply some suitable collecting bag. A disposable polythene ileostomy bag, or one of the more permanent types of ileostomy bag, can be readily adapted for this purpose.

*Closure of Fistula.* This is necessary for those with short, broad and mucous membrane lined tracks. The conditions in such cases are closely akin to those in a planned intestinal stoma. The opening is carefully dissected out, the gut sutured in two layers, and the abdominal wall repaired.

### Summary of Treatment

(1) Simple cases without obstruction. Spontaneous closure the rule, and only local and general care needed.

(2) Simple cases with short mucosal tracks. Formal closure is necessary.

(3) Complicated cases without obstruction. Specific treatment of diseases such as actinomycosis and exclusion or resection of Crohn's disease.

(4) Complicated cases with distal obstruction. Relieve obstruction, and, if necessary, remove diseased bowel (diverticulitis or growth).

## CHAPTER 9

### INTESTINAL OBSTRUCTION

INTESTINAL obstruction may be *mechanical* or *functional* in origin. In mechanical obstruction there may be *simple occlusion* or *strangulation* of gut. Simple occlusion implies obstruction to the lumen of the gut, but in strangulation there is obstruction to the blood vessels as well. Functional obstruction may also be of two types, *paralytic ileus* and *spastic ileus*. Combinations of mechanical and functional obstruction can occur in the same patient, and in mechanical obstruction both simple occlusion and strangulation may be present. These descriptive terms are not, therefore, mutually exclusive. In practice, post-operative paralytic ileus is one of the commonest varieties seen. Finally, a condition closely akin to intestinal obstruction may be produced by occlusion of the mesenteric vessels.

#### CAUSES OF OBSTRUCTION

The causes of intestinal obstruction fall into three main groups, mechanical, functional and vascular.

##### (a) Mechanical Causes

(1) *Outside Gut*. External hernias; internal hernias; congenital bands and inflammatory adhesions; tumours and inflammatory masses.

(2) *Stricture of Gut*. Congenital stenosis; post-operative; post-inflammatory (e.g. tuberculous); stenosing neoplasm.

(3) *Blocked Lumen*. Congenital atresia; gallstone, foreign body, faecal impaction; tumour.

(4) *Intussusception*. Idiopathic or secondary.

(5) *Volvulus*. Of pelvic colon, caecum or small gut.

##### (b) Functional Causes

(1) *Paralytic Ileus*.

(2) *Spastic Ileus*.

##### (c) Vascular Causes

(1) *Mesenteric Arterial Embolism and Thrombosis*.

(2) *Mesenteric Venous Thrombosis*.

The commonest cases of mechanical obstruction are those due to external hernias. The various conditions listed above show much variation in incidence at different ages. Thus, in the new-born infant, congenital stenosis and atresia are commonest; from the third month to about the second year, intussusception; in adolescent and young adult life, inflammatory adhesions; in middle life, hernia predominates, and in later life neoplasm of the large bowel is most often responsible. Strangula-

tion of bowel may occur in any of those cases due to pressure on the outside of the gut, and also in intussusception and volvulus. Primary strangulation occurs in cases of mesenteric arterial embolism and venous thrombosis. In most of these cases there is complete obstruction of the bowel. Strangulation can, however, be associated with incomplete obstruction, as when a Meckel's diverticulum, the appendix, or a marginal portion of the bowel (Richter hernia) is trapped in a hernial orifice.

### EFFECTS OF OBSTRUCTION

**Local Effects.** Below the site of obstruction the bowel tends to empty. Above the site of blockage the gut fills with gas and fluid. The gas is derived mainly from *swallowed air* and partly from *diffusion* from the blood and *bacterial fermentation*. The relative importance of these factors varies with the circumstances of obstruction. The *fluid* in the bowel consists of bile, pancreatic and other digestive juices as well as exudate. There is rapid bacterial growth in the stagnant fluid which becomes brown and foul-smelling. Such fluid, when vomited, is sometimes described as "*feculent*" but it is in no sense *faecal*. As the pressure in the gut rises the mucosa becomes congested and oedematous, absorption ceases, more fluid is poured out, and the intraluminal pressure rises still further. A loop of gut blocked at both ends, or an obstructed diverticulum, is referred to as a *closed-loop obstruction*.

The effects of strangulation are rather different from those of simple occlusion of gut. In cases due to mechanical factors the veins obstruct first and the arteries later. There is considerable exudation of blood into the lumen of the bowel and peritoneal cavity with quite rapid decrease in blood volume. Bacterial growth is rapid in the lumen and wall of the ischaemic haemorrhagic bowel with consequent toxæmia and peritonitis.

**General Effects.** The early general effects of obstruction are due to loss of water and electrolytes into the lumen of the bowel. The amount of fluid lost depends principally on the *level* of the obstruction. In *high small gut obstruction* the loss is rapid and severe, but in *low small gut obstruction* it is slower and less serious. The fluid and electrolytes thus lost from the blood are made good from the tissue spaces, and the kidneys attempt to conserve the available water and electrolytes by reduced urinary output. The compensatory mechanism of the body is so effective that there may be little change in the blood chemistry until a relatively advanced stage of obstruction. Furthermore, the fall in sodium and chloride level of the plasma may be masked by the accompanying dehydration which also causes haemoconcentration with raised haemoglobin and haematocrit readings. If, as is usually the case, the loss of chloride is disproportionately greater than that of sodium, then the spare sodium ions combine with carbonic acid with consequent rise in the alkali reserve. The non-protein nitrogen may also rise due to increased protein breakdown and impaired renal function.

The general effects of strangulation of gut are partly due to the above factors, and partly to blood loss and toxæmia. Death in intestinal obstruction is due to a combination of fluid and electrolyte loss, hæmo-concentration, toxæmia and peritonitis; the last three factors are especially important in cases of strangulation.

### CLINICAL FEATURES

Since certain points are common to all types of intestinal obstruction it is best to discuss these first and to refer to individual groups later. *Pain, vomiting, distension and absolute constipation* are the four cardinal features. *Pain* is usually, though not always, present in mechanical obstruction and is usually colicky in character. It is not a feature of paralytic ileus. In intestinal strangulation, swelling and traction on the mesentery sometimes causes pain in the back. *Vomiting* is early and repeated in high small gut obstruction, later with low small gut and commonly absent with large gut obstruction. The amount and character of the vomitus varies with the level of obstruction. *Distension* is usually most obvious with left colon obstruction, and less obvious the higher the blockage. It is chiefly peripheral with colonic, but mainly central with small gut obstruction. Visible peristalsis is sometimes seen in small gut obstruction, but this depends more upon the thinness of the abdominal wall than the degree of obstruction. *Absolute constipation* is characteristic of complete obstruction; but, if an enema is given, some fæces and even a little flatus may be returned from below the site of obstruction. When essential to the diagnosis a second enema is more informative.

In examining a case of suspected intestinal obstruction, the hernial orifices should be inspected first. The temperature is usually unhelpful and the pulse rate is only raised later. *Tenderness and rigidity* are unusual in simple obstruction and usually occur in relationship to a strangulated loop of bowel. Sometimes the latter can be felt as an abdominal or pelvic swelling. Indeed, these three features may be the only means of distinguishing strangulation from simple occlusion. In strangulation, delay in operation is dangerous; but, in simple occlusion, non-operative treatment may be desirable and it is, therefore, of importance to differentiate these conditions. The chief diagnostic difficulties occur in cases of obstruction due to bands or adhesions in which either strangulation or simple occlusion may occur. *Auscultation* of the abdomen reveals active, high-pitched and sometimes tinkling bowel sounds in mechanical or spastic ileus. In paralytic ileus the abdomen is generally silent.

*Plain X-ray films* of the abdomen show gas-distended bowel and multiple fluid levels. A film in the erect position is generally of most value. Small gut loops are seen in the central abdomen, whereas the colon is more peripheral. However, the differentiation between small and large bowel can sometimes be quite difficult. Thus the valvulæ conniventes of distended jejunum must be differentiated from large bowel

haustrations, and a grossly distended small bowel loop can look just like colon. Although volvulus of the pelvic colon can produce central abdominal distension like a small gut obstruction, the X-ray appearances are usually characteristic (Fig. 63). A plain film of the abdomen is often of value in demonstrating the likely site of large gut obstruction.

### INDIVIDUAL CAUSES OF INTESTINAL OBSTRUCTION

The general features already referred to are evident to a variable degree in different cases. Certain features of individual cases need further discussion.

**Hernias.** Time and again an external hernia, especially a femoral one in a fat patient, is overlooked through incomplete examination and

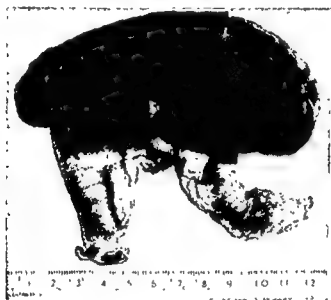


FIG. 61. Resected specimen of small gut strangulated by a band.

failure to pull the bed clothes or the trousers down sufficiently. Apart from the possibility of missing such a hernia, obstruction is sometimes wrongly attributed to a coincidental hernia. Thus, an external hernia may become tense and difficult to reduce as a result of intra-abdominal obstruction, but the true diagnosis is unlikely to be missed by a careful observer. Internal hernias are rare, and it is generally impossible to make an exact diagnosis prior to operation.

**Bands and Adhesions.** Congenital bands, such as that between the apex of a Meckel's diverticulum and the umbilicus, are uncommon causes of obstruction (Fig. 57). Adhesions are usually due to a previous operation, most often for appendicitis or peritonitis. The gut may become adherent to the back of the incision, the stump of the appendix, or the site of a former abscess or inflamed glands. Usually there is simple occlusion due to angulation of the gut, but a loop of gut is sometimes

snared and strangulated by an adhesion (Fig. 61). When obstruction is due to adhesions the rapid relief of pain on aspirating the gut may serve to differentiate simple occlusion from strangulation.

**Stricture of Gut.** Inflammatory stricture of the small gut due to tuberculosis is nothing like so common as it was. Malignant stricture of the small gut is rare, and such cases are more often due to *carcinoid tumour* than *adenocarcinoma*. Malignant stricture of the colon is more common on the left side.

**Volvulus.** This most commonly affects the *pelvic colon* which is often unusually long in these cases. Though rather infrequent in Great Britain, this is one of the commonest causes of intestinal obstruction in Eastern Europe. The onset is often sudden and dramatic, with severe pain and rapidly increasing distension. Swelling of the abdomen is often rather asymmetrical. Sometimes there is a history of previous mild attacks of abdominal pain and distension. *Volvulus of the caecum* is very much less common, occurring only in those who have a well-developed ascending mesocolon. The symptoms are rather like those of a low small gut obstruction with a grossly distended caecum. *Volvulus of the small gut* occurs in a rare neonatal form (Chap. 19). It occasionally occurs later in life when a loop of small gut, anchored at its apex by an adhesion, twirls like a skipping-rope.

**Intussusception.** Idiopathic intussusception occurs mainly in infants and young children (Chap. 22), but is occasionally seen in adults too. Intussusception in adults is usually secondary to carcinoma, submucous lipoma or some other tumour of the gut which initiates the condition (Fig. 62). In some of these cases, there are recurrent attacks of transient and incomplete obstruction and the diagnosis can be difficult to establish.

**Intraluminal Obstruction.** Occlusion by growth needs no further description here. Gall stone ileus and obstruction by such objects as ill-masticated fruit are both uncommon. The former tends to occur in elderly women in whom a stone ulcerates into the duodenum from Hartmann's pouch or the fundus of the gall-bladder. When the stone is too large to pass through the gut it is generally held up about 3 feet from the ileocaecal valve, i.e. in the narrowest part of the ileum. Despite the fairly simple nature of the obstruction the mortality is quite high, for the patients are often fat, elderly and generally poor-risk subjects. Blockage of small gut by an ill-masticated mass of unripe fruit is occasionally seen in old patients who use their false teeth for social occasions rather than eating and sometimes after partial gastrectomy.

### DIFFERENTIAL DIAGNOSIS

*Appendicular, intestinal, biliary and renal colic* may be suggestive of intestinal obstruction in some cases. When an inflamed appendix is adherent to the terminal ileum the symptoms can be most suggestive of obstruction, and true obstruction does sometimes occur. *Intestinal colic*

due to *gastro-enteritis*, *lead poisoning* and *acute porphyria* may be confusing, but further observation will reveal no supporting signs of obstruction, and in none of these conditions is there absolute constipation. Once diarrhoea supervenes in a case of enteritis there will be no further doubt. *Lead colic* can sometimes trap the unwary: the diagnosis may be suggested by a blue line on the gums but is only proven by basophil stippling of red cells and, if necessary, by identifying abnormal amounts of lead in the urine.

*Henoch's purpura* may closely simulate intussusception.

*Peritonitis* can sometimes mimic obstruction quite closely, especially in



FIG. 62. Secondary intussusception due to an angiodysplasia of the ileum. Outside of resected specimen showing ileo-ileal (a), and ileocecal invagination (b). (Courtesy of Alan Small.)

post-operative cases. There is an element of mechanical and paralytic ileus in many cases of peritonitis which may make it difficult to exclude a primarily obstructive condition. Plain X-ray films of the abdomen are often of great value, but such cases require careful assessment by an experienced surgeon. *Acute pancreatitis* may present with symptoms suggestive of high small gut obstruction though doubt about the diagnosis is unlikely to persist for long. *Faecal impaction*, occasionally deceptive in the old, is readily exposed by rectal examination.

It remains to mention those cases in which *meteorism* and *vomiting* accompany some extra-peritoneal disease. Most notable of these conditions are *pneumonia* and *uræmia*. In the former cases, clinical and radio-

logical evidence of consolidation is sometimes slight. Marked abdominal distension in an elderly bronchitic subject may land him in a surgical ward and even the enema test may be unhelpful. Fortunately, the wheezing dyspnoea of some of these patients discourages surgery for just long enough to enable the true diagnosis to be established. The surgeon should be put on his guard by an X-ray which shows gross gas distension without fluid levels. Nevertheless, especially in prodigious air-swallowers, true obstruction sometimes presents with very little fluid on X-ray.

### TREATMENT OF INTESTINAL OBSTRUCTION

The same basic principles apply to all cases, but the details of treatment may vary with the circumstances of the individual case. Treatment includes : (1) Efficient gastro-intestinal suction ; (2) carefully prescribed intravenous fluids ; (3) maintenance of accurate fluid-balance chart ; (4) properly timed surgical relief. Many cases of simple obstruction due to adhesions are relieved without operation and, providing strangulation can be confidently excluded, this is the treatment of choice for such cases. On the other hand, a patient in good general condition with an early strangulated hernia simply requires operative relief and little adjuvant therapy. In between these two extremes are those patients with gut strangulation who need prompt resuscitation, early operation, and continued suction and intravenous therapy afterwards. Non-operative treatment is reserved for all cases of paralytic and spastic ileus, many cases with post-operative adhesions (especially if multiple and diffuse) and for some of those with simple occlusion.

**Gastro-intestinal Suction.** A Ryle's tube is unsuitable for most of these cases since its lumen is narrow and the tiny holes in the distal end tend to block with debris. A tube of adequate calibre is a No. 10 or 11 (F.) Jacques œsophageal catheter which is passed into the stomach. Simple gastric suction is certainly adequate in most cases though some prefer to use special intestinal suction tubes (Miller Abbott or Cantor). Unfortunately, it is in just those cases in which intestinal suction seems most desirable that it is most difficult to manipulate the tube beyond the pylorus. A good deal of skill is necessary in using some of these special tubes ; X-ray control may be needed, and the manipulation can be disturbing to the patient. For these reasons, and because simpler methods seem just as satisfactory, most surgeons rely on gastric suction which may be either continuous or intermittent.

**Intravenous Therapy.** The fluid and electrolyte requirements can be fairly accurately assessed from the time the patient enters hospital. Any deficit acquired prior to admission can only be guessed at, and even detailed blood examination is of limited value. The clinically dehydrated patient may require 5 litres in the first twenty-four hours, but only half of this need be normal saline. Once the initial deficit has been made up, the basic fluid requirement is about 3 litres of fluid a day and 5 gm. NaCl.



due to *gastro-enteritis*, *lead poisoning* and *acute porphyria* may be confusing, but further observation will reveal no supporting signs of obstruction, and in none of these conditions is there absolute constipation. Once diarrhoea supervenes in a case of enteritis there will be no further doubt. *Lead colic* can sometimes trap the unwary: the diagnosis may be suggested by a blue line on the gums but is only proven by basophil stippling of red cells and, if necessary, by identifying abnormal amounts of lead in the urine.

*Henock's purpura* may closely simulate intussusception.

*Peritonitis* can sometimes mimic obstruction quite closely, especially in



FIG. 62. Secondary intussusception due to an angioma of the ileum. Outside of resected specimen showing ileo-ileal (a), and ileocaecal invagination (b). (Courtesy of Alan Small.)

post-operative cases. There is an element of mechanical and paralytic ileus in many cases of peritonitis which may make it difficult to exclude a primarily obstructive condition. Plain X-ray films of the abdomen are often of great value, but such cases require careful assessment by an experienced surgeon. *Acute pancreatitis* may present with symptoms suggestive of high small gut obstruction though doubt about the diagnosis is unlikely to persist for long. *Faecal impaction*, occasionally deceptive in the old, is readily exposed by rectal examination.

It remains to mention those cases in which *meteorism* and *vomiting* accompany some extra-peritoneal disease. Most notable of these conditions are *pneumonia* and *uraemia*. In the former cases, clinical and radio-

colour, loss of peritoneal sheen, non-pulsation of mesenteric arteries or thrombosis in the corresponding veins. The most important part of the gut to inspect is at the site of constriction by a band or the neck of a hernial sac. Little patches of non-viable small bowel, such as at the margin of a Richter's strangulation, are inverted with a few Lembert sutures so long as this does not unduly narrow the lumen of the bowel. Larger segments of non-viable gut, or doubtful areas associated with damage to the mesentery are resected. With a dubious patch of bowel, rather than undertake a possibly unnecessary resection in a poor-risk subject, it may be wise to leave the affected loop close to the parietal peritoneum with a large drain down to it. If a fistula does supervene this will usually heal spontaneously or be readily dealt with.

*In the colon* most cases of simple occlusion are due to growth or diverticulitis, and can be relieved by a suitably placed colostomy. Obstruction to the caecum or ascending colon is relieved by ileo-colostomy; the terminal ileum is anastomosed to the transverse colon. *Non-viable colon* is best exteriorised, as the risks of intraperitoneal resection are usually prohibitive in such cases. The further management of obstruction due to carcinoma of the colon is discussed in Chap. 10.

### FUNCTIONAL OBSTRUCTION

**Paralytic Ileus.** Paralysis of the intestinal muscle is relatively common and may occur in a variety of circumstances. The exact cause is unknown although various aetiological factors are suggested. *Handling* of the gut in the course of abdominal operations usually results in a period of diminished peristalsis. Complete ileus is less common, and in the majority of cases clears up quite rapidly. *Reflex paralytic ileus* may be due to fractures of the spine or pelvis, retroperitoneal haemorrhage, prostatectomy, childbirth, and sometimes it occurs in association with chest injuries and pneumonia. *Peritonitis* is regularly accompanied by paralytic ileus. The ileus may be regarded as a protective mechanism to rest the bowel and limit spread of infection. It is further believed that some cases of paralytic ileus are related to use of the relaxant drugs and depletion of the plasma potassium.

*Prevention.* In abdominal operations, it is generally felt that a well-prepared patient is less liable to ileus. Ritual purgation is rigorously eschewed, as this unnecessary flogging of the bowel certainly produced its share of ileus in the past. The avoidance of trauma and undue handling of the bowel is also important. An indwelling suction tube which keeps the gut empty may be of especial importance in habitual air-swallowers.\* The proper use of rectal and intravenous fluid therapy after operation helps to rest the bowel, but a warning must be given against over-

\* If the end of the suction tube is left open in a piece of gauze air can escape without aspiration.

The daily volume of fluid aspirated is replaced volume for volume, half by normal and half by fifth normal saline. Thus, in the event of the aspirated fluid measuring 2,000 ml. in a day, the patient would be given 1,000 ml. N. saline and 1,000 ml. N/5 saline with 4 per cent. glucose, in addition to his basic 3,000 ml. N/5 saline with 4 per cent. glucose.\* *Potassium* deficiency may develop after a few days on intravenous fluids due to low intake and potassium diuresis. Therefore, if the patient has not returned to normal feeding after forty-eight hours, it is wise to give KCl 11 gm. daily (either by mouth or not exceeding 3 gm./litre I.V.).

**Fluid Balance Chart.** The maintenance of a clear and accurate fluid balance chart is fundamental to proper treatment. The daily volume of urine, its specific gravity, and sometimes its chloride content give valuable clues to treatment. The urine output should be maintained at not less than 1,000 ml. daily, and a low urinary output may indicate excessive loss from sweating or suction. Generally speaking the best estimate of daily fluid and electrolyte requirements is provided by the clinical condition of the patient and his balance chart. Only with prolonged intravenous therapy is it necessary to resort to daily or more frequent estimation of the blood chemistry. The balance chart is completed once or twice daily when the fluid and electrolyte requirements for the ensuing period are calculated.

**Operative Treatment of Obstruction.** On no account should an anæsthetic be given until a suction tube is passed and the gastric contents aspirated. In cases of strangulated hernia the incision is made appropriate to the particular hernia. In all other cases a paramedian incision is most suitable. When obstruction is due to adhesions from a previous operation the site is usually readily identified. In the absence of any such clues to the cause the cæcum is first identified. If the cæcum is ballooned the obstruction is in the colon and the latter is traced to the cause. If the cæcum is normal or collapsed the ileum is traced upward to the site of obstruction. In many cases of small gut obstruction a collapsed loop is apparent immediately on opening the abdomen and there is then no need to inspect the cæcum. In a case of obstructed umbilical hernia, omentum and transverse colon are in the upper part of the sac, but the obstruction commonly involves small gut which has recently entered the sac below the omentum. In most cases of obstruction due to an external hernia, as well as in some of those due to bands and adhesions, there is some degree of strangulation. As soon as the obstructed bowel is relieved the surgeon must decide whether resection is necessary. After applying a warm, moist pack to the affected bowel, the normal colour and lustre is usually restored in a few minutes.

*Non-viability* of bowel is indicated by a persistent blue-black or greenish

\* One standard transfusion bottle contains 540 ml. fluid. One bottle of N. saline contains approximately 5 gm. NaCl, and one bottle of 4 per cent. glucose and N/5 saline contains approximately 1 gm. NaCl.

" milk = the gut contents down through the affected area. It is probably better then to make no further attempts to relieve the condition or paralytic ileus may ensue. Following operation the appropriate drip and suction is continued.

**Vascular Obstruction.** Mesenteric embolism and thrombosis are dealt with in Chap. 7.

enthusiastic "drip and suction" when normal feeding could be resumed. Despite these observations and the rational management of abdominal cases, paralytic ileus still occurs and can be a most tiresome complication.

*Clinical Features.* Abdominal distension and vomiting occurring within a day or two of operation or injury is commonly due to paralytic ileus. Sometimes the onset of nausea precedes vomiting and distension. This and a rising pulse rate forewarns the observant clinician. The condition itself is generally relatively painless, but when distension is marked the patient may be very uncomfortable. Constipation is absolute and bowel sounds are absent. Plain X-ray of the abdomen in the erect position shows gas distended gut and fluid levels.

*Treatment.* The first three principles of treatment differ in no way from those referred to in the management of mechanical obstruction. Suction, intravenous therapy and careful fluid balance charting are essential. Rest for the gut is ensured by these measures; by withholding nourishment by mouth; and by the regular injection of Omnopon, 10 mg. ( $\frac{1}{4}$  grain) to 20 mg. ( $\frac{1}{2}$  grain) every four to six hours, or morphine, 10 mg. ( $\frac{1}{4}$  grain) to 16 mg. ( $\frac{1}{2}$  grain). Treatment is continued on these lines until bowel sounds are heard or flatus is passed per rectum. In case of doubt the suction tube can be clipped off and the effects of oral feeding observed for a spell. Occasionally, on starting normal feeding, there is a return of symptoms and the full regime must be implemented once more. Some have advocated the routine administration of potassium, but potassium depletion is probably important in only a minority of cases. In cases of resistant paralytic ileus, it seems that intramuscular injection of pantothenic acid (50 mg. four-hourly for two or three doses) is sometimes of value.

*Spastic Ileus.* A less common development, the very antithesis of the last condition, is spastic ileus. Prolonged segmental contraction may affect a short or long segment of gut. This is seen in lead poisoning as well as a variety of other circumstances. Thus, it has been described with extensive malignant disease of the retroperitoneal glands when it is presumed to be due to involvement of the sympathetic nerves in the coeliac plexus. Spastic ileus sometimes occurs following the administration of prostigmine as an antidote to the relaxant drugs.

The clinical features are those of a mechanical obstruction, and active peristalsis is usually heard. The spastic segment of gut may relax under the influence of an anaesthetic. Sometimes its persistence in the colon has led to a diagnosis of carcinoma, and a proximal colostomy has been done only to find that the "growth" has disappeared on subsequent laparotomy.

Treatment is by suction and intravenous therapy. It is generally felt that pethidine (50 to 100 mg., four- to six-hourly) is safer than morphine which may aggravate the condition. When the abdomen is opened under the impression that mechanical obstruction is present, if the spasm does not relax under the influence of the anaesthetic, an attempt is made to

is severely congested, strangulated or near-gangrenous, it may be exteriorised by the Paul-Mikulicz procedure (described later in this chapter), but if the lower limit of the damaged bowel cannot be brought to the surface a Hartmann's type operation is best. In the latter cases

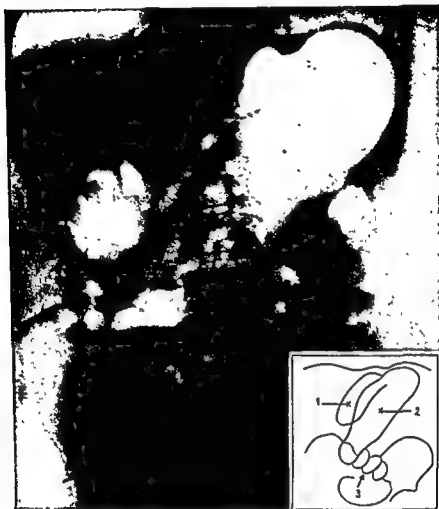


FIG. 63. Volvulus of sigmoid colon. Plain X-ray (erect) film of abdomen. Inset shows how the two limbs of the affected loop are clearly seen (1 and 2). Not visible on the film are three complete twists in the bowel based on its mesentery (3). (Courtesy of Dr. L. S. Carstairs.)

continuity of the bowel can be restored by end-to-end anastomosis at a later date, although a restorative resection of this kind would be quite unjustified as an emergency procedure.

**Volvulus of the Cæcum.** This is less common than volvulus of the sigmoid, and can only occur in those patients who have a mobile cæcum with an ascending mesocolon. Volvulus of the cæcum sometimes gives rise to recurrent attacks of abdominal pain associated with localised

## CHAPTER 10

### THE COLON, RECTUM AND ANAL CANAL

CONGENITAL anomalies of the colon and rectum are discussed in Chaps. 19 and 20. This chapter is concerned only with acquired conditions.

**Volvulus of the Sigmoid Colon.** This is uncommon in Great Britain, but it constitutes one of the common emergencies in eastern Europe and Russia. The main factors predisposing to its development are a long sigmoid loop with a relatively narrow base to the mesentery and a loaded bowel. Constipation may be a further factor, but in this connection it is difficult to differentiate cause and effect.

*Clinical Features.* Volvulus is commoner in men, and the majority of cases occur after middle age. Usually there is sudden onset of severe colicky pain, rapid distension of the abdomen and absolute constipation. Sometimes there is a history of less severe attacks of pain and distension in the past. There may be reflex vomiting at the onset, but it is then unusual excepting in advanced cases. Occasionally a little blood and mucus is passed per rectum. The abdominal distension may be so severe that the diaphragm is pushed up and respiration seriously embarrassed. Marked shock and collapse occur in those with strangulation of gut.

The sigmoid loop is rotated through anything from 180 degrees to several complete twists, and there is a closed loop obstruction with or without strangulation. The affected loop rapidly fills with gas, and the colon proximal to it is obstructed. However, the latter obstruction is usually overshadowed by the effects of the volvulus, and rarely is there much distension of the proximal colon by the time of operation. Plain X-ray films of the abdomen usually show a grossly distended loop of bowel (Fig. 63).

*Treatment.* When the patient is seen comparatively early and there is only a relatively minor degree of volvulus, it is sometimes possible to pass a soft rubber tube per rectum into the affected loop. This is best attempted through a sigmoidoscope, but it is only likely to succeed in a small proportion of cases. If a tube cannot be passed per rectum then operative relief is necessary. Preparation follows routine lines for dealing with intestinal obstruction and any circulatory collapse should be rapidly corrected by transfusion. In the simpler cases the loop of bowel can be untwisted. It is often helpful if an assistant passes a soft rubber tube per rectum to deflate the bowel directly it has been restored to normal position. The loop can then be fixed with a few sutures to the parietal peritoneum. However, in most of these cases it is wise to advise subsequent resection of the sigmoid as a definitive operation. If the bowel

usually larger, solitary and include all coats of the bowel. They are probably developmental in origin, although increased pressure within the bowel enlarges them. The multiple false diverticula are acquired, probably due to a combination of increased intraluminal pressure together with fat deposition and weakening of the bowel wall. The affected subjects are often obese, sometimes constipated, and they may suffer from biliary disease and other afflictions which are more common in the obese.

If the neck of a diverticulum is relatively broad it is likely that the bowel contents will pass freely in and out. If the neck is narrow, then faeces may accumulate and lead to ulceration and infection of the mucosa, converting a state of diverticulosis into one of *diverticulitis*.

The early stages of diverticulosis coli are relatively symptomless, but they can often be detected on X-ray. Initially there is spasm of the bowel and a ragged outline; later a spiky irregularity, and finally full-blown diverticula appear (Fig. 64). By this stage the condition can be more easily recognised at operation, but the diverticula are often concealed in fat. Muscle fibres are only present around the neck of a fully-established diverticulum. The mucosa may be ulcerated and, when inflammation occurs, the opening into the bowel and external evidence of the lesion may be concealed by oedema. This diverticulitis and peridiverticulitis may lead to considerable thickening of the bowel wall with formation of a mass which can be difficult to distinguish from carcinoma. In long-standing cases, the colon becomes infiltrated with fibro-fatty tissue.

Various complications may arise. *Perforation* can lead to a localised abscess and uncommonly to peritonitis (Fig. 65). *External fistula* may follow spontaneous or operative drainage of an abscess. *Internal fistulae* may occur and they tend to form in the pelvis between the colon and the bladder, the vagina or a loop of small gut. In the more chronic cases obstruction may occur, either due to the colonic mass or adhesion to neighbouring structures. In the latter cases the small gut is more often obstructed than the colon itself.

**Clinical Features.** Low abdominal pain is common and is usually left-sided, consisting of a dull ache or, less often, colic. Many patients have reflex indigestion and a feeling of fullness, indeed a combination of these two symptoms with low abdominal pain is very suggestive of diverticulitis. Loss of appetite, nausea and vomiting occur in a proportion of patients, and this sometimes leads to investigation of the stomach in the first instance. Some patients have three or four bowel actions a day whereas others suffer from constipation. Tenesmus, and the passage of mucus and blood may also occur. Infrequently there is quite heavy bleeding from the rectum. Urinary symptoms and pyrexial attacks occur especially in those who have an inflamed segment of bowel adjacent to the bladder. Whereas some of the milder symptoms referred to above may occur with diverticulosis alone, in most cases there is some degree of diverticulitis before symptoms occur. In the more severe varieties the



distension and tenderness in the right iliac fossa. In more severe cases, there may be some obstruction to the terminal ileum. *Treatment* is by operative fixation of the cæcum, and if the latter is much distended it is wise to do a temporary cæcostomy. Occasionally the condition recurs, and then a right hemicolectomy may be necessary.

### DIVERTICULOSIS OF THE COLON

Diverticula of the colon are found in at least 5 per cent. of people over the age of forty. They are most commonly multiple and tend to occur in



FIG. 64. Diverticulosis coli in malrotated (left-sided) colon.

the left half of the bowel, especially the pelvic colon. Diverticula are often stated to be rare in the rectum, but they are, in fact, not uncommon there.

**Pathology.** Diverticula consist of herniations of mucosa through the overlying muscle of the bowel and they appear at the site of penetration of the small blood vessels close to the lateral taeniæ. They are usually about 5 to 10 mm. in diameter. As all coats of the bowel are not found in the wall, such multiple diverticula are frequently referred to as *false diverticula*. In the ascending colon and cæcum, however, a less common variety of *true diverticulum* is sometimes found. These are

with diverticulosis coli never require surgical treatment. Operation is only needed in those with complications and more severe symptoms. Mild symptoms are usually relieved or held in check by such simple measures as a low-residue diet and attention to the bowels. *Acute diverticulitis* usually settles down on conservative treatment, but if an abscess is present it is usually better to drain it. Not only is the patient more comfortable after drainage of an abscess, but the trouble settles down quicker and there is less chance of such complications as peritonitis and subsequent internal fistula or obstruction. Since many of these abscesses form in the left iliac fossa, they can be drained extraperitoneally. *Acute perforation of the colon* should be treated by closure of the perforation with omentum and adequate drainage. *Antibiotic therapy* is indicated for those with peritonitis and also for those with rigors and marked constitutional disturbance; oxytetracycline is generally the most suitable agent.

Intestinal obstruction may either occur in the colon at the site of the disease or else in an adherent loop of small gut. It is not uncommon for a combination of obstructive and inflammatory symptoms to be present. Colonic obstruction is treated by a preliminary colostomy which is usually made in the right side of the transverse colon, and the diseased bowel is resected six weeks or more later. Sometimes, owing to the slower development or intermittent character of the symptoms, it is possible to prepare the patient for one-stage resection and anastomosis. Exteriorisation of the affected bowel by Paul-Mikulicz technique is rarely practicable owing to shortening and fat infiltration of the mesentery.

*Internal fistula* may necessitate difficult surgery. For most of these cases, in which the patient is fat and in which there is marked inflammation and adhesion formation, it is wise to do a preliminary colostomy. If the patient is slimmed during the ensuing months, the second operation will generally be made much easier. Sometimes resection of the diseased bowel and closure of the fistula can be effected in one stage. *External fistula* usually follows a pericolic abscess and commonly does not heal unless the diseased bowel is resected.

It may well be asked what happens to those patients whose disease necessitates resection of the bowel. It is an interesting observation that, despite the frequent presence of diverticula elsewhere in the colon, rarely does the patient develop further severe disease which requires surgical treatment. The late results of surgery are therefore good, but the diseased and fatty condition of the colon often renders the operation more difficult than that for carcinoma.

### ULCERATIVE COLITIS

The cause of ulcerative colitis is unknown and the condition is usually a chronic one with remissions and relapses over a period of years. There is no evidence that any specific infective agent is responsible for this

clinical features are more marked. Thus, there may be acute symptoms akin to those of appendicitis, but on the left side, or there may be the symptoms of such complications as abscess, perforation, peritonitis, fistula and obstruction.

Physical signs in the uncomplicated case are often conspicuous by their absence. Tenderness in the left iliac fossa is the most common sign. In complicated cases a bowel mass or abscess may be palpable. *Sigmoidoscopy*



FIG. 65. Diverticulitis coli. Middle-aged man presenting with obstructive symptoms and gas-filled abscess adjacent to diseased bowel in left iliac fossa. (Courtesy of Dr. L. S. Carstairs.)

sometimes reveals œdema and reddening of the mucosa, or narrowing, rigidity and fixation of the bowel higher up. *Barium enema X-ray* is the most useful method of investigation. A mixture of 1 per cent. tannic acid with barium is valuable in demonstrating mucosal pattern and the contractility of the bowel, but it may cause some colic. "Saw edge" outline, spasm and shortening of the bowel may be observed in addition to full-blown diverticula. In chronic cases a length of narrowed bowel is sometimes seen.

**Treatment.** It will be appreciated that the great majority of patients

**Clinical Features.** More than half the patients with ulcerative colitis develop it between twenty and forty years of age, and even children are occasionally afflicted. Rarely does the disease appear for the first time over the age of fifty. At most sites women are afflicted rather more frequently than men. The most characteristic features are *diarrhœa*, accompanied by the passage of *mucus, pus* and *blood* in the stools. The rapidity of onset and severity of these symptoms varies a good deal from case to case. Sometimes there is abdominal discomfort and *colic*. *Pyrexia* is common and is indeed one of the most constant features of active disease. *Anæmia* is almost invariable and *loss of weight* may be marked. Cases may be divided into several main groups: (1) The acute fulminating type which may go on to rapid death; (2) the chronic relapsing type with repeated attacks over a long period of time; (3) the chronic persistent type in which, despite minor fluctuation in symptoms, there is never a complete remission. Patients in any of these groups may develop complications such as abscesses and fistulæ, infective arthritis, pyoderma, perforation or hæmorrhage.

On examination, there may be pyrexia, rapid pulse, and evidence of anæmia and weight loss. Abdominal signs are usually limited to tenderness over the colon which may be palpable. *Sigmoidoscopy* is an essential step in diagnosis. It may reveal changes varying from slight reddening and granularity of the mucosa to severe thickening with ragged ulcers and pseudo-polypi. The latter are sometimes easily palpable on digital examination of the rectum. *Barium enema* X-ray may show varying degrees of change. At first there is irritability of the bowel but ulceration is most difficult to detect. Later the bowel outline becomes smooth and the colon tends to be stiff, narrow and short (Fig. 67). Obvious pseudo-polypi and strictures may be seen.

**Differential Diagnosis.** Mild and early cases have to be differentiated from *mucus colitis* and other causes of *simple diarrhœa*. Only by continued observation of the case is certain diagnosis possible, and sometimes the diagnosis of ulcerative colitis is not established for some time. The more advanced and chronic cases are generally more easily diagnosed, but have to be distinguished from *chronic amœbic infection*. Localised varieties of ulcerative colitis must be distinguished from *carcinoma*, *diverticulitis* and *regional colitis* (Crohn's disease in the colon). True *polyposis coli* is distinguished from the pseudo-polyposis of advanced colitis by the normal mucosa between the polypi.

**Treatment.** In the first instance, the treatment of all cases of ulcerative colitis is strictly medical; the details are not discussed here. The essentials of such treatment are a high protein and low residue diet, correction of anæmia and relief of distressing bowel symptoms.

The indications for surgery are: (1) Chronic disease, with repeated relapse and invalidism, or with persistent symptoms despite intensive medical treatment; (2) complications, such as abscess, fistulæ, pyoderma, perforation, hæmorrhage and carcinoma.

disease. Some consider psychogenic factors of importance, but the remarkable improvement in personality shown by some patients after operation suggests that psychological upsets may often be an effect rather than a cause of the disease.

**Pathology.** In the majority of patients, the disease starts in the left side of the colon and commonly in the rectum and sigmoid area. The inflammation may stop quite abruptly in the descending or transverse colon, but in the more severe cases the whole colon is often involved. Infrequently the disease is confined to the right half of the colon and may extend into the terminal ileum. Occasionally there are two separate diseased areas with normal bowel between, and at operation the appearances are very suggestive of the "skip" areas of Crohn's disease.



FIG. 66. Ulcerative colitis with extensive pseudopolypsis.

the lymph nodes. Scarring and pseudo-polypoid granulation tissue appear in the course of time (Fig. 66). In long-established cases there is a very strong tendency to development of carcinoma of the colon. The incidence of this complication depends upon the severity and duration of the disease and the age of the patient. As many as 40 per cent. of patients who have suffered from colitis for more than ten years may develop carcinoma, but the figure in most published series is in the region of 10 per cent., since many cases of shorter duration are included. Such carcinomas have a very bad prognosis, firstly because they spread rapidly in and through the damaged bowel wall, and secondly because they are often difficult to diagnose.

Initially there is swelling and hyperemia of the mucosa with petechiae and ulceration. The ulcers are usually fairly superficial but they sometimes extend into the muscle layers, and rarely perforation occurs. The mucosa between the ulcers is oedematous and gives rise to characteristic "pseudo-polypi" in advanced cases (Fig. 66). *Microscopically*, the lesions are largely confined to the mucosa and submucosa. There is marked infiltration with inflammatory cells, and there may be abscesses in the mucosal crypts or inflammation in the walls of the vessels with intravascular thrombosis. Inflammatory changes may be seen in

**Clinical Features.** More than half the patients with ulcerative colitis develop it between twenty and forty years of age, and even children are occasionally afflicted. Rarely does the disease appear for the first time over the age of fifty. At most sites women are afflicted rather more frequently than men. The most characteristic features are *diarrhœa*, accompanied by the passage of *mucus*, *pus* and *blood* in the stools. The rapidity of onset and severity of these symptoms varies a good deal from case to case. Sometimes there is abdominal discomfort and *colic*. *Pyrexia* is common and is indeed one of the most constant features of active disease. *Anæmia* is almost invariable and *loss of weight* may be marked. Cases may be divided into several main groups: (1) The acute fulminating type which may go on to rapid death; (2) the chronic relapsing type with repeated attacks over a long period of time; (3) the chronic persistent type in which, despite minor fluctuation in symptoms, there is never a complete remission. Patients in any of these groups may develop complications such as abscesses and fistulæ, infective arthritis, pyoderma, perforation or hæmorrhage.

On examination, there may be pyrexia, rapid pulse, and evidence of anæmia and weight loss. Abdominal signs are usually limited to tenderness over the colon which may be palpable. *Sigmoidoscopy* is an essential step in diagnosis. It may reveal changes varying from slight reddening and granularity of the mucosa to severe thickening with ragged ulcers and pseudo-polypi. The latter are sometimes easily palpable on digital examination of the rectum. *Barium enema* X-ray may show varying degrees of change. At first there is irritability of the bowel but ulceration is most difficult to detect. Later the bowel outline becomes smooth and the colon tends to be stiff, narrow and short (Fig. 67). Obvious pseudo-polypi and strictures may be seen.

**Differential Diagnosis.** Mild and early cases have to be differentiated from *mucus colitis* and other causes of *simple diarrhœa*. Only by continued observation of the case is certain diagnosis possible, and sometimes the diagnosis of ulcerative colitis is not established for some time. The more advanced and chronic cases are generally more easily diagnosed, but have to be distinguished from *chronic amœbic infection*. Localised varieties of ulcerative colitis must be distinguished from *carcinoma*, *diverticulitis* and *regional colitis* (Crohn's disease in the colon). True *polyposis coli* is distinguished from the pseudo-polyposis of advanced colitis by the normal mucosa between the polypi.

**Treatment.** In the first instance, the treatment of all cases of ulcerative colitis is strictly medical; the details are not discussed here. The essentials of such treatment are a high protein and low residue diet, correction of anæmia and relief of distressing bowel symptoms.

The indications for surgery are: (1) Chronic disease, with repeated relapse and invalidism, or with persistent symptoms despite intensive medical treatment; (2) complications, such as abscess, fistulæ, pyoderma, perforation, hæmorrhage and carcinoma.

*Time of Intervention.* It is essential that operation shall be undertaken at the right time, neither too early nor too late, and this demands good liaison and understanding between physician and surgeon. In fulminating cases, the disease can progress with such rapidity that surgery may be necessary within a month or even a week or two of the onset. Diarrhoea may be so intense, constitutional disturbance so extreme, and blood loss so massive, that emergency surgery is necessary. On the other hand, in some of the chronic cases, the disease may have followed a relatively mild



FIG. 67. Ulcerative colitis. Barium enema X-ray showing rigid smooth colon and free reflux into the dilated ileum. (Courtesy of Dr. L. S. Carstairs.)

course for several years, but with serious relapses which greatly interfere with the patient's working capacity and normal life. Sometimes there is surprisingly little correlation between the general condition of the patient and the state of the bowel on sigmoidoscopy or barium enema X-ray. In those patients with the more persistent type of chronic disease, surgery may be needed after a somewhat shorter interval, since the patient does not even enjoy the illusion of "cure" which a remission provides. In either the persistent or relapsing varieties of disease, the presence of complications loads the scales in favour of earlier surgery.

*Choice of Operation.* The operations to be considered are total procto-

colectomy with permanent ileostomy and, for selected cases, total colectomy with ileorectal anastomosis. Ileostomy alone has no place in the definitive treatment of ulcerative colitis, and whereas ileostomy was once widely practised as a preliminary to the total colectomy operation, it is now more usual to complete the operative treatment in one stage. Not so many years ago, it was customary to undertake ileostomy in the first instance, and only proceed to colectomy if there were complications, e.g. abscesses, fistulae or pyoderma. Now that the serious risk of subsequent carcinoma has been fully realised, colectomy is done as a routine.

*Preoperative Care.* Anaemia, hypoproteinaemia and vitamin depletion should be carefully corrected. Repeated blood transfusion until the haemoglobin is raised to 70 per cent. or more is essential. A high protein diet is sometimes difficult for these patients to take, but every effort is made to get them as well nourished as possible. Ascorbic acid and the vitamin B complex are the most important vitamins, and are preferably given by injection to ensure absorption and saturation. Prior to colectomy the bowel is prepared with sulphathalidine, 10 gm. daily, for five days. The possibility of salt depletion occurring in the early post-operative period following ileostomy should be anticipated. It is wise to accustom the patient to increased salt intake for a week or two prior to operation. An extra 5 gm. daily of sodium chloride may be given by mouth during the preoperative period, for this appears to help the patient to tolerate extra salt afterwards.

*Ileostomy.* Either a right paramedian or oblique incision may be used. A terminal ileostomy is made; the gut is divided 6 inches or less from the ileocaecal valve. The distal ileum is securely closed, and the proximal end is brought to the surface about  $1\frac{1}{2}$  inches from the umbilicus. The gut is held at the surface either with a small glass rod through the mesentery or by suturing the latter to the parietal peritoneum. It is sometimes advantageous to suture mucosa to the skin, after excising an ellipse of the latter, to minimise the possibility of stricture later. The space lateral to the ileum is closed to prevent obstruction due to the snaring of a loop of small gut at this site. Some surgeons like to bring the distal end of the ileum to the surface as well to prevent possible distension of the blind loop with secretions. Such a development is certainly rare, and only occurs if the ileocaecal valve is obstructed and if sufficient time elapses before the subsequent colectomy. The opening into the distal ileum is occasionally used to introduce antibiotics and chemotherapeutic agents into the colon before its removal.

*Post-operative Care.* It is wise to stick a suitable bag over the ileostomy opening from the time of operation. Initially this may be of the plastic disposable variety, which is replaced once daily or more often. In the first twenty-four to forty-eight hours, there is commonly little discharge, but after this it may be profuse for a few days. For this reason a careful check is kept on the fluid and electrolyte balance. Initially there is a



tendency for the skin to become digested despite the most careful protection with aluminium and zinc oxide cream. After two or three weeks the skin becomes sound and a permanent ileostomy bag can be used.

*Complications of Ileostomy.* A number of patients develop partial obstruction about ten or fourteen days after operation. This usually seems to be due to œdema of the stoma, and settles down in a few days. Later obstruction may occur due to adhesions within the abdomen. *Prolapse* of the ileostomy of some degree occurs in about 10 per cent. of cases. *Stenosis* of the stoma is usually due to fibrosis, and may be relieved by digital dilatation in most cases. A few require further operation to enlarge the opening. *Salt depletion* is always liable to occur if the patient develops diarrhœa. Therefore it is wise to warn all ileostomy patients that in the event of frequent and profuse action of the ileostomy, it is important to step up the salt intake by mouth. On occasion such patients go into a state of marked circulatory collapse as a result of the fluid and electrolyte loss.

*Second-stage Colectomy.* In many patients there is considerable improvement after ileostomy so that it may be wise to wait three to six months before proceeding to colectomy. This period can be shortened in the fitter patients or whenever there is deterioration of the condition after ileostomy. However, ileostomy and total colectomy is nowadays often done in one operation, but this may be unwise unless the patient has massive hæmorrhage from the colon which ileostomy alone will not help. Alternatively, at the time of ileostomy, the colon can be excised down to the sigmoid area (the distal end of the bowel being brought to the surface in the left iliac fossa). The rest of the colon and rectum is then excised at the second stage. For all second stage colectomies, the bowel is prepared with daily instillations of sulphathalidine 5 gm. and streptomycin 1 gm. for five days prior to operation. The technique of operation is mentioned later.

*"Hook-up" Operations.* In an endeavour to preserve something akin to normal bowel action some surgeons have practised ileorectal anastomosis following colectomy, but some of these patients are greatly troubled by the passage of loose stools, and there is always the possibility of subsequent carcinoma in the rectal stump. Most surgeons are, therefore, opposed to taking the risk of leaving the rectum behind, and this type of so-called "hook-up" operation is not widely favoured. Permanent ileostomy is usually the price which the patient has to pay for preservation of his health.

*Partial colectomy* is perfectly justified for those uncommon cases of localised ulcerative colitis. In some of these, however, there is subsequent recurrence elsewhere in the colon.

*Prognosis in Ulcerative Colitis.* It will be appreciated that the surgical programme outlined above is not devoid of risks for the patient. Quite apart from the mortality of ileostomy and of subsequent colectomy in

these cases there is also the possibility of later complications. However, the patient with ulcerative colitis who cannot be controlled by medical treatment is usually in a pathetic and often dangerous state. An ileostomy life is a welcome exchange for this state of affairs, and colectomy does afford protection from the very strong possibility of carcinoma later. Good teamwork between physician and surgeon is essential to the satisfactory management of such patients. Although none would wish to minimise the initial difficulties of many ileostomy patients it is gratifying to see how well the majority of them learn to cope with life and return to their former work.

### TUMOURS OF THE COLON AND RECTUM

**Benign Connective Tissue Tumours.** These are rare, and include lipoma, leiomyoma, fibroma and angioma. They all tend to become pedunculated, and the angioma, in particular, is liable to bleed. Other symptoms include colicky pain and occasional intussusception. Rarely they slough and are passed per rectum to effect spontaneous cure. In women, *endometrioma* is seen and this is usually associated with endometriosis elsewhere in the pelvis.

**Benign Epithelial Tumours.** Three types of benign epithelial tumour may occur in the colon and rectum. These tumours include adenoma, papilloma and multiple (familial) polyposis, the last of which is described separately (*vide infra*).

**Adenoma.** This type of tumour arises from the mucosal glands, and may be sessile or pedunculated. Any adenoma which becomes large tends to form a stalk, but it is not uncommon to observe small and rather sessile adenomas when sigmoidoscopying adult patients. Indeed the latter are present in between 5 and 10 per cent. of males over the age of forty. Adenomas higher up in the bowel are only discovered on barium enema X-ray; the large ones may cause intussusception, and malignant change is not uncommon. A special type seen in young children is the pedunculated rectal polyp which commonly bleeds and may trouble the child by prolapsing at stool. On microscopic section all adenomas show regular proliferation of glandular tissue.

**Papilloma.** In contrast to the relatively common adenoma which tends to become polypoid, the papilloma is an uncommon and usually sessile tumour. It occurs only in the rectum and is frequently large. The surface of such a tumour is covered by fine villous processes which consist of one or two layers of columnar cells with a delicate and vascular stroma. They often cause profuse watery discharge or bleeding. Malignant change is not uncommon.

**Treatment of Benign Tumours.** Adenomas in the rectum and colon within reach of the sigmoidoscope can be destroyed or removed by diathermy through that instrument. To minimise the risk of explosion of

bowel gases it is wise to run carbon dioxide gas into the rectum before diathermising a tumour of this type. Tumours higher up can only be dealt with at laparotomy. Although a pedunculated tumour in the colon can be readily removed through an incision in the bowel (*colotomy*), the possibility of malignancy in these tumours makes resection of the bowel a sounder procedure in most cases.

**Multiple (Familial) Polyposis.** This is a remarkable condition which is inherited as a Mendelian dominant characteristic. The bowel may be studded with hundreds or even thousands of adenomas of varying size though most of them are quite small. There is a great deal of variation both in the number and size of the polypi and also in the effects produced in different members of the same family. One member of a family may have marked symptoms, whereas another, with disease of similar extent, may have little or no trouble. Indeed it is not uncommon to demonstrate further asymptomatic cases simply as a result of routine search. Very rarely familial polyposis is associated with multiple familial sebaceous cysts, or with multiple melanin spots on the oral mucosa and digits. Malignant change occurs in most cases in the course of time but, whereas in some patients this may take place even before the age of twenty, in others it is delayed for many years.

**Clinical Features.** As already emphasised, the clinical features are very variable. Most patients defecate more frequently than normal, and mucus, and sometimes blood, is present in the stools. The diagnosis is only established by sigmoidoscopy and barium enema X-ray. Tannic acid with barium or special air-contrast techniques may have to be used to demonstrate these lesions. Differentiation from the pseudo-polyposis of ulcerative colitis is usually fairly simple, for in that condition the surface between the tumours is inflamed and the barium enema generally shows characteristic inflammatory changes in the bowel.

**Treatment.** To eradicate the disease and prevent any possibility of malignancy it would be necessary to excise the colon and rectum in all cases. However, since many of these patients are young folk to whom such a drastic procedure would often seem well-nigh intolerable, it is usual to adopt a more conservative plan. The alternative is to deal with all rectal polypi up to about 15 cm. by diathermy coagulation through a sigmoidoscope. When the disease here has been controlled total colectomy is done and the ileum is anastomosed end-to-end or side-to-end to the rectum. Such patients should be followed up at regular intervals for the rest of their lives so that fresh polypi in the rectum can be repeatedly diathermised.

*Total procto-colectomy and terminal ileostomy is the treatment of choice when familial polyposis is associated with carcinoma of the rectum. It is also necessary when the rectal polypi are so profuse that diathermy removal is impossible, or when, subsequent to a previous colectomy and ileorectal anastomosis, the rectum becomes more thickly involved. An*

abdominal ileostomy is established in these cases as experience has shown that ileo-anal anastomosis is usually quite intolerable to the patient.

### CARCINOMA OF THE COLON AND RECTUM

Carcinoma of the large bowel is very common. Thus some 20,000 people in England and Wales die each year from cancer of the colon and rectum. About 50 per cent. of these tumours are situated in the rectum and, although certain specific features of the latter disease are discussed later, it would seem best to discuss the basic problems together with carcinoma in the colon. Whereas there is little difference in the sex incidence of carcinoma of the colon, the rectal type is commoner in men (3/2). Some 90 per cent. of these tumours occur in people over the age of forty, but no age group is exempt, and young people are especially liable to be affected if they suffer from the rare familial polyposis coli. Further predisposing factors are other types of benign epithelial tumour and long-standing ulcerative colitis. The relationship between carcinoma and diverticulitis is, however, purely coincidental. Some 75 per cent. of carcinomas in the large bowel occur in the rectum and sigmoid colon, and a large proportion of these can be felt on digital examination or seen through a 30 cm. sigmoidoscope. That a fair proportion of carcinomas may develop from innocent polypi is suggested by the close similarity in the distribution of both varieties of tumour (Fig. 68).

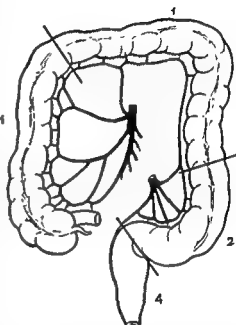


FIG. 68. Distribution of carcinoma in the large bowel. Of every eight growths roughly four are in the rectum, two in the sigmoid, one in the transverse or descending colon and one in the right colon. Adenomatous polypi have a similar distribution.

**Pathology.** These tumours are divided into three main groups: (1) Proliferative; (2) ulcerative; (3) annular.

*Proliferative growths* are commonest in the right colon, and are also seen in the rectum. They are usually bulky tumours and frequently ulcerate on the surface. It is the regular blood loss from an extensive surface which causes so many patients with carcinoma of the right colon to present with secondary anaemia. This type of tumour tends to spread into the lumen rather than the wall of the bowel and therefore does not metastasise so soon as the ulcerative type. However, the less ready occurrence of obstruction to the more liquid contents of the right colon,

with consequent delay in diagnosis, results in little difference in prognosis when compared with ulcerative growths on the left side.

*Ulcerative growths* are the commonest variety in the rectum, but may occur elsewhere as well. There is a raised nodular edge and an irregular base, with a tendency to penetrate the muscle coats of the bowel.

*Annular growths* are commonest in the pelvic colon and upper rectum. There is usually a good deal of scirrhus reaction in these tumours and, as they narrow the bowel, they are especially liable to cause obstruction on the left side. Spread to the lymph and blood vessels tends to be found most often in this type of growth.

*Microscopic Features.* The great majority of these tumours are adenocarcinomas which can be conveniently divided into low, intermediate or high grade malignancy. About two-thirds of cases are in the first group; they have a fair degree of cellular differentiation and less tendency to spread. The most undifferentiated and anaplastic tumours spread more rapidly to the glands and blood vessels. There are, however, a number of exceptions to these general rules, and it must be remembered that not all parts of a tumour show the same degree of differentiation. On the whole, the histology of the edge of a tumour gives the best idea of its character.

*Routes of Spread.* As with other growths of the gut spread may occur directly through the wall of the bowel and also through lymphatics and veins. *Direct spread* is relatively slow in the better differentiated tumours which constitute the majority of those seen. Until the growth has penetrated the muscle coat it is uncommon for lymph or blood vessels to be involved, but once the muscle barrier has been breached it is common for free spread to occur. Parts of the bowel which lack a serous coat (e.g. back of ascending and descending colon) may allow direct spread to occur more easily. *Lymphatic spread* to the regional glands tends to occur as soon as lymph vessels are invaded. Such spread is more or less strictly confined to the lymphatic territory of the affected part and, on an anatomical basis, it is possible to plan effective glandular clearance together with the primary growth. Many of the enlarged glands seen at operation or in the pathological specimen are the site of inflammation or reactive hyperplasia. For that reason the surgeon does not allow the superficial appearance of the mesentery to influence him unduly at the time of operation. *Blood spread* usually does not seem to occur until the muscle coat has been penetrated, but then it may take place quite independently of lymphatic spread. Careful examination of operation specimens from patients with rectal carcinoma shows that vein involvement has occurred in about one-fifth of cases. The details for carcinoma elsewhere in the large bowel are possibly of a similar order. The less differentiated the tumour, the more likely is venous spread to be found.

*Clinical Features.* The symptoms of carcinoma in the colon and rectum depend upon the type and site of the tumour, and are divided into the

following groups: (1) Large bowel symptoms; (2) other alimentary symptoms; (3) constitutional symptoms; (4) symptoms due to metastasis. There is some difference in the clinical picture with growths on the right and left of the colon. Rectal tumours cause additional symptoms peculiar to that particular region and are described later.

*Right Colon Cancer.* About one in eight of all tumours are on the right side. Obvious disturbance of the bowel habit is not a common or prominent early feature, for the lumen is relatively capacious and the contents liquid. The symptoms are more often those in the second and third groups referred to above. Thus symptoms of secondary anæmia, such as *lassitude*, *shortness of breath* and *pallor* are common, and digestive upsets, such as *anorexia* and *indigestion* may suggest upper abdominal disease. In roughly a quarter of these cases there is some alteration in the bowel habit, frequent loose stools and occasionally *intermittent diarrhœa* and *constipation*. It is unusual to see obvious blood in the stools, but the *occult blood* test is generally positive and may serve to heighten suspicion in a doubtful case. *Pain* consists either of a vague central abdominal ache, a more definite colic or discomfort in the right iliac fossa which is sometimes suggestive of appendicitis. In thin patients a lump in the right iliac fossa is frequently felt, and sometimes the patient himself reports the tumour before any other symptoms.

*Left Colon Cancer.* The smaller calibre, more solid fecal contents and the frequency of annular growths leads to a rather different symptomatology on the left side. The majority of patients present with increasing *constipation*, often with alternating bouts of *diarrhœa* and attacks of *colicky abdominal pain*. The latter is frequently associated with subjective *distension*. The patient states that the abdomen feels tight or that he has to loosen his clothes. *Mucus* and *blood* are commonly seen in the stools.

*Acute Obstruction due to Growth.* The proportion of cases which present for the first time with acute obstruction varies with the type of hospital from which these figures are collected. A busy district hospital may receive as many as 20 per cent. of its colon carcinoma cases in this condition, and nine out of ten of these growths are in the left colon. Obstruction on the right side is usually due to a relatively advanced annular carcinoma of the ascending colon, or one situated close to the ileocæcal valve.

*Metastasis.* Occasionally a patient presents with symptoms of hepatic metastasis, ascites or more distant deposits. An interesting feature of many such cases is that the primary tumour is often quite small. Indeed, as with many other growths, the aggressiveness of the primary tumour bears little relationship to its size.

*Diagnosis.* It cannot be emphasised too strongly that any change in the normal bowel habit in a patient over forty should be regarded with suspicion. It is melancholy to relate that the clinician sometimes finds

himself faced with advanced growths which just could not have been diagnosed any sooner. However, such cases are in the minority, and the doctor who cultivates a high index of suspicion will be rewarded by the prompt diagnosis of a number of more favourable cases. An isolated incident of rectal bleeding should not be labelled as "piles," nor a short-lived attack of diarrhoea as "enteritis," without proper investigation. General physical examination, digital examination of the rectum, sigmoidoscopy (including biopsy of any accessible lesion), and barium enema X-ray should be undertaken in that order (Figs. 69, 70 and 71). If sigmoidoscopy is handicapped by a loaded bowel, or the barium enema

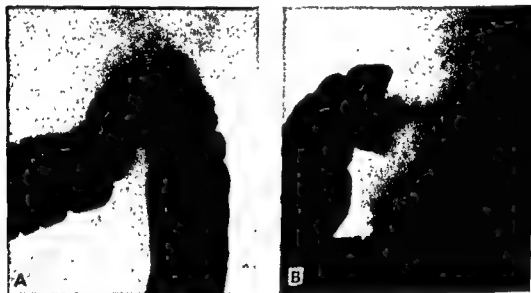


FIG. 69. Carcinoma of the splenic flexure. A. Not visible in A.P. film. B. Well shown in oblique view. (N.B. Proper manipulation of the patient is essential to demonstration of tumours at this site as also in the pelvic colon.) (Courtesy of Dr. L. S. Carstairs.)

examination is likewise impaired, it is wise to repeat these procedures. Persistence of symptoms after negative investigation should encourage the doctor to get his patient investigated again.

**Differential Diagnosis.** In many cases there is no doubt whatsoever about the diagnosis after sigmoidoscopy and X-ray. In the remainder, certain difficulties may be experienced. One of the commonest problems occurs in the differentiation of *diverticulitis* from carcinoma. If there is much peridiverticulitis an inflammatory tumour is formed which may closely resemble a carcinoma in the barium enema X-ray. Occasionally it is difficult to be sure of the diagnosis even at operation. The two conditions may coexist although there is no causal relationship, and sometimes the radiologist may be sorely exercised in trying to decide whether a narrowing in the colon has anything to do with diverticula demonstrated in the bowel nearby (Fig. 71). Other inflammatory



FIG. 70. Double carcinoma of transverse and pelvic colon. Barium enema X-ray. (Courtesy of Dr. L. S. Caretairs.)



FIG. 71. Polypoid adenocarcinoma of sigmoid, and diverticulosis.



conditions must also be considered. Patients with *Crohn's disease* confined to the colon, or in which the disease spreads from the terminal ileum, occasionally present with a tumour and other features suggestive of growth. *Hyperplastic tuberculosis* is a further possibility although many tend to regard this condition as Crohn's disease nowadays. *Actinomycosis* may occasionally give rise to confusion. The disease is rare, and in most cases its inflammatory character will be recognised. *Amœboma*, although generally rare in Great Britain, is a supremely important differential diagnosis in those who have lived abroad. Furthermore, it should be remembered that amœbomas have occurred in patients who have never left Britain (Chap. 11). *Benign tumours* of the colon, such as *adenoma* and the much less common *papilloma*, are so liable to become malignant that there need be no worry about the practical consequences of doing a colectomy in the mistaken belief that the tumour is a carcinoma. *Secondary anaemia*, often due to a right colon growth, is sometimes treated with iron for months or even years before the correct diagnosis is made. Unexplained anaemia, associated with digestive symptoms, demands investigation of the right colon as well as the stomach.

#### TREATMENT OF CARCINOMA OF THE COLON

In the absence of obstruction, the treatment of choice is primary resection and anastomosis. If there is acute obstruction the immediate object of treatment is to relieve this and to leave removal of the growth to a second operation.

**Unobstructed Cases.** It is most satisfactory for patient and surgeon if the disease is diagnosed before obstruction has occurred. However, in some of these cases, and especially with growths in the left colon, the bowel may be loaded with faeces and require most careful preparation. *Preoperative Treatment.*—Although sulphonamides and antibiotics can secure radical modification in the flora of the bowel, it cannot be emphasised too strongly that mechanical preparation of the colon is still very important. Generally the patient is admitted five days before operation. The preliminaries include *breathing exercises*, *low residue diet* and bowel preparation with one of the relatively insoluble sulphonamides. An initial dose of sulphathalidine 4 gm. is given, followed by 1.5 gm. six-hourly. The colon is washed out with water on the third day and repeated the night prior to operation. Those cases in which there is clinical evidence of a loaded bowel, or in which the initial wash-out seems unsatisfactory, may need longer preparation. Careful supervision by the surgeon and the collaboration of experienced nursing staff are of vital importance in these cases. Sulphathalidine achieves its optimum effect in three to five days and there is no point in giving it for longer. A warning should be sounded about too enthusiastic attempts to "sterilise" the bowel contents by the use of such antibiotics as streptomycin and neomycin. It is extremely doubtful whether any advantage is secured by

using these more powerful agents. Peaceful modification of the bacterial flora is probably safer than ambitious attempts to exterminate them completely, for there are thus none of the risks of resistant strain development or serious fungus infection.

Any marked anaemia should be corrected by transfusion ; but, to avoid hurried preparation, it is wise to check the haemoglobin percentage when the patient is first seen in consultation. In this way the need for longer general preparation is appreciated before the bowel preparation is started. In male patients, especially those requiring low colonic resection, it is wise to exclude the possibility of associated prostatism, for post-operative retention may seriously disturb a recent anastomosis.

*Operative Principles.* The object of operation is to remove the growth together with a sufficiently wide margin of bowel and the associated lymphatics. The details of the various procedures are discussed later. Even when hepatic metastasis is found, it is frequently wise to excise the primary tumour. Local fixation of the growth is often due to inflammation and, unless some vital structure is involved, this need not prevent radical resection. It is not uncommon to include parts of the abdominal wall, and even the uterus or part of the bladder, in the resected specimen.

If the local disease is clearly advanced and irremovable, or if there is extensive hepatic and other metastasis, it is usually best to by-pass the growth by side-to-side colo-colic anastomosis.

*Obstructed Cases.* In the presence of obstruction, it is usually necessary to undertake operative treatment in two or three stages. With obstruction in the distal part of the left colon a right transverse colostomy is admirable. If the growth is higher up in the bowel such a colostomy may prejudice the later resection, and in such cases a caecostomy is preferable. Obstruction due to a tumour in the right colon is relieved by preliminary ileotransverse colostomy.

The interval between the first and second stages allows the obstructed bowel to settle down and also permits proper preparation of the colon between colostomy and growth. The second operation is done as soon as possible afterwards, and generally within three weeks of the first stage. Any colostomy present is sealed off and the operation is done in a clean field. The colostomy is closed at a third operation a week or ten days later, for this discourages any tendency to stenosis of the anastomosis. Alternatively, the colostomy can sometimes be closed at the time of the resection and so save the patient a further operation.

*Mildly Obstructed Poor-risk Cases.* It was the prohibitive mortality of colonic resection operations in the latter part of the last century which led to the introduction of exteriorisation operations. This procedure was tried independently by several different surgeons but Paul, of Liverpool, and Mikulicz, a German, usually share the credit for its introduction. Although some surgeons rarely use the Paul-Mikulicz operation to-day it is important to realise what a great advance it represented in its time.

However, even to-day, with the poor-risk patient who has a mild degree of obstruction, it is often safer to exteriorise the bowel rather than run the risks of primary resection and anastomosis.

### Summary of Treatment

(1) Without Obstruction. One-stage resection and anastomosis.

(2) With Obstruction. (a) Right colon : First-stage, ileo-transverse colostomy ; second-stage, right hemicolectomy. (b) Left colon : first-stage, transverse colostomy in most cases and caecostomy for proximal growths ; second-stage, resection of growth ; third-stage, colostomy closure.

(3) Mild obstruction and poor-risk cases. Paul-Mikulicz operation ; second-stage closure of colostomy.

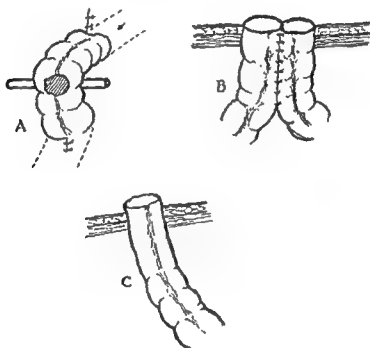


FIG. 72. Colostomy. (A) Simple loop. (B) Spur (double-barrelled). (C) End colostomy.

### OPERATIONS ON THE COLON

**Colostomy.** The making of an artificial anus is used : (1) To relieve obstruction ; (2) to rest the distal bowel ; (3) to facilitate preparation of the distal bowel for operation. There are three main types of colostomy : end, loop and dysfunctioning (Fig. 72). **End Colostomy.**—This is the type made after excision of the rectum. The bowel above the resected specimen is brought out in the left iliac fossa well clear of the anterior superior iliac spine, and it can either be held up by a glass rod passed through the mesentery or by suturing the latter to the parietes. Some surgeons leave

about an inch of bowel protruding and trim it later, whereas others prefer to suture the mucosa to the skin at the time of operation. The lateral space between the colon and the parietal peritoneum of the paracolic gutter is closed with a catgut suture to prevent possible obstruction to a loop of small gut. *Loop Colostomy.*—This is most commonly used for temporary drainage, and is situated in the transverse or pelvic colon; the bowel is held up by a glass rod passed through the mesentery. *Dysfunctioning Colostomy.*—The object of this type of colostomy is to prevent contamination of the distal bowel as, for example, in the preliminary treatment of a rectovesical fistula. Several different methods are used, and these vary from simple division of a loop of bowel to separation of the ends by a bridge of skin. A further type of colostomy with a "double-barrel" opening is described below in connection with the Paul-Mikulicz operation.

*Complications of Colostomy.* The main complications of colostomy are : (1) *Prolapse.*—This may be partly prevented by avoiding leaving redundant bowel above. (2) *Obstruction.*—Usually due to snaring of a loop of small gut lateral to the emerging bowel. This can be avoided by proper closure of the lateral space. (3) *Stricture.*—This generally occurs at skin level and can be obviated by excising an ellipse of skin when the colostomy is first made.

*Closure of Colostomy.* A temporary colostomy made for the relief of large bowel obstruction, or one formed as part of the Paul-Mikulicz procedure (*vide infra*) is normally closed as soon as possible. However, if this is attempted too soon, there is frequently a good deal of œdema of the tissues around the stoma which handicaps dissection. When a simple loop colostomy is fashioned, it is usual to remove the glass rod after ten or fourteen days unless, for any special reason, the surgeon wishes to prevent the loop sinking in too soon. At the time of closure, an elliptical incision is made around the stoma and all cutaneous and subcutaneous scar is removed. The incision is carried down to the extraperitoneal tissues to facilitate mobilisation of the bowel. In some cases, the whole procedure can be done extraperitoneally, whereas in others it is advisable to enter the peritoneal cavity, and there is little risk in so doing. The defect in the colon is closed transversely with interrupted inverting catgut sutures, usually in two layers. After this, the abdominal wall is sutured. Sometimes the muscle layers have become fused at the edge of the defect, and there is then no point in trying to close them separately. Occasionally there is a small faecal leak a few days after operation; but, in the absence of any obstruction distally, the small opening usually closes quite soon.

*Resection of the Colon.* In the majority of cases resection is undertaken for the treatment of carcinoma, and is planned so as to remove an adequate length of bowel together with the regional lymphatic field. The orthodox extent of resections is shown in Fig. 73, but lately some surgeons have tended to resect a greater amount of colon and mesentery on the left side.

However, even to-day, with the poor-risk patient who has a mild degree of obstruction, it is often safer to exteriorise the bowel rather than run the risks of primary resection and anastomosis.

### Summary of Treatment

(1) Without Obstruction. One-stage resection and anastomosis.

(2) With Obstruction. (a) Right colon: First-stage, ileo-transverse colostomy; second-stage, right hemicolectomy. (b) Left colon: first-stage, transverse colostomy in most cases and caecostomy for proximal growths; second-stage, resection of growth; third-stage, colostomy closure.

(3) Mild obstruction and poor-risk cases. Paul-Mikulicz operation; second-stage closure of colostomy.

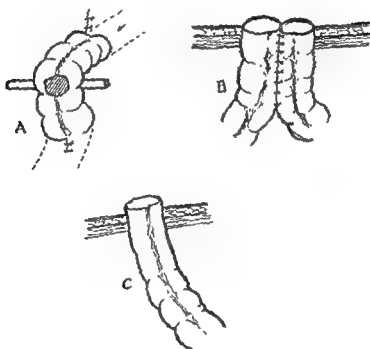


FIG. 72. Colostomy. (A) Simple loop. (B) Spur (double-barrelled). (C) End colostomy.

### OPERATIONS ON THE COLON

**Colostomy.** The making of an artificial anus is used: (1) To relieve obstruction; (2) to rest the distal bowel; (3) to facilitate preparation of the distal bowel for operation. There are three main types of colostomy: end, loop and dysfunctioning (Fig. 72). **End Colostomy.**—This is the type made after excision of the rectum. The bowel above the resected specimen is brought out in the left iliac fossa well clear of the anterior superior iliac spine, and it can either be held up by a glass rod passed through the mesentery or by suturing the latter to the parietes. Some surgeons leave

cecostomy opening as a safety valve, but others doubt the value of this procedure.

(c) *Exteriorisation (Paul-Mikulicz) Operation.* This type of operation was originally limited to the more mobile parts of the colon, but any part of the bowel may be exteriorised after suitable mobilisation. The colon and its mesentery are prepared for resection as for a closed anastomosis, but the parts of the bowel immediately proximal and distal to the specimen are sutured together for about 3 inches. The loop to be resected is then exteriorised, usually through a separate incision, and after its removal a double barrel opening is left. The two ends of the bowel are often best held up by applying clamps which are left on for twenty-four to forty-eight hours. The object of the double-barrel opening is to facilitate subsequent restoration of continuity of the colon. Accordingly, after two or three weeks, the spur between the two ends of bowel is crushed with a special clamp (enterotome) which is tightly applied and usually severs the spur in the course of a few days (Fig. 74). Sometimes the enterotome has to be applied again to complete the crushing of the spur. Continuity of the colon is subsequently completed by closure of the colostomy after an interval of a few weeks.

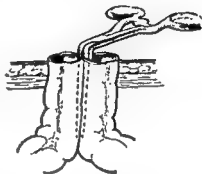


FIG. 74. Crushing clamp applied to spur of double-barrelled colostomy.

(d) *Total Colectomy.* This operation is used only in the treatment of ulcerative colitis and multiple (familial) polyposis coli. An ileostomy is made either at an earlier operation or at the time of the colectomy. A single long left paramedian incision suffices for many patients, but sometimes a second incision on the right side is also necessary. The splenic flexure, which is the least accessible part of the bowel, is often the most difficult part to get at, but in chronic ulcerative colitis the bowel is often much shortened and relatively easily removed. A decision as to whether the operation is done in one, two or more stages depends upon the circumstances of the case and the experience of the surgeon. Total proctocolectomy is sometimes done in one stage together with ileostomy. Alternatively, the ileostomy can be established first and proctocolectomy effected at the next stage. Some prefer to remove the bowel down to the sigmoid colon at the first stage, the distal end being brought out in the left iliac fossa. In both ulcerative colitis and multiple polyposis, there is no need to resect the mesentery unless, of course, the latter condition is associated with a carcinoma.

(e) *By-pass Operations for Inoperable Growths.* A palliative colocolostomy may be done in cases of inoperable carcinoma of the left colon. Usually a side-to-side anastomosis is made well clear of the affected part.

It is doubtful whether the latter operations achieve results which are any better than those secured by the more orthodox procedures. In the case of resection for diverticulitis and other inflammatory conditions removal of the lymphatic field is unnecessary.

(a) *Right Hemicolectomy.* The resection includes the last 6 inches or so of ileum, and the colon up to the junction of right and middle thirds of the transverse colon. The ileocolic and right colic arteries are ligated close to their origin from the superior mesenteric vessels (Fig. 73). Continuity

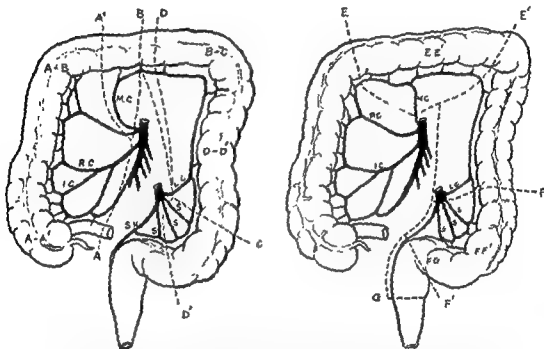


FIG. 73. Carcinoma of colon. Extent of resection for different growths. A-A', cecum and ascending colon. A-B, hepatic flexure. B-C, splenic flexure. D-D', descending colon. E-E', transverse colon. E'-F', more extensive resection for left colon. F-F', sigmoid colon. F-G, rectosigmoid.

of the bowel is restored in one of several ways. The anastomosis of ileum to colon may be end-to-side, side-to-end, side-to-side or even end-to-end. It is fair to say that each method can be equally successful in the hands of the surgeon who practises it regularly and that no one method is ideal for all cases. Such an anastomosis must be meticulously carried out with a seromuscular layer of interrupted non-absorbable sutures, and an inner all-coats layer of fine catgut. The mesenteric defect is repaired with interrupted sutures, and usually no special effort is made to reperitonise the raw area unless this can be done without tension or difficulty.

(b) *Resection of the Transverse and Left Colon.* The extent of the resection for growths in different parts of the colon is shown in Fig. 73. Mesentery and bowel are resected, and it is most usual for continuity to be restored by end-to-end anastomosis. Some surgeons like to provide a

## THE COLON, RECTUM AND ANAL CANAL

separates the two sphincters and also sends fibres (anal int. septum) through the internal sphincter and they condense under canal skin. The external sphincter is divided into three parts. The deep part is contiguous with the sling-like puborectalis fibre levator ani muscle. These fibres of the puborectalis muscle form anorectal ring which is a prominent landmark on digital palpation. The deep and superficial parts of the external sphincter form the roof of the ischioanal space whereas the subcutaneous part lies in the perianal space.

**The Blood Vessels.** In the healthy subject, the blood supply of the rectum is so good that it is almost impossible to jeopardise its nutrition at operation. The superior hæmorrhoidal artery divides into right and left branches, and the right vessel subdivides again. Internal hæmorrhoids usually develop in relation to these main subdivisions. The right posterior and left anterior branches occasionally divide again, and when secondary hæmorrhoids occur they are usually related to the primary hæmorrhoids at these two sites (Fig. 76). The tributaries of the superior hæmorrhoidal vein tend to be grouped with the arterial branches. The main vessel drains into the inferior mesenteric vein. The middle and inferior hæmorrhoidal veins enter the internal iliac. The anastomosis between veins of the portal and systemic circulation occurs in the region of the white line where there is free communication between the internal and external hæmorrhoidal plexuses.



FIG. 76. Position of primary and secondary hæmorrhoids.

### INTERNAL HÆMORRHOIDS (PILES)

Amongst patients it is customary to attribute almost any symptoms to piles but careful examination reveals such patients suffering from a variety of complaints. True hæmorrhoids only involve the internal hæmorrhoidal plexus and are referred to as internal hæmorrhoids. Extravasation of blood from the external plexus forms a *perianal hæmatoma* which is sometimes misleadingly called an hæmorrhoid.

**Ætiology.** Degenerative changes in the hæmorrhoidal vein walls, those occurring in the superficial veins of the lower limbs, are the chief ætiological factor. Excessive straining at stool, the unaccustomed exercise by otherwise sedentary people, and other factors may also play some part in the production of hæmorrhoids.

**Clinical Features.** Bleeding at stool is the commonest symptom.



On the right side, an ileotransverse colostomy is done. Such procedures are reserved for those patients with obstructive symptoms and extensive hepatic metastasis, and those in whom the growth is locally inoperable.

### THE RECTUM AND ANAL CANAL

Whereas the rectum is lined by columnar-cell epithelium and loosely attached mucosa, the upper half of the anal canal has vertically disposed

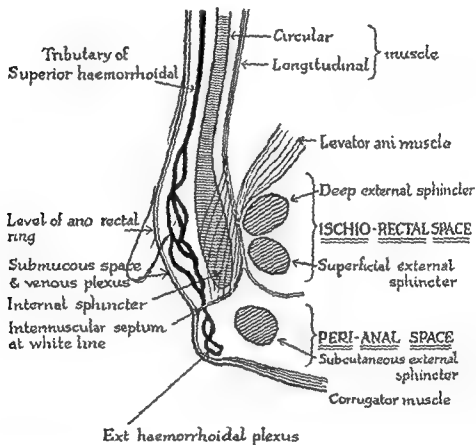


FIG. 75. Anatomy of rectum and anal canal. Longitudinal section showing arrangement of muscles and spaces.

folds (*columns of Morgagni*) which are covered by a flatter epithelium. In the middle of the anal canal the junction between this flatter epithelium and the squamous lining of the lower half is marked by the crescentic "valves" of Ball. This junction, sometimes described as the *white line*, also marks the point of attachment of the *intermuscular septum* derived from the longitudinal muscle layer of the rectum.

**The Sphincter Muscles.** The external sphincter ensheathes the internal sphincter which is continuous with the circular muscle layer of the rectum (Fig. 75). The lower part of the longitudinal muscle layer

prolapsed and thrombosed piles. In the more severe cases, the patient is put to bed with the foot of the bed raised. Compresses of lotio plumbi evaporans B.P.C. are applied locally. The swelling and inflammation settles down within two or three weeks, and by then the condition can be reassessed and a decision made about further treatment. Operation should not be done in the acute stage, for there may be exacerbation of infection, and, furthermore, it is extremely difficult to plan a satisfactory operation at that time.

(2) *Injection Treatment.* This is suitable for uncomplicated piles of the first and second degree. The greater the degree of prolapse and stretching of the tissues the less is the likelihood of success with injection treatment. Third degree piles, or very fibrous ones with an associated polyp, should usually be dealt with by operation. It is, however, quite reasonable to try injections in the first instance in some third degree cases. Injections are contra-indicated in the presence of infection or fissure, and they certainly do no good in those patients who have marked stretching of the longitudinal muscle attachment (Fig. 77). The injection solution most commonly used consists of 5 per cent. phenol in almond oil with 4 mg. menthol to 1 ml. The injection is made with a 10 ml. syringe using special needles which are strong enough and also long enough to reach through a proctoscope. After the preliminary examination, the proctoscope is passed above the anorectal ring. The patient is encouraged to relax and breathe quietly, and the injection is given into the submucosa just above the level of the anorectal ring. It is important to see the mucosa raised by the injection. No swelling suggests that the injection is going in too deeply, whereas a white patch in the mucosa shows that injection is too superficial. The usual technique is to inject enough solution to produce a prominent balloon at each pile site. An average of 3 ml. is commonly used at each site although, with a very lax mucosa, as much as 10 ml. can be injected at one place. Most surgeons prefer to avoid injecting a total of more than 20 ml. at one session, and generally the amount used is a good deal less than this. The patient is allowed to continue with his normal activities immediately afterwards.

The results of injection are assessed by the symptomatic improvement and also by subsequent digital examination. Within two weeks of treatment an indurated plaque can usually be felt at the injection site and this is an indication that a good reaction has been produced. Further injections are given at intervals of ten to fourteen days until the sub-mucous sclerosis is sufficient to fix the mucosa and tighten the stretched tissues. Rarely are more than two or three visits necessary, and the

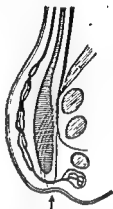


FIG. 77. Showing how stretching of longitudinal muscle allows prolapse to occur.

The results of injection are assessed by the symptomatic improvement and also by subsequent digital examination. Within two weeks of treatment an indurated plaque can usually be felt at the injection site and this is an indication that a good reaction has been produced. Further injections are given at intervals of ten to fourteen days until the sub-mucous sclerosis is sufficient to fix the mucosa and tighten the stretched tissues. Rarely are more than two or three visits necessary, and the

usually the first. The blood sometimes splashes around the lavatory pan or dribbles away immediately after defaecation. With longer established trouble there may be bleeding between stools and soiling of the underwear. This state of affairs, in the absence of prolapse, is referred to as *first degree hæmorrhoids*. Prolapse at stool is at first followed by spontaneous reduction, but sometimes a little manual assistance may be necessary (*second degree*). Eventually the piles may prolapse almost every time the patient gets up and about, and at this stage they are referred to as of the *third degree*. Prolapse is not possible until the longitudinal muscle attachment is stretched. Discharge is not uncommon and may cause *pruritus*. The discharge usually consists of mucus, but sometimes there is leakage of paraffin in those who make this their favourite nightly "tipple." Pain, contrary to the layman's ideas, is not a feature of uncomplicated piles. When pain occurs there is usually thrombophlebitis, infection, an associated fissure or an abscess.

*Secondary anæmia* is not uncommon in those with bleeding piles, and its onset may be so insidious that the cause of the symptoms may be overlooked. Occasionally the hæmoglobin level may be greatly reduced, even to the region of 25 per cent. or less, and the patient may report with shortness of breath or angina of effort as a result of it. In such cases, the bleeding piles may have been present so long that the patient regards them as part of his normal existence.

Other complications of hæmorrhoids may occur. *Thrombosis* in the hæmorrhoidal veins, or prolapse with *strangulation* may cause considerable pain. *Submucous abscess* may be secondary to thrombosis or result from ulceration of the mucosa. *Sloughing* of piles sometimes follows strangulation, and occasionally a spontaneous cure is so effected. *Portal pyæmia* as a complication of infected piles is so excessively rare that, even when it is suspected in a patient with prolapsed and infected piles, it is usually wise to think of some other cause of the pyrexia and rigors.

Uncomplicated internal piles cannot be detected on digital examination unless they are very large, but thrombosed ones are readily felt. *Proctoscopy* is essential to diagnosis. If the instrument is gently passed to full length and then slowly withdrawn to the anorectal ring the piles appear and they are seen to enlarge if the patient strains. Blood is sometimes present in the rectum higher up. *In every case of suspected hæmorrhoids sigmoidoscopy should be done as a routine.* Only in this way is it possible to exclude a neoplasm higher up in the rectum.

**Treatment.** (1) *Palliative.* In many cases of mild degree, the symptoms have been aggravated by habitual constipation or excessive purgation, and simple attention to the diet and the bowel habit is all that these patients need. Sometimes there is associated rectal congestion and discomfort which can be relieved by suppositories of hamamelis and zinc oxide B.P.C., or by the insertion of zinc oxide and castor oil ointment B.P. Palliative treatment is also called for in those patients who present with

hours after operation, and from then onwards the patient "floats" his dressing off in a bath and subsequently has fresh eusol dressings applied. A lubricated finger is passed on the sixth day, and subsequently a St. Mark's type anal dilator is inserted daily until healing is completed by about the end of the third week. The patient is normally ready to go home about the tenth or twelfth day.

**Complications of Operation.** Pain is variable and is generally more troublesome in anxious and unrelaxed patients. Morphine is given when needed during the first twenty-four hours. Patients often greatly fear the first bowel movement, and much good can be done by skilled and reassuring nursing. *Retention of urine* is commoner in male patients. It may be relieved by subcutaneous injection of carbachol 1 ml. but occasionally catheterisation is needed. *Reactionary bleeding* within a few hours of operation may be due to a slipped ligature, but sometimes comes from tiny vessels close to the internal sphincter. If persistent, it may be necessary to return the patient to the operation theatre for control of the bleeding point. *Secondary hæmorrhage* is uncommon, and is more likely if there is sepsis. All blood clots are evacuated through a proctoscope, and the rectum is packed for forty-eight hours with paraffin gauze wrapped around a rubber tube. A *fissure* may form from a persistent dorsal wound; and an *abscess* or *fistula* occasionally occurs. *Stricture* is a rare sequel to the modern operation, and should be completely avoided if adequate skin bridges are left between the pile sites. Recurrence of hæmorrhoids following operation is certainly very uncommon. It may occur if only one or two of the primary hæmorrhoid sites have been dealt with, or if secondary hæmorrhoids later enlarge.

**External Hæmorrhoids.** So-called external hæmorrhoids consist either of extravasated subcutaneous blood or of various types of skin tag. *Anal hæmatoma* is of two types. The commoner variety results from rupture of a subcutaneous vein and forms a painful, tender, bluish and globular swelling at or just within the anal margin. The second type is more deeply situated under the corrugator muscle and is due to rupture of the external hæmorrhoidal plexus. It usually forms a more diffuse swelling. *Both types of anal hæmatoma appear quite suddenly, and cause sharp pain* for a day or two, and then the discomfort gradually eases. Ulceration of the skin may occur and be followed by spontaneous evacuation or infection of the clot. It is important to differentiate anal hæmatoma from prolapsed and thrombosed internal hæmorrhoids; there is no difficulty about this if a careful examination is made.

**Treatment.** A painful hæmatoma, seen shortly after its appearance, should be evacuated under local anæsthesia and sufficient skin excised to leave a clean elliptical wound. A small eusol dressing is applied, and normally the patient can continue at work without trouble. Daily bowel movement is encouraged. The small wound rarely causes more than slight discomfort. When a patient is seen several days after the onset, the pain

results are extremely gratifying in those who have bleeding alone. With second degree piles the results are less certain. In third degree piles with stretching of the longitudinal muscle, no more than temporary relief can be anticipated. If symptoms recur it is possible that the response to treatment will not be so successful on a second occasion. Nevertheless, there are some patients whose symptoms are well controlled by repeated injections over a period of years.

*Complications of Injection.* These include mucosal ulceration and submucous abscess; hæmaturia, due to puncture of the prostate in the male; rectal granuloma; and fibrous stricture. These last two conditions are usually due to the use of excessively strong or otherwise unsuitable solutions; they are exceedingly rare with the use of 5 per cent. phenol in almond oil.

(3) *Operative Treatment.* Operation is indicated in third degree piles and also in those of lesser degree which have failed to respond to injection. From the point of view of results, the operation of hæmorrhoidectomy is one of the most satisfying in surgery. *Preoperative Preparation.*—Two days prior to operation a suitable aperient, such as cascara, senna or magnesia, is given. The next day the patient has an enema which is followed by a rectal wash-out of water a few hours before operation. *The Operation.*—Several different techniques are used, but one of the most satisfactory is the method of ligature and excision used at *St. Mark's Hospital, London*. The operation is usually done under general anaesthesia with the patient in the lithotomy position. Each of the skin tags at the site of the three main hæmorrhoids is grasped in forceps and held outwards. The pedicles of the piles thus displayed are then caught with Kocher tissue forceps. The forceps at each pile site are then drawn towards the opposite side of the anus. The skin is cut with scissors on each side of the skin forceps. The muscle fibres of the corrugator cutis ani and the internal sphincter are exposed in this wound (Fig. 75), and the pile is dissected up until the fibres of the longitudinal muscle are exposed in the pedicle. The latter is now transfixed and ligated with No. 10 tubular silk and the pile is drawn firmly downwards before it is cut off just below the ligature. After excision of each of the piles the resultant wounds are inspected. The skin is trimmed so as to afford good drainage and leave an adequate bridge between each of the raw areas. Any redundant skin and prominent parts of the external hæmorrhoidal plexus may need to be reduced, but it is important to avoid "flaying" the anus. The three raw areas are dressed either with small squares of soft paraffin mesh or with plain gauze soaked in half-strength eusol. A soft tube in the anus helps to show up any post-operative bleeding and also lets out wind.

*Post-operative Care.* For most patients it is probably best to have the bowels confined for three or four days. An aperient is given on the third evening. The soft rubber tube is removed twenty-four to forty-eight

cured by the application of 3 per cent. amethocaine ointment for ten to fourteen days. This is either applied digitally or on a St. Mark's dilator which is inserted twice daily. If the ointment is applied ten minutes or so before bowel movement, the latter can usually be undertaken relatively painlessly. After a fortnight, there is usually little pain, but if the fissure is still unhealed it is usually wise to substitute zinc and castor oil ointment B.P., for continued use of the amethocaine may cause dermatitis. When there is much sphincter spasm, it is sometimes helpful to inject an oily anaesthetic solution of proctocaine.\* Five ml. of this solution is injected deeply on each side of the fissure under the strictest aseptic precautions.

*Chronic Fissure.*—In dorsal fissures with much induration, the key to successful treatment is division of the internal sphincter muscle in the base of the ulcer. This can be done under local or general anaesthesia. The wound should be flared out for at least an inch beyond the anal margin to ensure adequate drainage (Fig. 78). Adjacent skin tags are excised at the same time. A cusol dressing is applied and the initial management of the wound is on similar lines to that following hæmorrhoidectomy. Simple analgesics relieve any discomfort during the first few days, and it is wise to pass a dilator daily from the sixth day until there is complete healing. In the uncommon type of anterior fissure it is wise to divide the sphincter to one side of the midline, for better healing is thus achieved.

**Anorectal Abscess.** Pyogenic abscesses are common in the anorectal region, and tuberculous abscesses are not uncommon. The various types of anorectal abscess are shown in Fig. 79. A *perianal* abscess may develop from a hæmatoma, fissure, or infected pile. There is pain, swelling and tenderness close to the anal margin. An *ischiorectal abscess* usually arises from a fissure, an infected crypt or inflammation in the intramuscular glands which open into the crypts. Pus can also spread to this site from the subcutaneous tissues and supralelevator region. In most cases there is throbbing pain, pyrexia and quite marked constitutional upset. Rectal examination reveals a firm, tender, and sometimes hot mass. Pus may

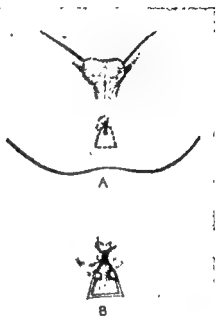


Fig. 78. Chronic fissure-in-ano. A. Extent of resection. B. Division of lower fibres of internal sphincter.

\* Proctocaine contains Butesin 6 per cent., procaine base 1 per cent., phenol 1 per cent., benzyl alcohol 5 per cent. in sterile almond oil.

is usually easier and there is no point in interfering with a resolving hæmatoma. The more extensive deep variety, due to extravasation from the external hæmorrhoidal plexus, is sometimes associated with internal hæmorrhoids. It is best treated by rest in bed for a few days with local application of *lotio plumbi evaporans*, B.P.C.

**Anal Fissure.** Anal fissure is due to a superficial tear in the skin, usually due to the passage of a hard stool. Most fissures are situated dorsally. Anterior ones are uncommon in men, but they are commoner in women in whom they may follow stretching of the perineum at childbirth. Apart from simple splitting of the skin a fissure may also be due to tearing downwards of one of the valves of Ball, or may follow infection in the anal crypts. The latter type tends to be multiple and is more common in women.

**Pathology.** The anal skin usually tears at the level of the internal sphincter (Fig. 75). Fibrosis of the latter, as a result of habitual purgation with consequent loose stools, may be a contributory factor in some cases. Initially the lesion is quite superficial, with only the fibres of the corrugator muscle exposed. Later on, the fibres of the internal sphincter may be seen in the base of the fissure. In more chronic cases there is a good deal of induration, and a large skin tag (*sentinel pile*) may appear at the lower end.

**Clinical Features.** Pain is the most characteristic feature. It is commonly severe, sharp or cutting in character, and occurs at or after evacuation of the bowel. It may persist for several hours, and the patient is often fearful of the next bowel movement. A vicious circle is thus set up, for delayed evacuation leads to harder motions, and these, in their turn, aggravate the trouble. Slight blood loss is sometimes noted on the toilet paper. Discharge of mucus is usually slight and may cause *pruritus*. Discharge of pus suggests underlying abscess formation. Examination of the patient must be exceedingly gentle. On separating the buttocks a sentinel tag may be seen, or the lower end of the fissure may be visible. Before proceeding further it is wise to apply a small dab of 10 per cent. amethocaine ointment on a throat swab. Within a few minutes it is possible to perform digital examination without serious discomfort. In acute cases, spasm of the sphincter is often marked. In long-standing cases there is much induration and, if there has been an associated abscess, fistula occasionally occurs.

**Differential Diagnosis.** Fissure must be differentiated from other ulcers of the anal canal most of which cause similar symptoms. *Tuberculous ulcers* usually have undermined edges and tend to develop in patients with open pulmonary tuberculosis. *Syphilis* may present either as a chancre or as multiple fissures with associated condylomas in the secondary stage. *Carcinoma* of the anus may be difficult to identify in the early stages, but a biopsy usually establishes the diagnosis.

**Treatment.** *Acute Fissure.*—The majority of cases can be quickly

**Fistula-in-Ano.** The great majority of fistulae result from pyogenic infection in relation to the anus and rectum. Such a fistula is a track lined with granulation tissue leading from an abscess cavity to the anal canal or skin. Spontaneous healing is impossible, but some fistulae remain quiescent for long periods and only discharge intermittently. It is convenient to classify fistulae into three main groups:—(1) Pyogenic fistula which nearly always originates from an abscess in the anorectal region; (2) tuberculous fistulae which comprise about 10 per cent. of cases in Great Britain. The patient is commonly suffering from pulmonary tuberculosis. The routine histological examination of operative material usually ensures that a tuberculous infection is not overlooked; (3) special fistulae. Failure to recognise the nature of such fistulae may lead to disappointing results. This group includes fistulae due to osteomyelitis of the pelvis, specific infections such as *Crohn's disease*, ulcerative colitis, actinomycosis and lymphogranuloma venereum and, lastly, those due to malignant disease.

On an anatomical basis fistulae may be classified as:—(1) Superficial. (a) Subcutaneous. (b) Submucous. (2) Anal. (a) Low. (b) High. (3)

Complicated. (a) Ischiorectal (sometimes called anorectal). (b) Pelvi-rectal. The first two groups make up roughly 95 per cent. of cases. The low anal fistula is the commonest type, and constitutes about 75 per cent. of all cases. The relationship of these various fistulae to the sphincters and rectal musculature is shown in Fig. 80.

**Clinical Features.** The symptoms in most cases are those of the initial abscess followed by persistent discharge from the track. An external opening is seen, and one in the anal canal can be palpated or inspected through a proctoscope. The extent of most superficial fistulae can usually be readily demonstrated with a probe. More complicated fistulae can only be fully defined at operation. Odd features in the history, or the presence of suspicious induration extending up the wall of the rectum may make the clinician suspect an unusual cause. In the less common types of fistulae, for example those arising from osteomyelitis of the pelvis, injection of lipiodol into the track and subsequent X-ray may help in the diagnosis. Sometimes a careful search must be made for unusual infections such as actinomycosis.

**Treatment.** The principles of treatment are to exclude underlying specific disease, and then, if practicable, to lay open the fistulous track.

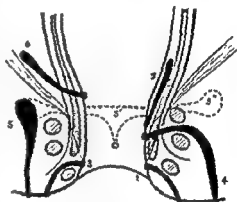


FIG. 80. Anatomy of fistula. (1) and (3) Low anal. (2) Submucous. (4) High anal. (5) Ischiocutaneous. This may have ischioanal (5') and horseshoe (5'') extensions. (6) High pelvi-rectal.



spread behind the rectum from one ischiorectal fossa to the other to form a so-called horseshoe abscess, and this may discharge into the bowel through a posterior fistula. A *submucous abscess* can follow a fissure, infection of piles or their injection treatment. It gives rise to a tender, indurated or soft swelling in the region of the anorectal ring. A *pelvirectal abscess*, although uncommon, is important to recognise. The infection arises from the parametrium or from the prostate and vesicles. There is often deep-seated pain, pyrexia and malaise. Increased frequency of micturition and dysuria are not infrequent, and walking is often slow or painful due to the relationship of the abscess to the pelvic muscles. It may be extremely difficult to locate such an abscess at first, and sometimes

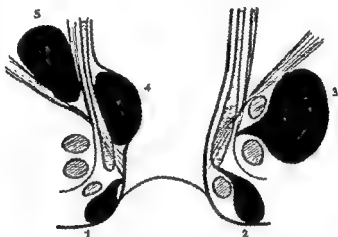


FIG. 7D. Anorectal abscesses. (1) and (2) Perianal. (3) Ischiorectal. (4) Submucous. (5) Pelvirectal.

the true state of affairs is only appreciated after the lapse of time when pus tracks to the surface. The differential diagnosis is from pelvic abscess and other varieties of pelvic inflammation.

**Treatment.** All these abscesses should be drained as soon as possible. Not only does drainage promptly relieve pain and other symptoms, but it also prevents such complications as fistula. If prompt admission to hospital is not possible, it is often wise to drain an accessible abscess by making a small incision under local anaesthesia. Definitive treatment should follow as soon as possible. The principle of definitive treatment is to uncap the abscess thoroughly, to open up all its extensions, and to ensure easy drainage by extending the wound into the lower part of the anal canal. Any superficial fistulae are laid open, but deep tracks may be better dealt with at a later operation if their relationship to the sphincters is in doubt. The excised tissue should always be submitted to histological examination. The most careful post-operative supervision of these cases is necessary to ensure sound healing without pocketing of pus and to exclude any overlooked fistulae.

attempt to secure some palliation, but the results are usually not impressive.

In those cases with poor sphincter tone treatment is much more of a problem, and rarely are the results wholly satisfactory. The condition of the sphincters may be improved by *muscle exercises* and by *surging faradism*. If these simple methods fail, it may be necessary to try to narrow the anal orifice. Unfortunately most of the operations devised



FIG. 81. Partial rectal prolapse. (Courtesy of W. B. Gabriel.)

to plect the sphincter or to place an effective muscle sling around the orifice are unsuccessful. It would be out of place to describe such procedures here, but two relatively simple operations which are sometimes of real value are: (1) Linear cauterisation of the anus; (2) Thiersch's operation.

*Linear cauterisation* is done with the object of promoting sphincter spasm. It is interesting that the linear burns so produced do not cause anything like the pain which would occur in people with normal sphincter tone. Many patients are much improved.

*Thiersch's operation* entails the insertion of a ligature of No. 20 s.w.g. silver wire into the perianal space. It is tied so as to leave sufficient room for passage of the index finger. Faecal impaction sometimes occurs following this operation; but, with the intelligent co-operation of the

In most cases, treatment on these lines is perfectly straightforward. Problems only arise with high and complicated fistulae and in those cases in which the surgeon, through failure to appreciate the anatomy of the lesion, does not lay it open properly.

*Superficial fistulae* are dealt with by passing a probe along the track and incising the overlying tissues. The wound is then made flat, and drainage assured by excising a sufficiency of surrounding skin. A flat eusol dressing is applied, and the most careful supervision is exercised until healing has occurred.

*Low anal fistulae* present no special problem. The subcutaneous and superficial parts of the external sphincter, as well as the lower part of the internal sphincter, may be divided. The wound is then properly saucerised to ensure drainage.

*High anal fistulae*, in which an internal track passes above the whole of the external sphincter, must be handled with caution. If there is reason to think that the patient may be rendered incontinent by laying the whole track open, then it is wise to pass a seton of stout silk through the track and only divide the superficial parts of the sphincter. Re-examination of the conscious patient will permit fuller assessment. A proctoscope is passed and, by observing the relative positions of the internal opening and the anorectal ring, it should then be possible to decide whether the rest of the track may be opened up. In practice, it is found that complete incontinence does not usually occur unless the puborectalis sling is divided. However, there are no puborectalis fibres anteriorly, and therefore complete division of the sphincter in that position is likely to be followed by some degree of incontinence. The large size of the wound may result in some stenosis as healing occurs, and in such cases it is wise to pass a rectal dilator daily. However, a word of warning should be given about the use of a dilator in cases of high fistula in which a large part or the whole of the external sphincter has been divided. In these circumstances there is some risk of the remaining sphincter fibres being stretched and so causing incontinence which would not otherwise occur.

### PROLAPSE OF THE RECTUM

There are two varieties of prolapse: *partial prolapse* which involves only the mucosa, and *complete prolapse* which includes all layers of the bowel, and always causes much stretching of the external sphincter.

**Partial Prolapse.** It is most important to assess the tone of the external sphincter, for if this is in good condition treatment is relatively straightforward. Rarely does more than an inch or so of mucosa protrude (Fig. 81), and this can be dealt with by *ligature and excision* in similar fashion to internal hæmorrhoids. Some degree of mucosal prolapse is not uncommon in elderly patients with hæmorrhoids. Sometimes operation seems inadvisable in very old and poor-risk subjects. In such people it is worth trying submucous injection of 11 per cent. phenol in oil in an

most common type of *post-irradiation* stricture follows the treatment of carcinoma of the cervix. This is usually preceded by a brisk irradiation proctitis. The diagnosis of the other types of stricture depends upon thorough examination and investigation, and an awareness of the possibilities.

**Treatment. Simple Dilatation.** This suffices for the treatment of most post-operative strictures especially if it is not delayed too long. Either a St. Mark's type dilator or uterine dilator can be used. In the more severe cases continued supervision over a period of years may be needed.

**Proctotomy.** A stricture confined to the lowest third of the rectum is dilated until a finger can be passed, and then incised in several places posteriorly. Dilatation is then effected up to the largest size Hegar, and is maintained for a few days with a rubber tube. Regular daily dilatation is then done whilst healing occurs. After this, dilatation is continued at increasing intervals until there is no further tendency for contraction to occur. Incision of the rectal wall as just described is referred to as *internal proctotomy*. A stricture of the anal orifice can be enlarged in similar fashion by *external proctotomy* in the posterior segment. The same diligent after-care is necessary.

**Colostomy.** With tubular strictures or active recurring inflammation a colostomy may be necessary. It should be of the dysfunctioning type to prevent continued contamination of the distal bowel.

**Excision of the Rectum.** This is indicated in those cases with continued chronic inflammation. Malignant disease may follow lymphogranuloma venereum or irradiation proctitis, and this is an added indication for excision in such cases.

**Proctitis.** From the practical point of view, the most important type of proctitis is that associated with ulcerative colitis. The rectal changes may occur much in advance of the colonic ones, and in the diffuse disease it is unusual for the rectum not to be involved. Continuing inflammation here following colectomy may give rise to persistent discharge and not infrequently to malignant change. For this reason it is usually wise to sacrifice the rectum when the colon is removed for ulcerative colitis. Proctitis may also be due to *specific bowel infections, trauma, irradiation or venereal disease*. Bacillary dysentery, amoebic infections and bilharziasis can be responsible. The last two are of importance, for a granulomatous mass may form which can be mistaken for carcinoma. Hard faeces, habitual purgation and the regular use of enemas may also produce inflammatory changes. Irradiation changes after treatment of carcinoma of the cervix have already been mentioned in connection with stricture. Gonorrhoea and lymphogranuloma may be complicated by proctitis, and the former is sometimes acquired by rectal coitus. In all cases of proctitis there is a variable amount of local discomfort, discharge and frequency of bowel action. Sometimes there are associated urinary symptoms.

patient and reasonable supervision, the method is of real value in elderly patients who have a completely patulous sphincter.

**Complete Prolapse.** Complete rectal prolapse is really one form of hernia of the pelvic floor. Sometimes a peritoneal pouch is included and the sphincters are badly stretched. Effective treatment of such a prolapse demands full appreciation of its anatomy. Two types of operation may have to be considered, and in some cases a combination of both procedures is needed.

*Rectosigmoidectomy* entails amputation of the prolapse; the peritoneum is opened anteriorly, and all redundant bowel is drawn down before excision. Vessels in the mesentery are carefully secured, and the cut end of the sigmoid colon is sutured to the anal mucosa about 1 inch from the anal margin. Sphincter exercises are started within a day or two of operation. The mortality of the operation is low, despite the great age of some of the subjects. The immediate results are good provided sphincter function is still present. Recurrence occurs in about two-thirds of cases within five years of operation.

*Repair of the Pelvic Floor.* Repair of the pelvic floor from the abdomen is indicated for some of those cases which recur after rectosigmoidectomy and for severe prolapse in fit subjects with irrecoverable sphincter tone. This operation can be exceptionally difficult in the narrow pelvis of the male and also in very fat women. The principle of current operations of this type (the redundant bowel having been excised) is to provide full support for the anterior rectal wall. This is done by placing silk sutures across the pelvic floor between the ischial spines, and secondly by careful approximation of the levator muscles in front of the rectum.

*Palliative Procedures.* In very poor risk patients, it is occasionally justifiable to attempt some relief by the Thiersch operation. Unfortunately faecal impaction is peculiarly liable to occur in these people because of the redundant bowel above the anus. If all else fails colostomy is to be considered as a better alternative to the misery of uncontrolled prolapse and incontinence.

### STRICTURE OF THE RECTUM

Most strictures are *post-operative* or *inflammatory* in origin. The malignant type does not require discussion here. The common causes of stricture are: (1) *Trauma*, due either to operative or accidental injury; (2) *Irradiation*, especially for carcinoma of the cervix; (3) *Chronic inflammation*. Ulcerative colitis, lymphogranuloma venereum and rare causes such as actinomycosis, Crohn's disease and bilharziasis.

*Post-operative stricture* is most likely to occur after haemorrhoidectomy, although, with modern technique, the occurrence is rare. It may also occur after operation for fistula or restorative resection of the rectum. Stenosis following the latter procedure is rarely severe. *Accidental injury* by strong chemicals, missiles and sharp objects may cause stricture. The

more complete assessment prior to operation. *It cannot be too strongly emphasised that the surgeon should never embark on excision of the rectum for a supposed carcinoma without preliminary biopsy.* This not only confirms the diagnosis but permits accurate assessment of the histological grade of malignancy in about 80 per cent. of cases.

**Lymphatic Spread.** The spread of rectal carcinoma has been investigated in some detail. Dr. Cutlibert Dukes divides these tumours into three groups. In Group A the growth is limited to the wall of the bowel. In Group B there is spread to the extrarectal tissues but no involvement of lymphatic glands, and in Group C the lymphatic glands are involved. The last group is subdivided into C1 cases in which the highest glands are not involved, and C2 cases in which glands are involved up to the site of



FIG. 82. Relationship of Dukes' grading to prognosis. Group A, growth confined to wall of rectum (15 per cent. of cases). Group B, extension to extrarectal tissues (35 per cent.). Group C, deposits in regional lymph nodes (50 per cent.). The respective five-year survival rates for these three groups are approximately 85 per cent., 64 per cent., and 33 per cent.

ligature of the main vessels. The incidence of the three main groups and their relationship to prognosis are illustrated in Fig. 82. The glands are involved in some 50 per cent. of cases, and rather more than a quarter of these are C2 cases.

**Venous Spread.** Careful examination of the veins in operation specimens reveals that they are involved by growth in about 20 per cent. of cases. Venous invasion is related to the malignancy of the tumour, being present in about 5 per cent. of those of the lowest grade and in at least 30 per cent. of those of the highest grade of malignancy. In most cases vein involvement implies a poor prognosis but there are some notable exceptions. These remarks on lymphatic and venous spread are largely applicable to the colon as well, but the many different tumour sites and variation in the anatomy of the colon makes it more difficult to relate pathological findings to prognosis.

**Histology.** It is convenient to subdivide carcinoma of the rectum into three histological grades. Grade 1 includes well-differentiated tumours of low grade malignancy. Grade 2 includes those of average differentia-

*Treatment.* In those cases due to a specific infection this is appropriately treated. For the rest there are only simple general measures which include regular oral paraffin, warm olive oil enemas and iron to correct any associated anæmia.

*Pruritus Ani.* In most cases irritation and itching of the anal or perianal skin is a symptom of some rectal complaint such as piles, fissure, fistula or proctitis. *Lack of hygiene, excessive sweating and mucus discharge from the rectum are factors of possible importance. Threadworms* may be responsible in adults as well as children. *Allergy to food and drugs* is operative in some. *Skin disease*, such as tinea of the natal cleft, psoriasis or seborrhoeic dermatitis constitute a further group. *Dermatitis medicamentosa*, particularly that due to the prolonged use of anæsthetic or antibiotic ointments, should not be forgotten. Finally, there is a group for which no cause can be found, and for whose complaints ingenious psychiatrists have devised many fascinating explanations. It is not to be denied that psychogenic causes are operative in some cases. Although it may be serious to overlook possible psychogenic causes, the doctor must beware of getting his patient embroiled in psychotherapy when some simple local cause has been overlooked. From the above list of causes of pruritus, it is self-evident that every case needs most careful local examination of the rectum and anus; and that in many, a detailed history and general examination are called for.

*Treatment.* Removal of the causative local condition is all that is necessary in many cases. Apart from this, local treatment includes careful attention to hygiene of the part. Much good is done by careful daily washing and drying of the skin. The patient should be enjoined not to scratch, but to pinch or press the skin if intolerable itching cannot be resisted. A valuable lotion for local application is that prescribed at St. Mark's hospital. The composition is as follows: Phenol 15 grains; Zinc oxide 30 grains; Prepared calamine 15 grains; Glycerine 30 minims; Spirit. rect. 30 minims; Aq. rosæ 1 drachm; Milk of magnesia to 1 ounce. This lotion can be dabbed on as often as necessary and usually proves very soothing. Such simple measures suffice for the majority of cases. In women there is occasionally associated vaginal discharge or *trichomonas* infection for which gynaecological advice may be needed. *Threadworms* are treated with anthelmintics such as piperazine hydrate. The treatment of allergic and dermatological conditions is not discussed here. In cases of tinea it is essential to clear up any interdigital infection whilst the anus is treated.

### CARCINOMA OF THE RECTUM

*Pathology.* Certain general features of carcinoma of the rectum have already been discussed in this chapter. The macroscopic characteristics of growths in the colon and rectum are similar, but the ease with which the latter may be seen through a sigmoidoscope and biopsied permits their

difficult, but with perseverance it is possible to see the growth and do a biopsy.

*Barium enema* X-ray plays no part in the diagnosis of rectal cancer. Indeed, a negative examination may deceive the clinician who has not examined his patient fully. It is no discredit to the radiologist to say that growths in the rectum are easily missed on X-ray, for there are technical difficulties in this region. Furthermore, it is the clinician's job to feel and see tumours in this region. The safe rule is never to ask for a barium enema X-ray until full examination including sigmoidoscopy has been done.

**Differential Diagnosis.** In the majority of cases the nature of the lesion, once felt and seen, is not in doubt. Innocent tumours, such as *adenomas* and *papillomas*, must be differentiated, for, whereas with such lesions in the colon it is sensible to do a resection, in the rectum a more conservative course is advisable. Uncommon conditions such as *amœboma*, *Crohn's disease*, *lymphogranuloma*, *endometrioma* and *inflammatory granulomatous strictures* following submucous injection of phenol in oil must all be considered. *Sarcoma*, *carcinoid* tumour and *malignant melanoma* are all rare. The surgeon must bear in mind all these possibilities and the safest rule is for every lesion to be biopsied before operation.

**Treatment.** Radical operation is the treatment of choice in the majority of patients with carcinoma of the rectum. The most widely used procedure is full excision of the rectum and establishment of a permanent colostomy, but the following types of operation may have to be considered.

### 1. *Radical Operation*

- (a) Combined Resection
  - (i) Abdomino-perineal.
  - (ii) Perineo-abdominal.
  - (iii) Synchronous abdomino-perineal.
- (b) Restorative Resection
- (c) Anterior Resection

} for suitable tumours.

### 2. *Non-radical Operations*

- (a) Restorative Resection
- (b) Anterior Resection
- (c) Perineal Resection.

} when deliberately done as a "second best."

### 3. *Palliative Colostomy.*

In the majority of cases a radical resection can be performed in one stage, and only occasionally does the patient present with a degree of obstruction which necessitates preliminary colostomy. When the latter course has to be followed the colostomy tends to handicap the second operation, and therefore every effort should be made to avoid a two-stage procedure.

When the growth is situated in the middle or lowest third of the rectum only one of the combined methods ensures proper clearance of disease. Downward spread of growth in the bowel wall is rare and it



tion, and Grade II tumours are the least differentiated and most malignant. The histological grading is, as might be expected, related to the degree of spread. Thus, Dukes' Group A includes a higher proportion of Grade 1 tumours than does Group C. Furthermore, lymph node involvement is found in about a fifth of Grade 1, almost a half of Grade 2, and in four-fifths of Grade 3 cases. As with other tumours, the histological grading may vary in different parts; the edge of the growth is likely to yield the most accurate information.



FIG. 83. Carcinoma of middle third of the rectum. Note several sessile adenomas above and below the carcinoma. The tumour was readily palpable on digital examination.

**Clinical Features.** Annular tumours at the rectosigmoid junction behave in much the same way as those in the left colon. Bleeding occurs in some 75 per cent. of cases and is often associated with change in the bowel habit. Constipation, mucoid discharge, diarrhoea, or alternating constipation and diarrhoea are further common features. The symptoms depend to some extent on the type of tumour and its situation in the bowel. Annular growths at the rectosigmoid junction behave like those in the left colon and tend to cause obstructive symptoms. Sometimes there is narrowing of the stools. The middle third of the rectum is of larger calibre, and therefore even a proliferative growth may produce little obstruction. Such growths may cause tenesmus, feelings of incomplete evacuation and local discomfort. Pain is an unusual feature, although it may occur with advanced and fixed growths or in those with associated perirectal abscess.

**Digital examination** of the rectum is the most important part of the physical examination. In skilled hands, some 90 per cent. of growths can be felt, but the unpractised observer tends to miss many of them (Fig. 83). It is extremely important to do a most careful and systematic digital examination, and to identify the various anatomical landmarks. If this is done regularly the doctor will certainly achieve a very much higher standard of diagnosis than if his examination is perfunctory and unsystematic. In some cases it is of help if the patient strains down and if bimanual examination is done with the other hand on the lower abdomen.

**Inspection** of the interior of the rectum is the next step and it should never be omitted. A proctoscope only allows examination of the lowest third, and therefore a sigmoidoscope should always be used. Mucoid discharge or leakage of liquid faeces past the growth may make examination

of the terminal ileum by an ileal band. The latter tends to pull on the ileum when the peritoneum of the pelvic floor is repaired. *Infection* of the perineal wound is more likely to occur if there has been damage to the bowel during the operation. *Peritonitis* also may be due to bowel damage, and is more likely to occur following removal of locally advanced tumours. *Chest complications* such as pulmonary collapse, pneumonia or infarction may occur. Each of these possible complications is probably minimised by the practice of breathing and limb exercises. *Urinary retention* and *infection* are more common in the male. The use of an indwelling catheter for a few days after combined resection ensures proper emptying of the bladder and thereby reduces the possibility of cystitis. *Impotence* occurs in the majority of men following combined resections due to unavoidable damage to the *nervi erigentes*.

**Mortality of Operation.** There is wide variation in the mortality rate according to the circumstances of the patients operated upon. The risk is about twice as high in men as in women over the age of sixty. The mortality rate of combined resections is seldom below 5 per cent. even in the very best hands, and in the average general hospital, it is not uncommon to find that the mortality is between 10 per cent. and 20 per cent.

**Other Methods of Treatment.** *Radiotherapy.* Patients too old and unfit for surgery, and those with advanced locally inoperable disease may benefit from radiotherapy. There are considerable advantages in using supervoltage X-rays in such cases, for the irradiation can be more effectively concentrated on the target. Pain is relieved, and discharge and bleeding controlled in about 70 per cent. of cases. Usually there is some persistence of the neoplasm, but in a few cases it disappears completely. The best results are obtained in those patients in whom the disease is considered resectable locally but deemed inoperable for general reasons. Radiotherapy is no easy alternative to surgery. The treatment may take six to eight weeks with some degree of constitutional and local reaction. The maximum irradiation to the tumour-bearing area is in the order of 6,000 r. Smaller doses are sometimes used in the treatment of recurrent disease, but only in patients who have previously been treated by surgery. Surgery after full radiotherapy is risky, for peritonitis may occur or wounds fail to heal properly.

*Diathermy.* On rare occasions a patient with a bulky inoperable tumour, which is causing offensive discharge and bleeding, may be helped by diathermy coagulation of the growth.

#### CARCINOMA OF THE ANAL CANAL

Squamous-cell carcinoma of the anus and anal canal constitutes about 3 per cent. of all growths in the rectum and anal canal. The overall incidence is about the same in the two sexes, and these growths tend to occur in older patients than does carcinoma of the rectum. Despite the similar overall incidence of disease in the two sexes it is interesting to note

is very uncommon for glands to be involved below the level of the tumour. Nevertheless, proper clearance of the perirectal structures, and division of the bowel a good 5 cm. below the growth is desirable in all cases. Most authorities agree that restorative resection is not compatible with proper clearance of tumours below the peritoneal reflection, whereas in the rectosigmoid region, either a restorative resection or anterior resection may be quite adequate.

Sometimes the surgeon is justified in doing a more limited operation as a calculated "second best" so far as ultimate cure is concerned, e.g. in a patient with a middle third growth who has refused colostomy and in whom restoration of continuity is possible. Similar considerations may apply in a poor-risk subject with a growth at the same level in whom anterior resection is felt to be safer than a combined resection. It is for these reasons that the restorative resection and anterior resection appear under the headings of both radical and non-radical procedures above. The perineal operation is not often practised to-day, although it still has some place as a frankly palliative procedure in selected poor-risk cases.

The expert rectal surgeon who constantly performs the combined operation does not often use a less extensive procedure; but, in the hands of those who deal with such patients less frequently, there is still some place for such operations.

*Inoperable Tumours.* If there are multiple secondary deposits in the liver it is usually best to leave the primary tumour undisturbed, however mobile it may be. In such cases a palliative colostomy is best. In this connection, it should be emphasised how important it is to see the liver and to be quite certain about the presence of deposits before any decision is made against radical operation.

In the presence of one or two hepatic secondary deposits, it is perfectly justifiable, and indeed desirable, to carry out a radical resection, for the patient may be fortunate enough to survive for several years. If the patient has what appears to be a solitary deposit in the liver, it may encourage the surgeon to do a partial hepatectomy at the time of the rectal resection, but most of these patients succumb from other deposits quite soon.

Local inoperability of a tumour may be due to invasion of surrounding organs. Anterior growths in men may prove inoperable due to involvement of the prostate. In women, however, the posterior vaginal wall and uterus can readily be removed with the rectum. Sometimes, there is widespread lateral or posterior extension which precludes radical clearance. Finally, it is generally unjustifiable to do a radical resection if a patient has peritoneal or omental deposits, or ascites.

*Complications of Operation.* *Paralytic ileus* is a common complication after combined resection and should be managed as outlined in Chap. 9. *Mechanical obstruction* may be caused by small gut adhesions to the pelvic floor or the region of the colostomy, and very rarely it is due to kinking

rectum is cleared from the urethral bulb and prostate in the male, or from the vagina in the female. With anterior growths in women, it is wise to remove part of the posterior vaginal wall with the rectum. By this stage the rectum can be drawn downwards a little, the peritoneum opened and the mesentery cleared behind. The vessels in the latter are ligated as high as possible before division. The bowel can now be drawn down and divided. The lower end of the proximal bowel is closed with continuous serosal and purse-string sutures, and this stump is included in the posterior end of the peritoneal suture as the pelvic floor is closed. The skin wound is then closed with adequate drainage, usually by a length of corrugated rubber.

**Combined Excision.** This may be done either by the abdominoperineal, the perineo-abdominal or the synchronous methods. Most surgeons working on their own prefer the A.P. to the P.A. excision as the latter necessitates turning the patient and opening the abdomen twice. However, there are some advantages in dissecting the perineum first, and the perineo-abdominal route is favoured by W. B. Gabriel who has an unrivalled personal experience of the technique. In Gabriel's method of perineo-abdominal excision, the abdomen is first explored and operability is assessed. The abdomen is then temporarily closed with a few through and through sutures. The patient is turned into the left lateral position and the perineal dissection is then carried out as described above. The mesentery and bowel are not divided from below, but the dissected specimen is pushed above the pelvic floor, the peritoneum is partly closed and the perineal skin is sutured. The patient is once again turned into the dorsal position, the abdomen reopened, and the operation completed; the inferior mesenteric trunk is ligated and divided just below the left colic branches, and the mesentery is divided. The lateral attachments of the rectum containing the middle rectal vessels have usually already been divided from below; if not, they are dealt with at this stage. The pelvic and iliac colon are suitably mobilised by dividing the parietal peritoneum lateral to the bowel, and the specimen is then brought out through an oblique muscle-cutting incision in the left iliac fossa and the proximal bowel divided to complete the resection. Before the colostomy is established the space lateral to the bowel is closed with a catgut suture, and the emerging colon is held up either by a glass rod through the mesentery or by suture of mucosa to skin. The actual specimen removed at this operation consists of some 24 to 30 inches of bowel, including the anal canal, rectum and pelvic colon together with the mesentery and peri-rectal tissues up to the left colic vessels (Fig. 84).

The *abdominoperineal operation* is more commonly used. After exploring the abdomen and assessing operability, an incision is made in the parietal peritoneum lateral to the iliac colon. The bowel is mobilised and the mesenteric resection is mapped out as already described. The stages in the operation are similar to those noted above but more or less in the

that, whereas carcinoma of the anus is much commoner in men; carcinoma of the anal canal is much commoner in women. Sometimes the disease complicates a long-standing fistula.

**Clinical Features.** The patient may complain of discharge, pruritus, local discomfort, or a lump. Usually there is a raised lesion with superficial ulceration, and the appearances may be like those of an epithelioma elsewhere. On palpation there is well marked induration of the tissues underlying the ulcer. A tumour within the anal canal may be difficult to differentiate from adenocarcinoma in the lowest third of the rectum, and the diagnosis may only be established by biopsy. Metastasis to the inguinal glands is not uncommon but, owing to the frequency of super-added sepsis, it is not at all easy to be sure whether enlarged glands are involved by growth.

**Differential Diagnosis.** An epithelioma in this region must be differentiated from *chronic fissure, simple papilloma, tuberculous ulcer, condyloma* and *amœbic ulceration*. When it occurs as a complication of fistula-in-ano, the diagnosis may be overlooked unless the excised tissue is subjected to careful pathological examination.

**Treatment.** Carcinoma of the anus of low grade malignancy and limited extent can be readily treated by local *diathermy excision*. The results of *radiotherapy* are, on the whole, disappointing, for the region is rather intolerant and therefore full dosage often cannot be given. Radium needling is usually preferable to X-ray therapy. Cases need to be carefully selected for radiotherapy; a preliminary biopsy is helpful in assessment. With more extensive growths of the anus, or those of the anal canal, colostomy and *perineal excision* of the rectum is best. If the growth has extended into the lowest third of the rectum, a *combined excision* is the treatment of choice.

*Block dissection* of the groins is only undertaken if there is clinical suggestion of involved glands. Usually it is best to wait until the primary growth has been removed so that the glands in the groin can be more accurately assessed in the absence of local sepsis. If at this stage there is no clinical evidence of involvement, the patient should be carefully reviewed at frequent intervals during the first year or so after removal of the primary disease.

#### OPERATIONS FOR RESECTION OF RECTUM

**Perineal Excision.** A pelvic colostomy may be established beforehand or at the same time as the operation. The patient may be operated on in the left lateral or lithotomy position. The anus is closed with two purse-string sutures of silk. An incision is made encircling the anus and extended back to one side of the coccyx. Removal of the latter may facilitate exposure, but is not essential in all cases. The dissection is firstly carried through the *retro-rectal fascia of Waldeyer*, and the levator ani muscle is divided close to the pelvic wall on each side. Anteriorly, the

continuity is not considered practicable or desirable, and when a combined operation is contraindicated. It is useful for some elderly and poor-risk patients. The procedure is much the same as for the restorative operation, but the distal stump of bowel is closed and the peritoneal floor repaired. The proximal end of the bowel is brought out in the left iliac fossa to form an end colostomy after removing the growth.

The preparation of the bowel for all the operations described above is just the same as that for resection of growths in the colon. It is once again emphasised that mechanical cleansing of the bowel above the tumour is probably more important than the use of antibiotics and sulpha drugs. The latter are certainly no substitute for skilled nursing preparation.

### PILONIDAL SINUS

By V. A. J. SWAIN

A pilonidal sinus, which literally means a tract associated with a nest of hairs, is usually applied to such a condition situated externally in the sacrococcygeal region. It is distinct from the congenital dermal sinus which is present at birth, for the pilonidal sinus is acquired, and develops in early adult life. Hirsute males and less commonly females are liable

to develop the condition. During World War II there was a high incidence of pilonidal sinus infections in military personnel who travelled on army vehicles. This so-called "*jeep disease*" was probably precipitated by the repeated jolting (*vide infra*). A pilonidal sinus presents as a small opening in the midline a few inches posterior to the anus. A few hairs may be seen protruding from the sinus. There is a single midline opening but often the sinus has multiple ramifications (Fig. 85); these lie a few millimetres laterally. These secondary tracts arise probably as a result of repeated infection. On passing a probe into the midline opening—the entry sinus—it enters a cavity extending almost down to the sacrum but has no communication with that bone, the spinal canal, the rectum, or

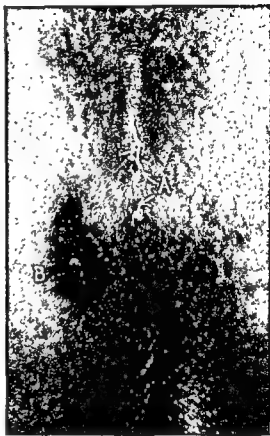


FIG. 85. Pilonidal sinus of the natal cleft showing two primary midline openings A, and a lateral secondary sinus opening B.

reverse order. After dividing the bowel and establishing the colostomy, the specimen is pushed below the pelvic floor and the peritoneum is closed. The perineal dissection is then carried out and the operation completed.

In the *synchronous combined operation* the patient is placed in the lithotomy-Trendelenburg position and, after preliminary exploration of the abdomen, two teams work simultaneously from both ends. This method greatly facilitates the resection of large and difficult growths, but

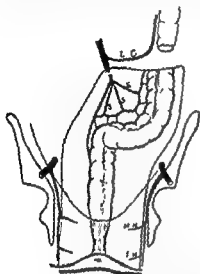


FIG. 84. Combined excision of rectum. To show extent of tissue removal in abdomino-perineal resection. L.C. Left colic. S.C. Sigmoid. M.H. middle and I.H. inferior hæmorrhoidal vessels.

it should be recognised that it is usually more shocking for the patient. Its expert performance requires good collaboration between the two teams. The position used in this operation is greatly favoured by many surgeons doing an A.P. excision on their own, for it is not necessary to change the patient's position during the operation.

**Restorative Resection.** Restoration of bowel continuity following resection of growths in the upper part of the rectum is perfectly feasible and satisfactory in many cases. Some have tried to extend the method to growths lower down but, although this procedure is quite practicable, there is undoubtedly a higher incidence of recurrent disease both in the bowel and the pelvis when restorative resection is used for growths in the middle

third of the rectum. The initial stages of the operation are much the same as the abdominal part of the A.P. resection. The rectum is most carefully cleansed from below by an assistant who patiently swabs out the lumen through an illuminated proctoscope. It is usual to clean the rectum out with swabs soaked in a 1/500 solution of perchloride of mercury with the object of killing any detached cancer cells which might become implanted at the suture line. The anastomosis needs to be most carefully done. About eight interrupted silk or linen sutures are used for the outer layer. These sutures are meticulously placed and laid before tying. After tying the outer posterior sutures, interrupted all-coats catgut sutures are inserted. The latter are begun at the back and completed in front after which the outer layer of anterior sutures is tied. The suture line may be placed above or below the pelvic peritoneum and, if below the peritoneum, it is possible to drain the pelvic cavity by passing a small corrugated drain into the lumen of the rectum through the posterior part of the suture line.

**Anterior Resection (Hartmann's Operation).** This method is applicable to growths in the uppermost third of the rectum when restoration of

plastic procedure rotating lateral flaps over the raw area, but this is rarely necessary and it is usually wiser to leave the wound open to heal by granulation, if there is any doubt about the chances of primary healing. An alternative method, which has not received wide recognition in Great Britain, is that advocated by Buie (1937) in which all of the tract is laid open and the overlying skin is trimmed; after the cavity has been mopped clean, the edge of the deep lining of granulation tissue is stitched to the adjacent skin; thus the pilonidal cavity is simply exteriorised. In this procedure the base of the cavity, which is left undisturbed, ultimately forms the healing surface of the wound, thereby achieving more rapid epithelialisation than by allowing the raw area, following excision, to heal. With all the above methods, recurrence is unlikely if all the tracts are dealt with and the adjacent skin is shaved and kept clean. Some surgeons advocate an epilation dose of irradiation. In the convalescent period exercises such as cycling and riding should be avoided, as they are liable to disrupt the wound.



the anus. The condition found on opening the pilonidal tract is well described by Hodges (1880) who first introduced the term "pilonidal sinus."

On exposing its interior by an incision a certain quantity of pus is evacuated, and a lock of loose hair is found occupying the space, more or less matted and curled and of varying size and amount. The hairs of which it is made up are always short, without bulbs and correspond in colour to those of the patient. The cavity containing them has no cyst or lining membrane, or other characteristic suggestive of a congenital dermoid wen; merely the granulating walls of an ordinary suppurating sinus, with no trace or suspicion of hairs growing from its surface, or of isolated spots of cuticle from which they might have been shed.

Microscopically, an epithelial lining is found in the superficial part of the sinus, whereas deep in the cavity, granulation tissue containing foreign body type giant cells is found: also detached bundles of hairs are seen within the purulent exudate (Fig. 80).

**Aetiology.** There are several observations which suggests that entry of hairs through the skin of the natal cleft is the cause of pilonidal sinuses: (a) It occurs in early adult life usually in the hirsute, when the hairs are coarse; (b) during walking, or riding in vehicles the suction movement produced between the folds of the buttocks attracts hairs to the natal

cleft; (c) the polarity of the hairs and the arrangement of their scales assist entry and penetration of the skin; (d) pilonidal tracts and abscesses occur at other sites where hair may be retained, such as in the interdigital clefts of barbers' hands, the axilla, and the umbilicus. Despite the above evidence many still hold that a pilonidal sinus is congenital.

**Treatment.** Sufferers from this condition usually first seek medical advice during the inflammatory stages. Drainage of the abscess and chemotherapy are then indicated. Reinfection, which is bound to occur, can be minimised by keeping the area clean and shaving the hairs around the natal cleft. After the acute infection has subsided, excision of the area followed by immediate suture cures the condition, providing primary healing is attained. An elliptical incision, including all sinus openings and the underlying cavity wall, is made down to the post-sacral fascia, and the edges are closed by deep mattress-sutures, followed by the application of a pressure dressing to prevent any dead space forming under the suture line, for such a space is liable to become infected. Some surgeons use "



FIG. 80. Coil of hairs from pilonidal sinus.

plastic procedure rotating lateral flaps over the raw area, but this is rarely necessary and it is usually wiser to leave the wound open to heal by granulation, if there is any doubt about the chances of primary healing. An alternative method, which has not received wide recognition in Great Britain, is that advocated by Buie (1937) in which all of the tract is laid open and the overlying skin is trimmed; after the cavity has been mopped clean, the edge of the deep lining of granulation tissue is stitched to the adjacent skin; thus the pilonidal cavity is simply exteriorised. In this procedure the base of the cavity, which is left undisturbed, ultimately forms the healing surface of the wound, thereby achieving more rapid epithelialisation than by allowing the raw area, following excision, to heal. With all the above methods, recurrence is unlikely if all the tracts are dealt with and the adjacent skin is shaved and kept clean. Some surgeons advocate an epilation dose of irradiation. In the convalescent period exercises such as cycling and riding should be avoided, as they are liable to disrupt the wound.

## CHAPTER 11

### AMŒBIASIS

by

LIEUT.-COLONEL P. ROSS WHEATLEY, D.S.O., R.A.M.C.

AMŒBIASIS is primarily a medical disease, but an important one to the surgeon. It may mimic many abdominal conditions and so lead to errors in diagnosis. Its unsuspected presence may cause disastrous complications after abdominal and anorectal operations. In its treatment surgical aid may be invoked to drain a liver abscess. Although the causal organism, *Entamœba histolytica*, has a world-wide distribution, the disease is essentially one of the tropics and subtropics, and when seen in Great Britain is usually in a patient who has been overseas. Infection is caused by ingestion of the cystic form of *E. histolytica* carried from human excreta. The amœbæ are liberated from the cysts in the intestine and invade and multiply in the mucosa of the large bowel, where, by secreting cytolytic ferments they cause necrosis and ulceration. Invasion of the veins brings them to the liver where they may flourish and destroy the tissues. Rarely they are carried to the lungs, brain and other organs through the systemic circulation. The important forms of amœbiasis are therefore intestinal and hepatic.

#### INTESTINAL AMŒBIASIS

**Pathology.** The cæcum and ascending colon are most commonly affected in this disease of the large bowel. The rectum and pelvic colon are also common sites. The lesion is one of necrosis with a mild reaction by dilatation of vessels, œdema and infiltration of a few leucocytes in the surrounding tissue. As seen through the sigmoidoscope the early lesion is a minute raised nodule surrounded by congested vessels. Pin-point ulcers form, and these may extend to an inch or more in diameter, but because of the undermining of the edges they often appear to be small. Their base is covered with slough and bleeds easily. The mucosa between the ulcers usually appears normal, but ulcers may communicate by sinuous tracts in the submucosa. Amœbæ may be found in scrapings from the ulcers. The wall of the colon is thickened by œdema, and peritoneal reaction may cause adhesions to the small bowel and other organs. Extension of the necrosis may cause severe hæmorrhage or acute perforation into the peritoneal cavity. Both of these calamities are rare, but anaemia from continued blood loss is common, and an intraperitoneal abscess limited by adhesions is not infrequently present. As a result of

secondary infection a granulomatous mass, the *amœboma* or amœbic granuloma, may form in the bowel wall or round the anus. This is hard, fixed and nodular and may be ulcerated, thus having all the clinical characteristics of a carcinoma. The healing of such a lesion can cause a stricture of the bowel. *Cutaneous Amœbiasis*.—The skin may be involved in a rapid painful ulcerative process around the anus, a colostomy, an appendicectomy wound or any sinus leading down to an amœbic abscess.

**Clinical Manifestations.** The various ways in which intestinal amœbiasis may present are classified as follows :

(a) *As Amœbic Dysentery*. The patient passes loose foul-smelling stools containing blood and mucus. Usually the onset is subacute, with no fever, some colicky pain and about a dozen stools a day, but it may take an acute form with very frequent watery stools, severe constitutional disturbance and sometimes massive hæmorrhage. The diarrhoea is the result of ulceration and irritation of the left half of the colon, and the rectum ; and it follows that, as these are not always involved, dysentery is not an essential part of amœbiasis. There may be no history of it to help in the diagnosis when later manifestations occur.

(b) *As a Vague Abdominal Disorder*. Discomfort and indefinite pain, anorexia, sometimes nausea and vomiting with or without diarrhoea, are accompanied by loss of weight, anæmia and general ill health. The tongue is furred, and there may be localised abdominal tenderness, particularly over a thickened and palpable cæcum or sigmoid colon. Many conditions must be considered in the differential diagnosis, especially appendicitis, diverticulitis, and carcinoma of the colon or stomach.

(c) *As a case of acute appendicitis* with colicky pain, nausea, constipation, slight fever, tenderness and rigidity in the right iliac fossa, sometimes a tender mass and leucocytosis. This is an amœbic infection of the appendix, spread from the cæcum, and there may be obstruction of the appendix resulting from it.

(d) *As a mass in the right iliac fossa* with the underlying pathology of a localised abscess surrounded by adhesions, or of an amœboma. The diagnosis must be made from appendix abscess, carcinoma of the cæcum, tuberculosis, regional ileitis and actinomycosis.

(e) *As an amœboma of the rectum or colon* with all the appearances and physical signs of a carcinoma. In addition to the tumour and the loss of weight, anæmia and debility simulate the effects of cancer. In the tropics a mass or ulcer in the rectum calls to mind an amœboma as the most likely diagnosis.

**Investigations.** *Examination of the stools* for amœbæ and cysts. This should be carried out after a saline purge and be repeated several times if at first negative. It is important that the stools be sent to the laboratory immediately they are passed and examined while still warm so that the motile amœbæ may be distinguished. *Sigmoidoscopy* may reveal the typical lesion, and scrapings from the ulcers, if examined immediately,

may reveal amœbæ. If a mass suspected of being an amœboma be seen, a biopsy is carried out (it must be remembered that cancer may co-exist with amœbiasis). *Therapeutic Test.*—Emetine given for five days will produce a definite response if the condition is of amœbic origin. This test is of great practical value.

**Treatment.** (1) *Medical.*—Details are given later under hepatic amœbiasis. (2) *Operative.*—Because the tissues in an amœbic lesion are friable and congested, operative procedures on them carry the grave risk of hæmorrhage and spread of infection. Abdominal operations may be followed by peritonitis or by the formation of a sinus. Rectal surgery in the presence of active infection is doomed to failure, leaving the patient often worse off than before, with abscesses and sinuses. From a sinus there may arise a spreading cutaneous amœbiasis. It is important, therefore, to exclude intestinal amœbiasis before operating on hæmorrhoids or other anorectal conditions in the tropics or in patients who have come from the tropics. To destroy the active amœbæ and to decrease the friability and congestion of the tissues, *emetine must be given for five days before any operation in the presence of amœbiasis*, except in an acute emergency when operation cannot be postponed; then emetine treatment should be begun without delay. *Laparotomy* may be indicated: (a) as a diagnostic measure to exclude the presence of a carcinoma or other operable lesion; (b) in the treatment of an acute emergency such as appendicitis or intestinal obstruction. If in operating for suspected appendicitis the cæcum be found to be inflamed and œdematous, the removal of the appendix is likely to be followed by leakage from the stump and peritonitis. Therefore, unless there is obvious danger of perforation, it is safer to leave the appendix undisturbed and close the abdomen.

### HEPATIC AMOEBIASIS

This complication of intestinal amœbiasis affects males ten times more frequently than females. Most cases occur between the ages of twenty and forty. It is very uncommon in children. Europeans are more subject to it than the natives of tropical countries.

**Pathology.** The amœbæ reach the liver by the portal vein, and there cause minute foci of necrosis surrounded by capillary congestion (*amœbic hepatitis*). Depending on the patient's resistance, there is either resolution or coalescence of foci to form an *abscess*. The abscess is usually solitary and situated in the right lobe, typically near the dome (Fig. 87). Multiple abscesses are the result of widespread infection in patients with low resistance, and usually terminate fatally. The solitary abscess may increase in size up to a capacity of several pints. The pus, which consists of blood and necrotic liver, is classically likened in colour and consistency to anchovy sauce. It is usually sterile, but may become secondarily infected. Amœbæ are rarely found in the pus, but can be demonstrated in the ragged wall of the abscess. Outside the necrotic wall is a zone of

connective tissue reaction, which in long-standing cases may become fibrotic. The abscess has a tendency to spread. The commonest direction is upwards through the diaphragm, but it may spread downwards to involve the abdominal viscera, or point forwards to the epigastrium or backwards to the loin. Adhesions usually obliterate the serous cavities in the path of the abscess; for example, the result of the upward spread from the right lobe is an abscess in the base of the right lung rather than an empyema. Rupture of an abscess into the pleural, peritoneal or pericardial cavity is a rare occurrence indicating a very active process.

**Clinical Picture.** Hepatitis usually presents within three months of the



FIG. 87. An amoebic abscess in the right lobe of the liver—the common site. The ragged wall and the zone of fibrotic reaction around it are shown. (From the Museum of the Royal College of Surgeons.)

intestinal infection, but it may be delayed even for many years. *There may be no history of diarrhoea.* The picture is variable and often very indefinite and confusing. The onset is usually insidious, but it may occur as an acute fever. The main symptoms are weakness, anorexia, loss of weight, sweating, discomfort in the region of the liver, often caused by lying on the affected side, and pain in the right shoulder. Pyrexia, usually of low grade at first, and a palpable tender liver margin are fairly constant signs. Pain may be elicited when the liver is jarred. When an abscess is present the symptoms are more severe; there is profuse sweating particularly at night, a hectic swinging temperature, and often an irritating non-productive cough. Localised tenderness, sometimes with oedema, may be present over the right lower ribs or in the epigastrium, according to the site of the abscess. There may be signs of consolidation at the base of the right lung.

**Investigations.** *Blood examination.*—There is a moderate leucocytosis (8,000 to 25,000) with no marked increase in the polymorphs or eosino-

phils. The count tends to drop in chronic cases. A higher count indicates secondary infection. There is a secondary anæmia with the hæmoglobin as low as 60 per cent. in chronic cases. The erythrocyte sedimentation rate is increased. *Examination of the stools* should be made repeatedly for amœbæ and cysts, but they may not be found. *Sigmoidoscopy* may reveal ulceration of the mucosa particularly along the mucosal ridges of the valves of Houston. A positive finding may clinch the diagnosis, but a negative finding is of no significance as only a small part of the large bowel can be examined by this means. *Radiology*.—In hepatitis, movement of the right side of the diaphragm is usually diminished. Later it becomes elevated and fixed. A localised bulging of the diaphragm is indicative of an abscess (Fig. 88). Very often signs of reaction in the chest may be seen as a shadow at the base of the right lung or a small pleural effusion. Radiological examination of the intestinal, biliary and renal tracts may have to be carried out to exclude other conditions.

**Differential Diagnosis.** Hepatic amœbiasis may be confused with other causes of fever, particularly malaria (they frequently coexist) or tuberculosis. Infective hepatitis, neoplasm of the liver, Hodgkin's disease, hydatid cysts and kala-azar have to be considered as causes of liver enlargement. Appendicitis, cholecystitis, subphrenic abscess, portal pylephlebitis, pneumonia and empyema are all included in the differential diagnosis. The typical features of the disease are the fever, the large tender liver, the fixed and raised diaphragm and the blood picture. The therapeutic test usually clinches the diagnosis, for there is an abatement of symptoms after four or five days of emetine or chloroquine in cases of hepatic amœbiasis.

**Complications.** *Thoracic spread* is common and pleuritic pains and signs of consolidation at the right base usually accompany the typical large abscess in the right lobe of the liver. A *lung abscess* may form from direct spread, and either burst into the bronchus and be expectorated, or become chronic and encapsulated. *Pleural effusion* and *empyema* may occur with or without marked lung involvement. *Pericardial effusion and abscess* may occur particularly in a left lobe abscess. *Abdominal spread* can cause adhesions or localised intraperitoneal abscess or involve any of the viscera. Such an abscess tends to point to the surface, typically as a fluctuating swelling in the epigastrium. *Rupture into the peritoneal cavity* is uncommon. It causes severe shock and may be fatal. *Secondary infection* with pyogenic organisms—a serious complication—may occur in a closed abscess, and it is to be expected when a sinus has formed. Such an infection may give rise to hectic fever and severe constitutional disturbance in an already debilitated patient, and accelerate the progress of the disease. *Cutaneous amœbiasis* may occur around a sinus and spread over a large area of skin as a painful sloughing ulcer. *Cerebral abscess* is a rare complication.

**Medical Treatment.** Early recognition and treatment of this debilitating



FIG. 88. Radiographs showing upward bulging of the diaphragm in liver abscesses.  
 (From the Tropical Medicine Department of the Royal Army Medical College.)



disease is very important. In the stage of hepatitis or of small abscess formation, drugs alone will suffice, but when an abscess is large, evacuation of the pus by aspiration or open drainage is necessary. Relapses are liable to occur in long standing cases. The general condition of the patient must be carefully assessed and appropriate measures taken to correct deficiencies of fluid, nutrition or hæmoglobin. Blood transfusions are usually necessary if the patient has reached the stage requiring open operation.

*Emetine Hydrochloride.* The dose is 65 mg. given by deep subcutaneous injection daily for ten days. Emetine is a powerful amœbicide which usually produces a response within five days. It is a protoplasmic poison with a selective action on muscle. Thus it may cause profound muscular weakness and cardiac depression, and must only be given to patients strictly confined to bed. *Chloroquine.*—A loading dose of 0.6 gm.\* (base) daily by mouth for two days is followed by 0.3 gm. daily for twenty-six days. This drug becomes concentrated in the liver, and in that organ it is at least as effective as emetine as an amœbicide, but it has no effect in the bowel. It is much less toxic than emetine and can be safely used when cardiovascular disease is present. Insomnia, blurring of vision, fall in blood pressure and occasional mental confusion are side effects. With either of these drugs relapses may occur, and therefore when treating a relapse it is advisable to give a course of both drugs, either simultaneously or consecutively. Neither has any effect on the cystic form of parasite which must always be assumed to be present in the bowel, even if such forms cannot be demonstrated. The treatment must therefore be continued with other drugs, of which a suitable combination is *emetine bismuth iodide*, 0.2 gm. daily in capsules by mouth for twelve days together, with *diodoquin*, 0.6 gm. thrice daily in tablets for twenty days.

*Aspiration.* Needling may be a diagnostic or a therapeutic measure in liver abscess. The dangers of the procedure are hæmorrhage from a friable liver and extension of the infection, particularly to the serous cavities. To counteract these complications, emetine or chloroquine must always be given for three to five days beforehand. *Aspiration should be carried out in an operating theatre with aseptic precautions.* Either light general anæsthesia or local anæsthesia may be used. The best needle to use is one of 2 mm. bore, 10 cm. long and with a short bevel point. It is attached to a 20 ml. syringe. The site chosen is the point of maximum tenderness or swelling, but it is inadvisable to aspirate through the anterior abdominal wall for fear of injuring viscera and spreading infection into the peritoneal cavity. If there is no clinical indication of the site of the abscess, the eighth, ninth or tenth intercostal space in the mid-axillary line is selected. It is not always easy to locate the abscess and, if no pus be found at the first attempt, the needle must be completely

\* Chloroquine is prescribed as a salt, usually the sulphate, but the dosage is calculated by the amount of the base.

withdrawn before reinsertion to avoid damage to the liver and dangerous hæmorrhage. When pus is found, air replacement after aspiration will allow radiological demonstration of the extent and shape of the cavity. Care must be taken not to create a positive pressure in the abscess cavity, otherwise an air embolism may result. A specimen of pus should be examined for pus cells and bacteria—evidence of secondary infection. If this should be suspected or proven, appropriate antibiotics should be administered systemically and a million units of penicillin and 1 gm. of streptomycin in 5 ml. of sterile water be instilled into the abscess cavity. The majority of amoebic abscesses can be cured by a combination of aspiration and drugs, but it may be necessary to repeat the aspiration if signs and symptoms persist.

**Operative Treatment.** Before the advent of antibiotics the risk of secondary infection of an abscess was a deterrent to operation, but now there should be no hesitation in applying surgery when it is indicated. A preliminary course of emetine or chloroquine must of course be given to lessen the dangers of hæmorrhage and spread of amoebic infection during the operation. *The indications for open operation are:* (a) Failure of the patient to respond to conservative treatment, because of the size or the position of the abscess, or because of secondary infection; (b) failure to locate the abscess by needling; (c) for an abscess in the left lobe of the liver, where needling is difficult and dangerous; (d) when the abscess is pointing to the surface; (e) when a lung abscess is present. The aim of the operation in this case is to drain the abscess in the liver with which the lung abscess communicates, and to do it before the latter bursts into a bronchus. (f) when a localised intraperitoneal abscess is suspected. (g) for rupture of an abscess into the peritoneal cavity. (h) for chronic empyema. *Technique.*—In an anterior approach the incision is made parallel to the costal margin; the peritoneum is opened, and the intestines are packed off. The liver can then be explored with a needle under direct vision. The abscess should be drained through a catheter around which the tissues are closed. The catheter is then connected to a closed drainage system. When an abscess is pointing to the anterior abdominal wall, it should be carefully opened avoiding the general peritoneal cavity. When an abscess is known to be in the posterior part of the right lobe it may be approached extraperitoneally through the bed of the twelfth rib. An alternative approach to a very large abscess pushing up the diaphragm is by the transthoracic route, with resection of part of the ninth and tenth ribs. The costophrenic pleural angle is usually obliterated in these cases, but if there is any doubt the operation should be performed in two stages with a pack left in the wound for forty-eight hours to create pleural adhesions before opening the abscess. *Convalescence* of several months, preferably taken in a temperate climate, is required when treatment has been completed.

**Prognosis.** In non-suppurative hepatitis, and in a solitary abscess

treated early and adequately, the prognosis is good and there should be no mortality. When, however, there has been delay in giving the drugs or in evacuating an abscess, chronic ill health may result, relapses occur and complications arise. There is particularly liability to neurosis (bowel and liver consciousness). The prognosis must be guarded in the presence of complications, e.g. extra hepatic extension of the disease or secondary infection of an abscess. Thus the mortality in pulmonary abscess even with emetine is 8 per cent. Without drugs it is over 50 per cent. Fulminating cases with multiple abscesses have nearly 100 per cent. mortality.

## CHAPTER 12

### ABDOMINAL INCISIONS

THE choice of a suitable incision, and its proper closure, are fundamental to the satisfactory execution of any abdominal operation. Many operations can be carried out equally well through a variety of incisions, and clearly there is room for differences of opinion and practice. Sometimes surgeons are unduly dogmatic and prejudiced about their preferred incisions. It is usually more agreeable to hear a surgeon say : " I use this incision because I like it," rather than to be treated to a theoretical diatribe of doubtful validity about an incision he *does not* favour.

The criteria of a good abdominal incision are that it should be : (1) Well placed for the procedure planned ; (2) of adequate length, and capable of reasonable extension should this be necessary ; (3) suitably disposed to avoid unnecessary damage to the abdominal wall ; (4) soundly closed to minimise the possibility of dehiscence or subsequent herniation, and (5) when practicable, so disposed as to ensure a discreet and cosmetic scar.

Vertical incisions are justifiably popular for many abdominal operations. They are suitable for such procedures as gastrectomy, colectomy and hysterectomy ; they allow wide access to the upper or lower abdomen or, if necessary, to both areas, and they certainly satisfy the first three criteria set out above. The strength of closure possible with a vertical wound is dependent on the quality of the patient's tissues. As for cosmetic appearance, although many of these incisions give a minimum of scarring, it is true that vertical wounds generally tend to heal with a rather more prominent scar than a transverse incision or one in a skin crease. Although it is sensible and desirable to leave the patient with as good an abdominal scar as possible, cosmetic considerations must never be allowed to overshadow those fundamental requirements of a well-placed incision of adequate size.

The remarkable relaxation and excellent operating conditions afforded by modern anæsthetic techniques ensure that surgeons can work almost equally well through a large variety of incisions. Closure of the incision (at one time not infrequently a battle of strength and wits) has now been made much easier. Indeed, one surgeon with a nice touch of humour has been heard to remark : " Closure of the abdomen, once so troublesome that I invariably left it to my assistant, is now so easy that I always do it myself " Notwithstanding this remark, it should be emphasised that adequate experience, together with care and precision in suturing, is very important in sound closure. The advantages of some incisions may be more evident post-operatively than during the operation itself. Thus,

there can be little question that coughing, straining and vomiting after operation are more liable to disrupt a vertical than a transverse incision. This is especially true of the upper abdomen where contraction of the powerful transversus abdominis muscle exerts a powerful disrupting force. The action of the transversus muscle can be well seen in a poorly relaxed patient when attempts are made to appose the edges of a vertical incision.

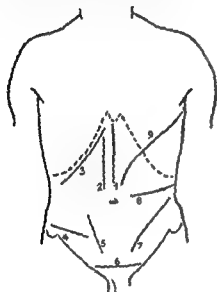


FIG. 89. The common abdominal incisions. (1) Midline. (2) Paramedian. (3) Subcostal. (4) Grid-iron. (5) Pararectal. (6) Transverse suprapubic. (7) Oblique muscle-cutting. (8) Transverse lumbar. (9) Abdomino-thoracic.

The transversus muscle fibres, acting in rather than across the line of the wound, do not exert such a potentially disruptive effect on a transverse incision. A second point of importance in securing sound closure of some wounds is that the fleshy fibres of the transversus can sometimes be sutured in one strong layer with the peritoneum. This last factor probably explains why disruption of a subcostal incision is so much less common than of a vertical one.

The action of the transverse abdominal muscles may account for the greater liability to disruption of upper as opposed to lower abdominal vertical incisions. In the upper abdomen, the muscular fibres of the transversus extend close to the midline and can be seen in the posterior rectus sheath in a paramedian incision. In the lower abdomen the transversus muscle fibres do not extend behind the rectus and, despite the frequently thin state of

the posterior rectus sheath, especially below the linea semicircularis, disruption is less common.

### INDIVIDUAL INCISIONS

(Fig. 89)

- (1) *Vertical*. These may be upper or lower abdominal or partly both.
  - (a) Midline ; (b) paramedian, either muscle-reflecting or muscle-cutting ;
  - (c) pararectal.
- (2) *Transverse*. Upper or lower abdominal ; unilateral or bilateral.
- (3) *Oblique*. (a) Subcostal ; (b) muscle-cutting ; (c) muscle-splitting.
- (4) *Miscellaneous*. These include odd shapes and combinations of other incisions.

**Midline Incision.** This has the virtues of simplicity, rapid execution, relatively little bleeding, good access for many procedures, and easy closure. In the upper abdomen it is widely used for operations on the

stomach and duodenum, and in the lower abdomen for gynaecological and bladder procedures. Some surgeons have a strong prejudice against this incision, and tend to favour the paramedian incision in the belief that it can be closed more soundly.

**Paramedian (Muscle-reflecting) Incision.** The paramedian incision is made about an inch from the midline; the anterior rectus sheath is incised in the same line. The rectus muscle is detached from the medial edge of its sheath at the level of the tendinous intersections and then displaced laterally. The posterior rectus sheath and peritoneum are opened in the line of the skin incision. When this incision is being closed, the layers are carefully sutured; the rectus muscle is lightly tagged to the medial edge of its sheath. It is not very profitable to debate the relative merits of paramedian and midline incisions. Some patients have poor rectus sheath tissue and a strong linea alba, whereas in others the reverse is true. Many surgeons would agree that there is little to choose between the two incisions.

**Paramedian (Muscle-cutting) Incision.** This is very similar to the above; but, instead of reflecting the rectus muscle, the incision is carried straight through it. The cut muscle tends to bleed a little but this is readily controlled. Devotees of this particular exposure feel that it obviates the need to retract the rectus in its sheath. Whereas the muscle-reflecting paramedian incision is used both in the upper and lower abdomen, the transrectal incision is confined usually to the upper part. Although those muscle fibres medial to the cut in the rectus are deprived of their nerve supply no practical disadvantage seems to accrue therefrom.

**Pararectal Incision.** In the lower abdomen some surgeons favour the pararectal (Battle or Bowlby) incision for such operations as appendicectomy. It is placed about an inch medial to the outer edge of the rectus, and, after the sheath is entered, the muscle is displaced medially, and care taken to avoid damage to the intercostal nerves. This incision does not enjoy wide popularity, but a lower pararectal exposure (McEvedy's incision) is useful for the repair of femoral hernia, especially in those cases complicated by obstruction.

**Transverse Incisions.** Transverse incisions can be used in the upper abdomen for operations on the biliary tract, pancreas and stomach. The exposure for gastrectomy is not always satisfactory, and many surgeons are understandably reluctant to use such an incision for this operation. Those patients with wide subcostal angles are especially suited to the upper transverse incision. Lower down and laterally, transverse incisions can be used for operations on the colon and sympathetic chain; Such incisions may be confined to the oblique muscles or extended into the rectus sheath. On those occasions when a transverse upper abdominal incision extends from one costal margin to the opposite side, meticulously careful suture is important, and the closure may take a good deal longer than that of a vertical wound.

**Subcostal (Kocher) Incision.** This is very widely used for operations on the gall bladder and bile ducts. Although two, or even three, intercostal nerves are commonly damaged by the incision, it almost invariably leaves an exceptionally sound scar and a good rectus muscle, and it rarely bursts. The rarity of dehiscence is probably due to the strength of closure afforded by suturing the peritoneum in one layer with the transversus abdominis.

### CLOSURE OF ABDOMINAL INCISIONS

The individual layers of the abdominal wall should be sutured with care and precision. The details of technique are best learnt in the operation room; only the more important points are referred to here.

**The Peritoneum.** The majority of surgeons favour a continuous chromicised catgut suture for the peritoneum. This may be inserted either as a simple running stitch or as a continuous longitudinal mattress suture which everts the edges of the peritoneum. It is vitally important to ensure that no chinks are left into which omentum or other viscera may protrude. Owing to the possible insecurity of a single continuous suture many operators interrupt the peritoneal stitch at intervals, or insert additional interrupted sutures. This certainly makes for greater strength and, in the event of the continuous suture breaking in the early post-operative period, it makes the peritoneal layer less likely to separate. The peritoneum is sometimes extremely thin, and, despite good relaxation, there may be a tendency for it to split and tear. In transverse and subcostal incisions, as already noted, the transversus muscle can be included in the peritoneal suture to ensure sounder closure. On those occasions when a drain is brought out through one end of the main incision it is wise to insert a single interrupted stitch next to the drain, and to stop the continuous suture just short of this. This last step ensures that when the drain is withdrawn it will not damage the continuous suture, thus jeopardising the integrity of the peritoneal layer.

**The Muscle Layers.** The muscles are closed individually, either with continuous or interrupted sutures. Whereas some surgeons use catgut throughout, there are many who prefer non-absorbable sutures of linen, silk, nylon or metal thread. Rather than discuss the relative merits of these different materials here it will suffice to say that, in the event of infection, monofilament materials such as single strand nylon or stainless steel wire generally cause less trouble than multifilament sutures such as linen, silk or plaited steel and tantalum wire. In practice, the choice of materials is very much a matter for individual preference.

**The Skin.** The skin is commonly closed with interrupted sutures of serum-proof silk or nylon. Some prefer to use metal clips. Whatever method is used the skin should be coapted neatly and carefully, not simply because it is the only part of the surgeon's handiwork visible to the patient, but also because this ensures sounder and more rapid healing.

## BURST ABDOMEN

An occasional somewhat dramatic complication of laparotomy wounds is complete dehiscence which occurs with variable and unpredictable frequency. Broadly speaking, disruption may occur in two sets of circumstances. Firstly, in a patient who is making relatively uneventful progress but who may have had a period of post-operative distension or cough. Secondly, it may be due to some major intra-abdominal mischief, such as leakage of digestive juices from the gut. The former type generally calls for no more than prompt resuture, but the second type is symptomatic of some underlying disaster which may also require attention before the abdomen is closed again.

In the majority of cases of burst abdomen, one or, more often, several of the following factors are responsible : (1) Post-operative gut distension, vomiting, persistent cough or retention of urine ; (2) poor abdominal tissues due to excessive obesity or previous operative damage ; (3) defective or badly inserted sutures, especially in the peritoneal and extra-peritoneal layers. Tightly inserted sutures may cut through the tissues ; (4) some intraperitoneal complication such as peritonitis or leakage from the gut.

However strong the suture materials used, and however carefully the repair is done, any abdominal wound may disrupt as a result of gross distension. Apart from this state of affairs, if a small portion of the peritoneal suture line gives way it is common for a piece of omentum or gut to become adherent to the defect and to form a kind of battering ram before which the rest of the wound gives way. In the early stages, before a burst occurs, it is common to see a little blood-stained fluid exude from the wound. This particular feature may precede disruption of the wound by several days. When such a discharge is observed, it is wise to ask the patient whether he has felt anything give way. A leading question of this kind frequently elicits the reply that a distinct snap has been felt in the wound a day or two earlier.

In the absence of distension and other traumatic factors, slow healing may be the main reason for wound dehiscence. Malnutrition and shortage of vitamin C in the diet leads to defective collagen formation. Instead of the wound edges being rapidly knit together by strong young fibrous tissue, there may be lax and ill-formed collagen which fails to mature normally. Furthermore, in obese patients, there is frequently a good deal of fatty infiltration of the muscles and aponeurosis of the abdominal wall making it softer and more friable. However strong the suture materials used, nothing will hold a poor abdominal wall really securely ; indeed, materials such as steel and tantalum wire may, by virtue of their very strength and sharpness, cut through the abdominal tissues like soft cheese.

On occasions, the peritoneal suture may snap or be torn out during a



bout of coughing at the end of the operation, and, unknown to the surgeon, the patient returns to bed with only the superficial layers of his wound intact. The skin may manage to heal soundly leaving an under-lying defect with consequent incisional hernia. In such cases, it is often best for a complete burst to occur so that the wound can be resutured there and then; the patient is thus discharged with a sound belly. To hold the skin sutures in position confers a doubtful benefit on the patient if he is left with an incisional hernia the repair of which may be quite difficult at a later operation.

*Resuture of a Burst Abdomen.* Owing to the "mushy" state of the abdominal tissues, it is usually impracticable to attempt to repair the individual layers. In most cases a series of strong non-absorbable sutures is inserted through all layers and left untied. A further layer of deep sutures can then be inserted, and these are only tied after all sutures have been placed. A few loose sutures are inserted in the skin and the initial through-and-through sutures are then tied over a broad gauze roll which largely prevents these stitches from cutting into the abdominal skin.

The abdominal wound usually heals uneventfully after secondary suture, partly because the original causes of disruption are no longer present, and also because the healing processes are already stimulated and active.

## CHAPTER 13

# ABDOMINAL HERNIAS IN ADULTS

by

D. L. B. FARLEY

A **HERNIA** may be defined as the protrusion of a body structure through its containing walls. The unqualified term hernia conventionally refers to a hernia of the abdominal wall. Examples of hernia in other situations include herniation of the brain through the skull, the lung into the neck, and part of a muscle through its sheath. An *external abdominal hernia* is the protrusion of an intra-abdominal structure through the abdominal wall (Fig. 90). The most common types of external abdominal hernia are the inguinal, femoral and umbilical hernias. Over 80 per cent. of external hernias are of the inguinal variety. Although femoral hernia is more common in the female than in the male, even in the female inguinal hernia is rather more frequently seen. An *internal abdominal hernia* is the herniation of a viscus into a peritoneal recess or through an abnormal hiatus.

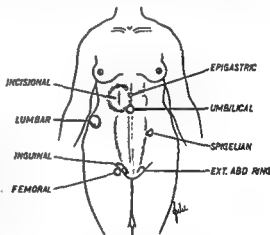


FIG. 90. The varieties of external abdominal hernia.

External hernias are common, whereas internal abdominal hernias are rare. Either external or internal hernias may lead to intestinal obstruction and impaired vascularity of the intestine. The serious nature of these complications accounts for the surgical importance of hernias.

### EXTERNAL ABDOMINAL HERNIA

**Aetiology.** Three factors predispose to the development of a hernia :

- (1) The presence of a congenital (or "preformed") sac of peritoneum ;
- (2) weakness of the abdominal wall ; and (3) increased abdominal pressure.

A *congenital sac of peritoneum* usually represents the abnormal persistence of a natural process, e.g. the *processus vaginalis* that accompanies the migrating testicle. Viscera easily slip into such a preformed sheath in spite of well-developed muscles. Congenital sacs are, however, often not

occupied by viscera for a varying period of time ; indeed a hernia may never occur despite the presence of a sac. *Weakness of the abdominal wall* may be congenital or acquired. Absence of the abdominal musculature on one or both sides is of great rarity, but absence of the insertion of the conjoint tendon (*falx inguinalis*) into the pubic spine may be noted in cases of direct inguinal hernia. *Poorness of physique* contributes to the development of hernia, as does obesity, since fat spreads muscle fibres and stretches hernial coverings. *Injury* is a factor in the formation of

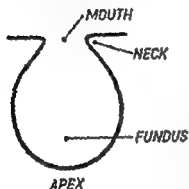


FIG. 91. The hernial sac.

hernias, although repeated minor strains are probably of more importance than a single major strain. Abdominal operations may be followed by a hernia, either as the result of stretching of the scar, damage to nerves, or infection of the wound. Injury to the ilio-hypogastric or the ilio-inguinal nerves during appendicectomy sometimes occurs, and may lead to the development of an inguinal hernia. *Increased intra-abdominal pressure* may arise from abdominal tumours, chronic cough, straining at stool in constipation, or dysuria in prostatic enlargement. A new

hernia may appear, or a long-standing one may become increased in size as the result of the increased abdominal pressure. A hernia may therefore be the first manifestation of intra-abdominal disease.

**Anatomy. The Sac.**—This consists of stretched peritoneum usually penetrating the whole of the abdominal wall (Fig. 91). Very rarely, the sac insinuates itself between muscular planes ; such a hernia is described as "*interstitial*." Still more rarely, the sac remains entirely deep to the muscles and is then termed "*properitoneal*." A sac may be *loculated*, especially in umbilical hernia, or very rarely *double*, especially in indirect inguinal hernia. Occasionally a hernia has *no sac*, e.g. the fatty hernia of the linea alba. *Hernia "en glissade"* ("sliding" hernia) refers to herniation of a viscus such as the cæcum or bladder which has an extra-peritoneal surface. In these circumstances the peritoneal sac does not invest the whole of the hernial contents (Fig. 92).

**The Hernial Contents.** Nearly every viscus has at some time become herniated, but the usual contents of a hernia are the omentum, small intestine and colon. The bladder, testes and ovaries are occasionally herniated. Hernial sacs may sometimes contain only fluid, although loose fibrinous or fatty bodies formed from detached fragments of omentum, or *tania epiploica*, are occasionally found. Hernial sacs are usually intermittently occupied ; congenital sacs may remain empty until adult life or may never be filled. *Richter's hernia* is a hernia of only part of the diameter of a loop of intestine (Fig. 93). It is most commonly seen in femoral hernias ; it is of the greatest importance, for necrosis of the

## ABDOMINAL HERNIAS IN ADULTS

intestinal wall may lead to perforation without any antecedent symptoms of intestinal obstruction. A Richter's hernia may be very small and in an obese patient can easily pass unrecognised. *Little's hernia* consists of a hernial sac containing a Meckel's diverticulum.

**The Coverings of a Hernia.** The covering layers of a hernia vary according to its situation; they are described with the different types of hernia. In long-standing hernias, the coverings become fused and cannot always be clearly identified at operation. Constriction occurs in inguinal hernias at the internal and external rings, and in femoral hernias at the lacunar (Gimbernat's) ligament and the saphenous opening in the thigh. In all hernias, fibrosis may be found at the neck; sometimes it is the sole cause of constriction.

**Clinical Features.** The patient usually notices a swelling. Hernias do not reveal themselves until complications develop. The swelling varies considerably. It often disappears on rest or recumbency, and returns after exertion or following coughing. At first a hernia is often painful as the result of the tissues, but a fully developed hernia is not usually painful. Complications ensue. When a hernia is "down" however, a

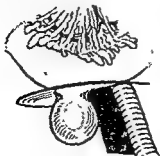


FIG. 93. Richter's hernia of the small intestine in a



FIG. 92. Hernia "en glissade." The peritoneum is shown by a line. The caecum and part of the ascending colon are herniated; the peritoneal sac is therefore incomplete.

colicky pain may be felt in the abdomen.  
**Examination.**—The patient should be examined when lying down. A swelling is usually present. If not, it may appear on coughing. In patients with long-standing hernias, the swelling is usually large. There is an expansile impulse on coughing. In uncomplicated external hernias, the impulse may be visible as well. When the patient lies down, the swelling disappears spontaneously; if not, gentle manipulation may induce the sac to empty. In this case, the hernia is termed "reducible."

intestine may be felt and possibly heard; omentum, as it slips under the fingers, feels doughy. Except in lax hernias reduction should never be attempted with the patient standing.

### COMPLICATIONS

**Irreducibility.** When a hernia is reduced its contents are returned to the abdomen, although the sac itself remains *in situ*. A hernia may be partly reducible, or irreducible, as a result of: (1) Fixation of the hernial contents by adhesions; (2) intestinal obstruction within the sac ("obstructed" hernia); (3) strangulation of the hernial contents. Other causes include the deposition of fat in the walls of herniated viscera; or, in long-standing massive hernias, reduction in the capacity of the abdominal cavity. In irreducibility the cough impulse does not necessarily disappear; in partly reducible hernias it can always be detected at the neck of the sac. A hernia may remain irreducible for a long time without causing any symptoms, beyond the presence of a swelling, if there is no disturbance of intestinal function, and no interference with the hernial blood supply, i.e. no strangulation. The commonest structure in such a hernia is omentum.

**Obstructed Hernia.** A hernia complicated by intestinal obstruction (without strangulation) is termed *obstructed*. This complication cannot be differentiated with certainty from strangulation before operation. The intestinal obstruction is most often due to constriction in the fundus of the sac or at its neck. Less commonly, obstruction results from kinking, torsion, or incarceration of bowel (*vide infra*). The patient may complain of discomfort in the hernia which is somewhat tender. Although usually irreducible, obstructed hernias may be partially reducible—especially if umbilical. Obstructed hernias usually soon become strangulated. The term *incarcerated* ("imprisoned") is applied to a hernia that is irreducible owing to the presence of bulky contents within herniated bowel. Incarceration is rare and usually affects the colon. The faecal mass can sometimes be moulded on palpation.

**Strangulated Hernia.** *Strangulation* implies impairment of the circulation of the hernial contents. It is most common in hernias with narrow necks and may occur the first time a hernia "comes down." Strangulation complicates approximately 10 per cent. of femoral hernias. Although sometimes the result of incarceration or torsion, strangulation usually results from an acute rise in intra-abdominal pressure, being initiated by the squeezing of an additional structure (usually a knuckle of bowel or omentum) into the sac. On the other hand, it is of interest to note that reduced external pressure, from flying at the high altitudes, may result in distension and strangulation of large unsupported hernias.

In strangulation, gangrene results from compression of the mesentery within the sac, or at its neck. Very rarely, bowel may become strangu-

lated within the abdominal cavity (Fig. 94). Rupture of intestine usually occurs at the neck of the sac, permitting leakage of infected material into the peritoneal cavity. If rupture of the bowel occurs within the hernial sac, there is a possibility of an external fecal fistula forming, with spontaneous relief of the intestinal obstruction; this is, however, a surgical curiosity, for unrelieved intestinal strangulation is almost invariably fatal. If omentum only is strangulated an abscess is likely to result, without the occurrence of peritonitis. Strangulation must be diagnosed early for, if surgery is delayed, resection of bowel may be necessary, and this considerably increases the mortality. Circular scarring of the bowel (stenosis) at the site of constriction occasionally follows strangulation and leads to the later development of intestinal obstruction. In strangulation there are pain and tenderness in the hernia, followed by vomiting and intestinal colic. Vomiting may at first be reflex, but it is later due to intestinal obstruction. Sometimes vomiting and abdominal pain are not early features and occasionally they are absent. In Richter's hernia (which is commonly femoral) there is no evidence of intestinal obstruction, although it is a dangerous hernia because of the likelihood of intestinal perforation. The patient with a strangulated hernia looks ill and may be shocked. The hernia is tense, tender, and partially or completely irreducible. The skin and subcutaneous tissues over the hernia may be cedematous. Confusion with inguinal adenitis is understandable, but fraught with grave danger to the patient. In the late stages of strangulated hernia acute peritonitis and paralytic ileus occur.

**Torsion.** Omentum is the commonest structure to undergo torsion. The hernia is painful and irreducible, and reflex vomiting is common.

**Intestinal Injury.** Herniated intestines may be injured directly as the result of a blow, or indirectly in "compression" injuries of the abdomen as the result of intestine being driven into an empty hernial sac.

**Infection.** Sepsis in a hernial sac is rare. Its development in strangulation has already been mentioned. However, it occasionally occurs in umbilical and femoral hernias in the absence of strangulation and leads to abscess formation. If the appendix is present in a hernia, the occurrence of acute appendicitis causes infection in the sac.

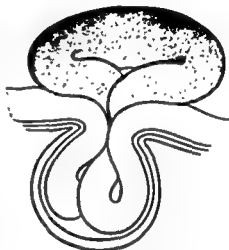


FIG. 94. *Maydl's hernia.* Intra-abdominal strangulation.

## TREATMENT OF COMPLICATIONS

"If there is anything wrong with a hernia it ought to be operated upon" (Grey Turner). Symptomless irreducible hernias should be operated on as soon as possible since there is always a risk of obstruction and strangulation. In obstructed or strangulated hernias, immediate surgical treatment is necessary, but gastric suction, replacement of fluids and correction of any electrolyte imbalance should first be undertaken.

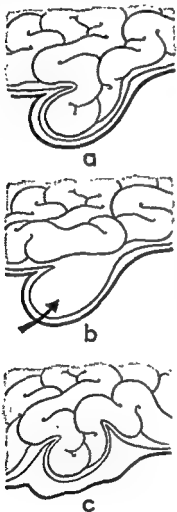


FIG. 95. (a) A loop of small intestine impacted in a hernial sac. (b) The intestine has been reduced and the sac is empty. (c) The hernial sac itself has been reduced and the intestine is still constricted (*reduction en masse*).

**Manipulative Reduction, or "Taxis."** Although taxis may be attempted in irreducible hernias, it should not be tried in strangulation. For taxis to be successful it must be performed with the utmost gentleness, and the patient's muscles must be relaxed. If the patient is unco-operative and the muscles taut the manœuvre is painful, and much harm may be done. In inguinal or femoral hernia, the patient should be recumbent, with the hip flexed and internally rotated. The key to starting reduction in an inguinal hernia is to press with the fingers from the lateral side of the

neck of the sac in the direction of the scrotum. This often "unlocks" the last-entering contents which are hitched on the external ring. Whilst this is done with a stroking movement, gentle pressure is produced within the sac by grasping it with the other hand. In a femoral hernia the pressure must be directed downwards at first, and then backwards. *The Dangers of Taxis.*—Damage to the hernial contents may result from the use of excessive force, or attempting reduction in unsuitable cases. Reduction "en masse" is rare; it consists of reduction of the hernial sac complete with its contents; thus the hernia appears to have been successfully reduced but symptoms persist (Fig. 95). Reduction of already perforated bowel, intestinal hæmorrhage, or delayed perforation, are also possible hazards. *Conservative Aids to Spontaneous Reduction.*—If taxis is contra-indicated, or fails, preparations are made for operation. During this time any necessary resuscitative measures are undertaken. In order to encourage spontaneous reduction of the hernia, the foot of the bed is raised, a cold compress is applied to the hernia, and an injection of morphia is given.

**Operation for Obstructed and Strangulated Hernias.** The aims are: (1) To relieve the constriction of viscera; (2) to remove devitalised tissues; and (3) to excise the hernial sac. Hernial repair may also be performed if the patient's condition is good. Adequate exposure of the neck of the sac must be achieved before release of the contents of the hernia is attempted. The wound should therefore be enlarged as required. The sac is opened and the accumulated serosanguinous fluid carefully mopped away. Great care is needed when dividing the constricting ring; with a finger in the sac as a guide this is often possible under direct vision. If the ring is very tight a round-ended tenotome may be used, and small nicks made until the constriction is released. The contents of the sac are then drawn down and bathed in warm saline. If their colour returns, and the peritoneal coat is shiny, the viscera may certainly be returned to the abdomen. If intestine remains of dusky hue, is non-contractile, and its peritoneal coat is ragged, resection is necessary; in inguinal and femoral hernias, an additional incision (paramedian) should be made if there is any difficulty in exposure. Small areas of intestinal damage, e.g. at the site of constriction, can be invaginated by a continuous serosal suture across the axis of the bowel. Devitalised omentum should be resected. The colon is only rarely suspect; either excision and double-barrelled colostomy, or simple exteriorisation through an incision in the flank should be undertaken in the first instance. Excision and anastomosis should not be done.

### INGUINAL HERNIA

**The Inguinal Canal.** In the adult the inguinal canal is 4 cm. long and runs from the internal to the external abdominal rings. Its course lies parallel to the inguinal ligament. It is occupied in the male by the



spermatic cord and in the female by the round ligament of the uterus. In both sexes the ilio-inguinal nerve and the genital branch of the genito-femoral nerve traverse the canal. The *internal abdominal ring* is situated in the transversalis fascia just above the inguinal (Poupart's) ligament. It lies half way between the anterior superior iliac spine and the symphysis pubis. It is supported above and to its lateral side by the lowest fibres of the internal oblique and transversus muscles. The deep inferior epigastric artery lies on its medial side. The *external abdominal ring* is a  $\Delta$ -shaped hiatus between the fibres of the external oblique muscle at the insertion of this muscle into the pubis. It lies just above and lateral to the crest of the pubic bone. It is larger in males owing to the size of the spermatic cord. The *floor of the canal* is the grooved inguinal ligament. The *roof* is made by the arching lower edge of the internal oblique and transversus muscles, which become tendinous medially, and unite to form the con-

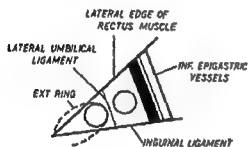


FIG. 96. *Hesselbach's Triangle*. Possible sites for the initial development of a *direct inguinal hernia* are shown by rings. The course of the more laterally placed hernia is oblique.

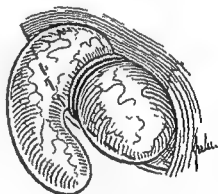


FIG. 97. A "saddle" hernia, i.e. combined direct and indirect sacs astride the deep epigastric vessels.

joint tendon (*falx inguinalis*). The *posterior wall* is formed by the transversalis fascia, together with the conjoint tendon in the medial third. The *anterior wall* is formed by the external oblique aponeurosis, together with the internal oblique muscle in the outer third. *Hesselbach's Triangle*.—The sides of the triangle are derived from the deep inferior epigastric artery, the inguinal ligament and the lateral border of the rectus abdominis muscle (Fig. 96). It is crossed by the obliterated umbilical artery (lateral umbilical ligament).

**Varieties of Inguinal Hernia.** An *indirect inguinal hernia* enters the inguinal canal through the internal abdominal ring, i.e. lateral to the deep inferior epigastric artery. A *direct inguinal hernia* protrudes through Hesselbach's triangle, i.e. medial to the inferior epigastric artery. Both hernias, if large enough, leave the inguinal canal through the external ring. Either type of hernia, if confined to the inguinal canal, is termed a "*bubonocoele*." *Combination of direct and indirect hernia*.—Both an indirect and a direct sac may be present, with the inferior epigastric

vessels stretched over the junction between them = "saddle" or "pantaloon" hernia (Fig. 97).

**Indirect Inguinal Hernia.** An indirect hernia in adults is often due to the presence of a preformed (i.e. congenital) sac, but the hernia may be acquired. An indirect hernia (Fig. 98) is slightly more common on the right side than on the left. It is found at any age, but often it does not make its appearance until the latter half of life. In bilateral hernias one usually appears some years before the second. A preformed indirect hernial sac represents the persistence of the whole or part of the *processus vaginalis* which during fetal development follows the path of the migrating testicle. The sac therefore lies within the substance of the spermatic



FIG. 98. Indirect inguinal hernia extending into the scrotum.

cord anterior to the vas and testicular vessels, to both of which it is closely adherent. The covering layers are the extraperitoneal fat and the three structures derived from the three layers of the abdominal wall, i.e. the internal spermatic fascia from the transversalis fascia, the cremaster muscle from the internal oblique muscle, and the external spermatic fascia from the external oblique. An acquired indirect hernial sac differs in no way anatomically from a congenital sac. An indirect inguinal hernia may be seen to descend obliquely on coughing. When large, it enters the scrotum in the male or the labium in the female. The association of indirect inguinal hernia with maldescent of the testicle is discussed in Chap. 14. *Complications.*—Irreducibility (Fig. 99), intestinal obstruction and strangulation occur, although they are less common than in moral hernia.

**Direct Inguinal Hernia.** This is nearly always an acquired condition, but sometimes the pubic insertion of the conjoint tendon is absent, suggesting a congenital muscular defect. The average age of onset is over

forty, and direct hernias are bilateral in 50 per cent. of cases. A direct hernia (Figs. 100 and 101) enters the inguinal canal through its posterior wall, either lateral or medial to the lateral umbilical ligament (Fig. 96). Since it does not become invested by the internal spermatic fascia it rarely enters the scrotum or labium. With the exception of this layer its coverings are the same as those of an indirect hernia. Although a well-formed sac may be present there is sometimes only a "bulge" of the posterior wall of the canal. Narrow, "funicular" sacs *through* the conjoint tendon occur less

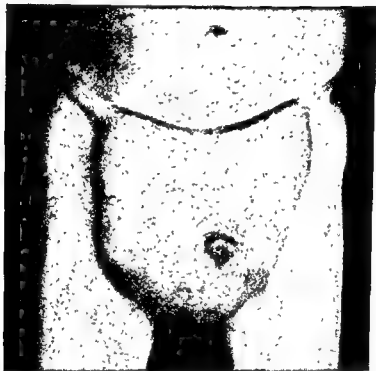


FIG. 99. Bilateral indirect inguinal hernias. Both were irreducible.  
(Case of Professor J. C. Goligher.)

commonly, but are sometimes found even in young men. *Complications.*—Irreducibility and strangulation are rare owing to the usually wide neck of a direct inguinal sac.

**Differentiation of Indirect and Direct Hernias.** Indirect and direct inguinal hernias are theoretically differentiated by their relationship to the deep inferior epigastric artery, but this vessel is palpable only on rare occasions. However, a differentiation can nearly always be made on clinical examination. In direct hernia the examining fingers pass directly backwards through the defect in the abdominal wall, whereas in indirect hernia this is possible only if its neck is very wide. When the sac is large enough, an indirect hernia may descend into the scrotum or labium, whereas a direct hernia almost never enters the scrotum. An indirect



FIG. 100. Bilateral direct inguinal hernias. This patient also had dysuria from an enlarged prostate.

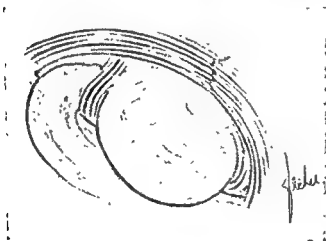


FIG. 101. A right direct inguinal hernia, showing a wide-necked sac medial to the deep epigastric vessels.

hernia can usually be prevented from coming down by pressure over the internal ring (i.e. half-way between the anterior superior iliac spine and the symphysis pubis); pressure over the internal ring never controls a direct hernia. Sometimes differentiation may be clinically impossible. Thus, pressure over the internal ring when the patient coughs may fail to control a small indirect hernia with a large neck, and a small direct hernia arising lateral to the lateral umbilical ligament may simulate an indirect hernia as its course is oblique.

### DIFFERENTIAL DIAGNOSIS OF INGUINAL HERNIA

(a) **Inguinal Hernia Limited to the Groin.** A *reducible* hernia has to be differentiated from:—(1) *Malgaigne's "Bulge."*—On standing, some spare people of poor physique may be seen to have a distinct bulge in each groin, yet *no expansile impulse* can be detected. This condition is sometimes followed by the development of direct inguinal hernia. (2) *Femoral Hernia.*—Inguinal and femoral hernias are closely situated; their differentiation is considered later. (3) A *Lipoma* within the inguinal canal.

An *irreducible* inguinal hernia confined to the groin has to be differentiated from:—(1) A *Hydrocele of the Cord* (or of the *Canal of Nuck*, in the female), which does not reduce. It often transilluminates and it becomes fixed on traction of the testicle. (2) A *Mal descended Testicle.*—If the scrotum is examined and found to be empty on the side of the swelling no error will be made. (3) *Torsion of the Testicle.*—In this condition the testicle lies either high in the scrotum, or in the groin (mal descended), and the cord is thickened. Differentiation from a strangulated hernia may be particularly difficult. A normal testicle, however, is present in the scrotum in most cases of hernia. (4) A *Psoas Abscess* may point in the groin and thus resemble a hernia.

(b) **Inguinal Hernia in the Scrotum.** (1) *Reducible* hernias present no difficulty. (2) *Irreducible* hernias must be differentiated from:—A *Testicular Hydrocele.*—This swelling lies in the scrotum but may extend up into the groin. On palpation, however, the examining thumb and index finger can be closely approximated above the swelling, for the inguinal canal itself contains nothing abnormal. A hydrocele usually transilluminates well. A *Haematocoele.*—This does not transilluminate and the swelling does not extend into the inguinal canal. Furthermore, there may be a history of a blow on the testicle. *Epididymo-orchitis.*—Acute epididymo-orchitis may be associated with frequency of micturition and pyrexia. The testicle is enlarged and, although the cord is thickened, the swelling in the canal does not feel wide enough for an inguinal hernia. A *Varicocele.*—On palpation a varicocele gives the feeling of a "bag of worms" and confusion with a hernia is most unlikely. The presence of a small hernia in addition, however, may be unrecognised if a careful examination is not made.

### TREATMENT OF INGUINAL HERNIA

Operation is the best treatment, but a *reducible* hernia may be controlled by a truss. Although this appliance causes a certain amount of scarring the hernial sac is never obliterated; therefore if a hernia is to be treated without operation the truss must be worn for life. In elderly patients a small hernia that is certainly direct may be left alone, provided that it is observed not to be increasing in size.

**Trusses.** Trusses are as a rule only prescribed for reducible hernias. An exception may be made in the case of a feeble patient with a gross irreducible inguinal hernia; a supporting "bag" truss may then be supplied if operation is contraindicated. A truss presses upwards and backwards on the external ring. There are two types of appliance: the elastic and the spring truss. The latter is more efficient both with regard to the *direction* and the *constancy* of the pressure applied. Small direct hernias in bony individuals may, however, be satisfactorily controlled by an elastic truss, but the patient must not be allowed to apply it too loosely.

The necessary measurement for prescribing a truss is taken around the pelvis in line with the base (upper margin) of the sacrum and the top of the symphysis pubis. Laterally, the tape is usually found to traverse the hips half-way between the anterior superior iliac spine and the greater trochanter. The appliance maker should also be informed of the exact nature of the hernia, i.e. whether it is indirect or direct, scrotal or confined to the groin, and whether it is easily reducible or not. Even to-day patients are sometimes referred for the fitting of a spring truss for a hernia which has not been diagnosed by the clinician as being irreducible! The appliance maker himself should examine and measure patients, for the expert technician can fashion a truss that will lie in a position natural to the particular patient. The cork pad of an inguinal truss is usually pear-shaped but in large scrotal hernias it may, with advantage, be projected downwards (the "scrotal" pad). In hernias restricted to the groin, extension of the normal inguinal pad by means of a soft extension downwards ("rat tail") may improve the direction of the pressure.

A truss must always be applied with the hernia reduced and with the patient recumbent. If it is satisfactory the hernia is controlled when the patient stoops with the legs apart and coughs. A truss may be left off at night but should be reapplied in bed before rising. For the first few weeks the skin should be rubbed daily with spirit, and powdered. The appliance must be kept in good repair. The rubber truss used in infancy is described in Chap. 14.

### OPERATIVE TREATMENT

Operation is the best treatment for most patients. Age is no bar to success and surgical treatment is only contraindicated in patients with severe heart or pulmonary disease, or when two previous attempts at surgical cure have proved unsuccessful. Early surgery is advisable for irreducible hernias even if they are symptomless. Strangulated hernias require emergency surgery.

**Selection of Operation.** The following techniques are in common use:—

- (1) *Herniotomy*—excision of the hernial sac alone.
- (2) *Herniorrhaphy*—herniotomy, together with repair of stretched muscles by suture.
- (3) *Hernioplasty*—herniotomy, together with the insertion of a new layer

or barrier by means of darning with living fascia, or foreign material, such as nylon, silk or wire; or the insertion of a preformed sheet, e.g. a metal filigree.

In childhood, herniotomy alone is satisfactory (see Chap. 14). It is generally agreed that in young adults with indirect sacs, and without weakness of the posterior wall of the inguinal canal, herniotomy alone is again adequate, for healthy muscles protect the canal better than surgical repair. In all other cases (i.e. in middle-aged and elderly patients, in patients with a stretched internal ring, or a deficiency in the posterior wall of the inguinal canal, and in all direct hernias) some form of repair should be undertaken.

There is considerable disagreement as to the relative advantages of herniorrhaphy and hernioplasty. The former joins thinned muscles and connective tissues under tension, whereas the latter leaves the tissues in their anatomical position. Hernioplasty depends for its success on the strength of the darn and of the fibrous tissue that forms in the interstices. The method used is probably of less importance than the skill and care with which it is applied. With either method success depends on:— (1) Complete excision of the peritoneal sac; (2) careful dissection and unhurried technique in the repair; and (3) correction of aggravating factors.

**Preoperative Measures.** The patient is examined for evidence of any condition which may cause or aggravate a hernia, e.g. chronic bronchitis, emphysema, constipation, or urinary obstruction. Should any such disease be found it must be corrected if possible. Chronic bronchitis is not a contraindication to operation if it can be benefited by medical treatment, although it increases the risk of recurrence. In obese patients weight reduction makes the operation easier and reduces the likelihood of recurrence. In all cases smoking should be discontinued before operation and forbidden during convalescence to minimise coughing. The patient's bladder must be emptied immediately before operation.

**Inguinal Herniotomy.** The incision begins just above the pubic tubercle and extends laterally in a skin crease in the groin. The external oblique muscle is divided parallel with its fibres. The incision is continued downwards so that the external ring is opened. During this manoeuvre care must be taken to avoid damage to the ilio-inguinal nerve. The cremaster muscle is next opened, and the hernial sac picked up. The thin white edge of the sac is then incised and the sac explored with a finger. With the finger still inside it, the sac is dissected free up to its junction with the parietal peritoneum. The sac, having been cleaned of adherent tissue, is then transfixed and ligated. The part distal to the ligature is removed. A simple way of ensuring that the sac is emptied prior to removal is to twist it, if care is taken to see that the vas is clear. Twisting may not be possible with large-necked sacs; a continuous peritoneal suture is then necessary. If the internal ring is stretched, it is tightened

by stitches placed in the transversalis fascia. The cremaster muscle is repaired, and the external oblique muscle closed. Care is taken not to include the ilio-inguinal nerve in a stitch, or to constrict the spermatic cord.

**Inguinal Herniorrhaphy.** In indirect hernia, herniotomy (*vide supra*) must be done before the repair, but when there is a large adherent sac the upper end may be divided and tied off as above, and the scrotal part of the sac left *in situ*. In direct hernias large sacs should be removed, but

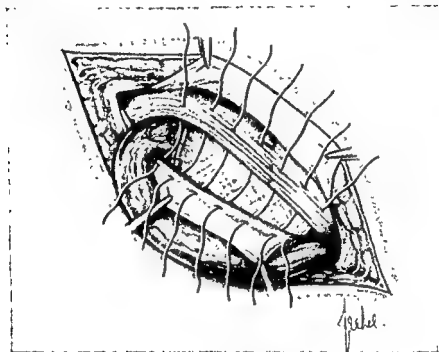


FIG. 102. Bassini's operation. The sac has been ligated and divided, and the repair begun.

mere "bulges" are best either plicated by sutures placed through the transversalis fascia or left alone behind the repair.

**Bassini's Operation.** The sac is removed as described above. The spermatic cord having been retracted, the conjoint muscle and the inguinal ligament (Poupart's) are stripped of their cremasteric and fascial attachments. The conjoint muscle and tendon are then united to the inguinal ligament by interrupted unabsorbable sutures behind the spermatic cord (Fig. 102). The external oblique muscle is closed. The most medial part of the posterior wall of the canal is carefully and firmly closed, for this is a common site for recurrence. Bassini's operation undoubtedly causes atrophy of the conjoint muscle and tendon, and the effectiveness of the repair therefore presumably depends on fibrosis occurring in the posterior wall of the canal. Modifications of the Bassini



or barrier by means of darning with living fascia, or foreign material, such as nylon, silk or wire ; or the insertion of a preformed sheet, e.g. a metal filigree.

In childhood, herniotomy alone is satisfactory (see Chap. 14). It is generally agreed that in young adults with indirect sacs, and without weakness of the posterior wall of the inguinal canal, herniotomy alone is again adequate, for healthy muscles protect the canal better than surgical repair. In all other cases (i.e. in middle-aged and elderly patients, in patients with a stretched internal ring, or a deficiency in the posterior wall of the inguinal canal, and in all direct hernias) some form of repair should be undertaken.

There is considerable disagreement as to the relative advantages of herniorrhaphy and hernioplasty. The former joins thinned muscles and connective tissues under tension, whereas the latter leaves the tissues in their anatomical position. Hernioplasty depends for its success on the strength of the darn and of the fibrous tissue that forms in the interstices. The method used is probably of less importance than the skill and care with which it is applied. With either method success depends on :— (1) Complete excision of the peritoneal sac ; (2) careful dissection and unhurried technique in the repair ; and (3) correction of aggravating factors.

**Preoperative Measures.** The patient is examined for evidence of any condition which may cause or aggravate a hernia, e.g. chronic bronchitis, emphysema, constipation, or urinary obstruction. Should any such disease be found it must be corrected if possible. Chronic bronchitis is not a contraindication to operation if it can be benefited by medical treatment, although it increases the risk of recurrence. In obese patients weight reduction makes the operation easier and reduces the likelihood of recurrence. In all cases smoking should be discontinued before operation and forbidden during convalescence to minimise coughing. The patient's bladder must be emptied immediately before operation.

**Inguinal Herniotomy.** The incision begins just above the pubic tubercle and extends laterally in a skin crease in the groin. The external oblique muscle is divided parallel with its fibres. The incision is continued downwards so that the external ring is opened. During this manoeuvre care must be taken to avoid damage to the ilio-inguinal nerve. The cremaster muscle is next opened, and the hernial sac picked up. The thin white edge of the sac is then incised and the sac explored with a finger. With the finger still inside it, the sac is dissected free up to its junction with the parietal peritoneum. The sac, having been cleaned of adherent tissue, is then transfixed and ligated. The part distal to the ligature is removed. A simple way of ensuring that the sac is emptied prior to removal is to twist it, if care is taken to see that the vas is clear. Twisting may not be possible with large-necked sacs ; a continuous peritoneal suture is then necessary. If the internal ring is stretched, it is tightened

(Gallie), but this technique leaves a bulge in the thigh. Many surgeons prefer to use long strands (single or double) of nylon, silk or stainless steel wire. The use of ~~slow~~ silk or ~~slow~~ nylon has given a low recurrence rate in the hands of some surgeons (Maingot). Monofilamentous nylon, used as a double strand, is probably as effective and safe as any other material. A darn is performed behind the cord between the aponeurotic portions of the internal oblique and transversus muscles, and the inguinal ligament (Fig. 103). Impeccable asepsis is essential in hernioplasty, for if the wound becomes infected it is unlikely to heal unless all unabsorbable suture material is removed.

**After-care.** Early ambulation reduces the incidence of complications in general; it also prevents harmful straining on the bed pan. Early activity is particularly important in the aged after operation for strangulation, to prevent pulmonary complications. After operation patients should carry out breathing exercises and graduated abdominal exercises under the supervision of the physiotherapist. Such treatment reduces pulmonary complications and prevents wasting of the abdominal musculature in the convalescent stage. The sooner a return to full activity is achieved after herniotomy the better; strenuous exertion is permissible after one month. After herniorrhaphy or hernioplasty, a more gradual return is required; light work may be begun in one month, but strenuous exertion should be delayed for three months. After herniorrhaphy and hernioplasty a truss does not reduce the chances of recurrence and may be harmful.

**Complications of Hernia Operations.** *Intestinal distension and difficulty in micturition* are not uncommon for a short while after operation. Early movements in bed reduce the severity of these symptoms. *Damage to Nearby Structures.*—Division of the vas deferens or of the testicular vessels should be avoided by care in dissecting the sac; if they are injured testicular atrophy results. The femoral vein is soft and difficult to feel; care must therefore be taken not to prick it when sutures are being passed through the inguinal ligament. The bladder must be safeguarded both during dissection on the medial aspect of the hernia and when the sac is tied; if it is damaged a urinary fistula or peritonitis may result. If a hole is made in the bladder, it is closed with interrupted sutures, and the bladder is drained for a few days by means of a urethral catheter. *Haematomas* may occur in the wound, or in the scrotum after excision of a large indirect sac. They are prevented by strict hæmostasis (coagulation diathermy is recommended) and by supporting the scrotum after operation. *Wound sepsis* is more common after operations for strangulation but it may be prevented if a strictly aseptic technique is used. Following hernioplasty, sepsis usually necessitates removal of buried foreign material, but if monofilamentous nylon has been used healing may occur with conservative treatment. *Thrombophlebitis of the spermatic cord (funiculitis)* is due to rough handling of the cord, or incomplete hæmo-

technique have been introduced in an attempt to reduce the recurrence rate. Thus, the tension produced in the conjoint muscle by Bassini's operation may be considerably relieved by incising the anterior sheath of the rectus muscle, deep to the external oblique (*Halsted-Tanner "slide"*). In another method (Brandon's) the internal ring is displaced laterally after incising the internal oblique and transversus muscles, moving the cord laterally, and resuturing the muscles on its medial side.

*Halsted's Operation.* In this operation the external oblique aponeurosis

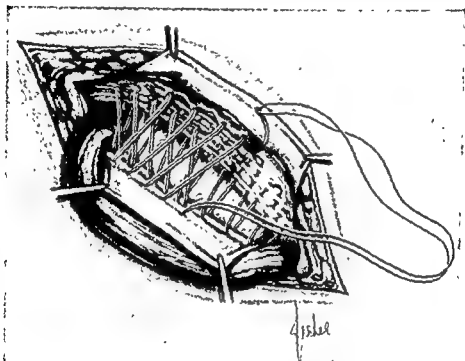


FIG. 103. Nylon darn hernioplasty for inguinal hernia.

is divided over the course of the spermatic cord, and the aponeurosis is sutured behind the cord which then passes directly through the abdominal wall. Although a good posterior barrier is formed, the "valvular" action of the normal inguinal canal is lost. Halsted later recommended transposition of the cord deep to all the abdominal muscles, but in this procedure there is a serious risk of recurrence at the medial end of the canal.

*Removal of the Testicle.* A complete closure of the inguinal canal may be achieved by excising the testicle and cord up to the internal ring. In senile patients this is sometimes justified. In women removal of the round ligament permits sound repair in operations for hernia.

*Hernioplasty.* Some form of hernioplasty is necessary for large direct hernias and for most recurrent hernias. Before the repair is begun the sac is dealt with as indicated previously. The repair may be carried out with fascia lata taken from the thigh by means of a fascial stripper

preformed sac is probably often present, remaining empty until increased abdominal pressure or muscular weakness causes it to fill. The majority (over 80 per cent.) of femoral hernias occur in females, presumably owing to the wider pelvis in this sex and the effect of childbearing.

**Contents of the Sac.** Often the sac is empty or contains only omentum. A sliding hernia of the bladder may be found in the medial part of the sac. If small intestine is herniated, intestinal obstruction or strangulation usually soon follows. The ovary or Fallopian tube is occasionally found in a femoral hernia. The *coverings* of a femoral hernia are the extra-peritoneal fat, the septum crurale and the cribriform fascia. The femoral lymph gland lies at the apex of the sac.



FIG. 104. A left femoral hernia in a woman aged thirty.

On examination the swelling is often found to be small. It is somewhat more *laterally* placed and a little *lower* than an inguinal hernia. A large femoral hernia rising up into the groin may at first appear to be an inguinal hernia. The neck of a femoral hernia, however, is found to lie below the pubic tubercle. If there is difficulty in palpating this point, the adductor longus muscle is traced to its insertion; the pubic tubercle will be found immediately above it. A femoral hernia is often incompletely reducible or irreducible. The apex of the sac is bulky as the result of thickening of its many coverings; the femoral lymph gland may be easily palpable. *Complications.*—The frequent occurrence of irreducibility has been mentioned. Strangulation occurs in approximately 10 per cent. of femoral hernias.

**Differential Diagnosis.** A *reducible* femoral hernia must be differentiated from:—(1) *Inguinal Hernia.*—A femoral hernia is rather more laterally placed and its *neck* lies below the pubic tubercle. The presence of an inguinal hernia may be excluded by introducing the little finger gently

stasis, and is thus avoidable. A *testicular hydrocele* may result from closing either of the abdominal rings too tightly, or from traumatic thrombosis of the pampiniform plexus. *Neuralgia*.—Care must be taken to avoid damaging, or including in a stitch, the iliohypogastric, the ilio-inguinal or the genital nerve, otherwise severe pain in the groin or scrotum may result.

**Recurrence.** Factors predisposing to recurrence include:—(1) Faulty selection of cases for operation. (2) Technical errors, including: (a) failure to recognise a double sac, or a "saddle" hernia (Fig. 97); (b) incomplete removal of the sac; (c) incomplete or inadequate suture of the peritoneum; (d) incorrect technique in repair. (3) Postoperative chest complications. (4) Postoperative sepsis. (5) Too early a return to strenuous activity. The incidence of recurrence in direct hernias has been reported to be as much as 20 per cent. Removal of congenital indirect sacs in young patients is most satisfactory, but in other types of indirect hernia long-term follow-up reveals a recurrence rate approaching 10 per cent. Although even in expert hands, surgical cure cannot be guaranteed, some recurrences are still due to faulty selection of patients and inadequate surgical technique. However, a second operation is often successful. In secondary operations the anatomy may be distorted; the vas and vessels must therefore be sought and safeguarded as soon as possible. The most satisfactory repair in recurrent cases is some form of hernioplasty. Operation is still worth while even for a second recurrence, provided that the patient is otherwise healthy; if this fails the patient must wear a truss.

### FEMORAL HERNIA

**The Femoral Canal.** This structure is conical with its base directed upwards. It is 1.25 cm. long and contains areolar tissue and a lymph gland (Cloquet's gland). The *femoral ring* is situated to the lateral side of the pubic tubercle. It is bounded in front by the inguinal ligament, behind by the pectineal (Astley Cooper's) ligament and the pectineal fascia, medially by the reflected part of the inguinal ligament (lacunar or Gimbernat's ligament), and laterally by the femoral vein within the femoral sheath. The femoral ring is normally closed by the *septum crurale*. The *saphenous opening* lies 1.25 cm. below the femoral ring. It is covered by the cribriform fascia, and its outer and upper margin is formed by the free edge of the deep fascia of the thigh.

**Relations of a Femoral Hernia.** A femoral hernia passes through the femoral ring and down the femoral canal (Figs. 90 and 104). Having made its way through the saphenous opening, it then passes forwards and upwards to lie in front of the medial end of the inguinal ligament. Although the neck of a femoral hernia is narrow, owing to the small diameter of the femoral canal, once the hernia has escaped through the saphenous opening it may become considerably enlarged. *Ætiology*.—A

preformed sac is probably often present, remaining empty until increased abdominal pressure or muscular weakness causes it to fill. The majority (over 80 per cent.) of femoral hernias occur in females, presumably owing to the wider pelvis in this sex and the effect of childbearing.

**Contents of the Sac.** Often the sac is empty or contains only omentum. A sliding hernia of the bladder may be found in the medial part of the sac. If small intestine is herniated, intestinal obstruction or strangulation usually soon follows. The ovary or Fallopian tube is occasionally found in a femoral hernia. The *coverings* of a femoral hernia are the extra-peritoneal fat, the septum crurale and the cribriform fascia. The femoral lymph gland lies at the apex of the sac.



FIG. 104. A left femoral hernia in a woman aged thirty.

On examination the swelling is often found to be small. It is somewhat more *laterally* placed and a little *lower* than an inguinal hernia. A large femoral hernia rising up into the groin may at first appear to be an inguinal hernia. The neck of a femoral hernia, however, is found to lie below the pubic tubercle. If there is difficulty in palpating this point, the adductor longus muscle is traced to its insertion; the pubic tubercle will be found immediately above it. A femoral hernia is often incompletely reducible or irreducible. The apex of the sac is bulky as the result of thickening of its many coverings; the femoral lymph gland may be easily palpable. *Complications.*—The frequent occurrence of irreducibility has been mentioned. Strangulation occurs in approximately 10 per cent. of femoral hernias.

**Differential Diagnosis.** A *reducible* femoral hernia must be differentiated from:—(1) *Inguinal Hernia.*—A femoral hernia is rather more laterally placed and its *neck* lies below the pubic tubercle. The presence of an inguinal hernia may be excluded by introducing the little finger gently

into the inguinal canal after invaginating the scrotum or labium and asking the patient to cough. If an inguinal sac is present an expansile impulse is felt. (2) *Saphenous Varix*.—Such a swelling is not uncommon in the groin (Fig. 105). It usually has a bluish tinge, vanishes on recumbency, and has a fluid jet thrill when the patient coughs. (3) *Psoas Abscess*.—In this condition a mass may also be felt above the inguinal



FIG. 105. A saphenous varix simulating a left femoral hernia.

ligament and fluctuation obtained between the two swellings. Examination of the spine may show evidence of Pott's disease. (4) *Femoral Aneurysm*. This is expansile with the pulse. (5) *Lipoma*.—Differentiation may prove difficult, particularly as there is always a considerable amount of fat surrounding a femoral sac. (6) *Obturator Hernia*.—This occasionally presents as a palpable swelling in the groin. There is no evidence of a femoral or an inguinal hernia.

An irreducible femoral hernia may, if small, easily be confused with an enlarged lymph gland. The presence of other large glands in the vicinity, or the presence of an infective lesion on the leg, back or anus, is suggestive

of adenitis. A glandular swelling also lies transversely rather than longitudinally; the reverse applies to a hernia. Often the diagnosis can only be settled by exploration. If the swelling is associated with vomiting urgent operation should always be undertaken.

**Rare Varieties of Femoral Hernia.** Very occasionally an inguinal hernial sac enters the femoral canal and presents at the saphenous opening instead of in the scrotum (*inguinofemoral hernia*). An *external femoral hernia* is one which passes laterally to the femoral vessels. Femoral hernias have also been described in other situations, e.g. in front of the vessels, or burrowing into the pectineus muscle.

### TREATMENT

Femoral hernias frequently become complicated. They should therefore be operated upon unless there is some very strong objection. In patients who are quite unfit for surgery a truss may be prescribed, but it is likely to slip on walking, and if it becomes misplaced the hernial contents may be damaged. The necessary measurements are the same as for an inguinal truss, but the spring is shaped to bring the pad to lie over the femoral ring. The pad differs from that of the inguinal truss in that it is prolonged slightly downwards over the thigh. Some surgeons prescribe an added thigh-encircling strap.

**Operation.** The inguinal approach of Lotheissen is the easiest and safest method of exposure. There is said to be a slight risk of inguinal hernia later, but it can be prevented by careful repair of the inguinal canal. Some surgeons prefer the "low approach," in which the sac is exposed and excised from below the inguinal ligament. In strangulated hernias a combination of both methods has many advantages.

*The Inguinal Operation (Lotheissen).* The skin incision is similar to that used for inguinal hernia except that it extends lower down at its medial end. The external oblique is divided and the round ligament or spermatic cord is retracted. The transversalis fascia is next incised to disclose the neck of the sac. The sac is opened and its contents reduced; it is then pulled up into the inguinal canal and dissected free. The neck is transfixed flush with the abdominal peritoneum and ligated, and the sac excised. Care must be taken not to injure the bladder on the medial side of the sac, or the femoral vein on the lateral side. When there is difficulty in delivering the sac through the femoral ring the lower end of the wound should be retracted downwards and the sac freed below the inguinal ligament, after incising its several fascial layers. Afterwards, the femoral canal is obliterated by inserting a single layer of interrupted unabsorbable sutures between the conjoint tendon and the pectineal (Astley Cooper's) ligament. Finally, the external oblique and the skin are sutured. The contraindications to operation, the after-care and complications are very similar to those of inguinal herniorrhaphy. Post-operative recurrence is about 4 per cent.



**Strangulated Femoral Hernia.** Taxis is contraindicated, but conservative aids to spontaneous reduction (*vide supra*) may be attempted whilst preparations are made for operation. At operation the lower edge of the wound is first retracted to expose the sac which is then opened. Next, the inguinal canal is exposed by dividing the external oblique aponeurosis, and the neck of the sac is defined as above. The reflected part of the inguinal ligament (Gimbernat's) may then be divided under direct vision. Any constriction is usually released in this manner, but division of the neck of the sac may also be necessary. If there is great difficulty in reduction, the inguinal ligament may be divided over the sac in order to release the constriction (Hey Groves). The rare abnormal obturator artery\* runs behind Gimbernat's ligament and must be avoided; in Lotheissen's approach, however, if such a vessel is damaged, it can be ligated with ease.

### UMBILICAL HERNIA

Umbilical hernia in childhood is described in Chap. 15. Acquired hernias occur most frequently in obese women in the fourth decade. They generally arise on the edge of the umbilical scar and are more properly called *para-umbilical hernias*. They may enlarge to a great size and the sac may be covered by only a thin layer of skin. Adhesions are frequently present in the sac, which is often loculated. Incomplete reducibility is common. Colicky pain is frequent and acute intestinal obstruction and strangulation are moderately common. Local signs in strangulation of umbilical hernias are less obvious than in femoral and inguinal hernias; it is easy to overlook a small strangulated hernia in a very obese patient.

**Treatment.** Many patients are obese, have a poor exercise tolerance, or are otherwise not suitable for operation. Weight reduction must therefore be undertaken, and other adverse factors corrected if possible, for radical surgery is advisable in view of the risk of intestinal obstruction or strangulation. Otherwise these hernias are controlled with a surgical corset, although small ones require a well-fitting umbilical truss.

**Mayo's Operation** (Fig. 106). This is the standard procedure, and is performed through a transverse elliptical incision in the skin. The hernial sac is opened at its neck and removed, together with the overlying skin and subcutaneous tissues. The peritoneal opening is repaired, if possible. The cutaneous tissues are then reflected sufficiently for the muscles to be divided outwards for a short distance on each side. Strong interrupted mattress sutures are then inserted through the muscles so that they are overlapped from above downwards; a second layer of

\* The pubic branch of the deep epigastric artery runs downwards and medially to the posterior surface of the pubis where it anastomoses with the pubic branch of the obturator artery. This anastomosis is sometimes enlarged to form the main trunk of the obturator artery (then a branch of the deep epigastric artery) and is then termed the *abnormal obturator artery*.

sutures is placed in the free overlapping edge. This operation has a fairly high recurrence rate, but for moderate-sized hernias no better method has been devised. Giant hernias may require some form of hernioplasty, e.g. the insertion of strips of fascia, silk, or a sheet of tantalum mesh. In these cases an abdominal support is of value after operation.

**Epigastric Hernia.** Small hernias of extraperitoneal fat through the linea alba occur usually above the umbilicus. They are small, and often

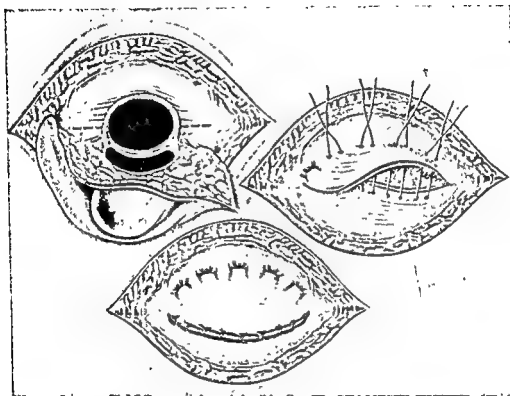


FIG. 106. Mayo's operation for umbilical hernia. (See text.)

tender and painful. Sometimes they may be difficult to feel. On occasion they are associated with reflex vomiting. In patients with this symptom, however, intra-abdominal disease must be excluded before operation on the hernia. Operative treatment consists of excision of the herniated fat. Usually there is no peritoneal sac, but if one is present it is removed. The defect in the linea alba is sutured.

**Divarication of the Rectus Abdominis Muscles.** This is due to stretching of the linea alba with the consequent production of a bulge, either above or below the umbilicus. It is most often seen in obese parous women. Reparative surgery is possible but a surgical belt usually proves effective. A similar condition is seen in infancy, but it tends to improve as the child grows.

### RARER FORMS OF EXTERNAL HERNIA

**Obturator Hernia.** The sac of an obturator hernia follows the obturator vessels into the thigh. Mild attacks of pain referred to the knee may be experienced, but in most cases the first evidence of such a hernia is the development of intestinal obstruction. On examination, the patient may be found to lie with the corresponding hip flexed. A swelling can occasionally be detected to the lateral side of the origin of the adductor longus muscle and there may be tenderness in this area. On vaginal examination the hernial mass may be palpable. Treatment consists of laparotomy and reduction of the hernia. The sac should be turned inside out and ligated, and redundant tissue removed. No repair is necessary.

**Gluteal and Sciatic Hernias.** These hernias pass through the superior and the inferior sciatic notches respectively. Pain and discomfort may be felt at the site of hernia. Occasionally there is severe "sciatica." A swelling may be felt in the buttock, but usually patients present themselves with acute intestinal obstruction. Treatment in such cases is laparotomy and excision of the sac via the abdomen.

**Spigelian Hernia.** A Spigelian hernia is one which passes externally through the lateral edge of the rectus sheath where it may be felt as a lump (Fig. 90). Operative repair may be undertaken.

**Lumbar Hernia.** A lumbar hernia (Fig. 90) passes through Petit's triangle, which is formed by the iliac crest, the lateral border of the external oblique muscle and the edge of the latissimus dorsi. Such a hernia rarely leads to symptoms. Incisional hernias in this region, however, tend to enlarge and should be repaired.

**Perineal Hernias.** These are rare and are usually due to trauma or, in women, to difficult labour. The hernia traverses the pelvic floor and presents in the perineum. Those in the anterior perineum enter the labium or scrotum and usually contain part of the bladder. More posteriorly, hernias enter the ischiorectal fossa and usually contain intestine. Operative treatment may be undertaken.

### INCISIONAL HERNIAS

Post-operative dehiscence of surgical wounds is considered in Chap. 12. Failure of an abdominal wound to heal firmly may eventually lead to the development of an incisional hernia. Causes of such failure include:— (1) *Inadequate suturing*; (2) the use of drainage tubes; (3) *post-operative wound sepsis*; (4) chest complications; (5) *post-operative distension*; (6) *obesity*; (7) *cachexia* or advanced malignant disease. Apart from the presence of a bulge, an incisional hernia is often symptomless. It tends to increase in size, and adhesions in the sac may lead to attacks of intestinal obstruction. Irreducibility and strangulation sometimes occur, either at the neck of the sac if the peritoneal opening is small, or within the sac. A bulge is noted when the patient stands. It may become much

larger on coughing, or on sitting up from the reclining position without the help of the hands. The muscular defect is most easily felt with the patient relaxed and lying down.

**Prevention.** Predisposing causes (*vide supra*) should be avoided as far as possible. Every abdominal scar should be inspected one month after operation and, if it appears to be stretching, an abdominal support is prescribed for a few months.

**Treatment.** Irreducible hernias, small hernias and those associated with colicky pains or vomiting should be operated upon at the earliest opportunity. A large symptomless reducible hernia in a patient who is very obese, or otherwise unsuitable for operation, is controlled by a surgical corset. *Operative treatment*, although often straightforward, may be most exacting. Division of the multitudinous adhesions between coils of intestine and the sac may result in damage to the bowel; as little separation as possible, therefore, should be undertaken. Although the sac need not be completely removed the various layers of the abdominal wall must be identified. Closure of the abdominal wall in layers may then be possible; if not, a Mayo overlap may be feasible. In large defects darning with fascia or nylon, or insertion of tantalum mesh is usually necessary.

### MASSIVE HERNIAS

Long-standing incisional, umbilical and, occasionally, inguinal hernias may be of enormous size (Fig. 107). Reduction of the contents at operation then proves difficult if the capacity of the abdominal cavity has become reduced. Weight reduction is essential before operation. A period of preoperative recumbency, with the foot of the bed raised, is also of considerable value. This not only assists the hernia to become partially reduced, but also helps the patient to become adapted to "bed life," so that there is less difficulty with the bladder and bowels immediately after operation. Post-operative complications are common, however, as a result of the technical difficulties of repair, the poor state of the abdominal musculature, and a reduction in the vital capacity from elevation of the diaphragm.

### INTERNAL ABDOMINAL HERNIAS

Hiatus hernia and other diaphragmatic hernias are considered in Chap. 43. Other types of internal abdominal hernia may be congenital or acquired, and usually contain small intestine. The bowel either enters a peritoneal recess (fossa), or passes through an abnormal opening in the peritoneum. The various sites of internal herniation are as follows:—  
(1) *Congenital recesses*, e.g. around the duodenum and caecum; (2) *anomalous openings*, e.g. in the omentum, mesentery or the broad ligament; (3) *post-operative openings*, e.g. following gastroenterostomy, or the resection of bowel; (4) *natural openings*, e.g. the lesser peritoneal sac;

(5) *other confined spaces*, e.g. between loops of adherent intestine, or by the side of a sigmoid colostomy.

**Symptoms.** The patient may suffer from attacks of mild abdominal discomfort or colic, for which no cause can be found. More often the



Fig. 107. Massive umbilical hernia. There is necrosis of the skin over the apex of the sac. (Case of Professor C. G. Rob.)

hernia is silent until acute intestinal obstruction occurs. Occasionally an internal hernia is an incidental finding on exploration of the abdomen for some other condition.

**Prevention.** Post-operative hernias may be prevented by closing all defects in the peritoneum and leaving no confined spaces into which small intestine can prolapse.

**Treatment.** This usually consists of emergency laparotomy and release of the hernial contents. The constricting neck of a peritoneal recess is divided but *any major blood vessels in it are avoided*. Viability of the gut must be checked and any devitalised bowel resected.

## SECTION II

### PÆDIATRIC SURGERY

by

### VALENTINE A. J. SWAIN

#### CHAPTER 14

### THE INGUINO-SCROTAL REGION

#### INGUINAL HERNIA IN INFANCY AND CHILDHOOD

INGUINAL hernia, which is a common deformity in children, arises as the result of incomplete closure of the processus vaginalis ; this extension of the abdominal parietal peritoneum, which traverses the whole length of the inguinal canal, lies closely adherent and anterior to the spermatic cord and testicle, and remains open till just before birth, when it closes at two points, namely, the internal abdominal ring and just above the testicle. Normally, the intervening part, called the funicular process, becomes obliterated, whereas the distal part, remaining patent, forms the tunica vaginalis of the testis. Failure of closure of the processus vaginalis at the internal abdominal ring may give rise to an incomplete inguinal hernia, but if the funicular process also remains patent, a complete inguinal hernia may result, whereby abdominal contents can enter the scrotum (Fig. 108). In children, inguinal hernia may develop at any age and is always of the indirect (congenital) type. The right side is more commonly affected than the left in the proportion of 4 to 1 ; it rarely occurs in girls.

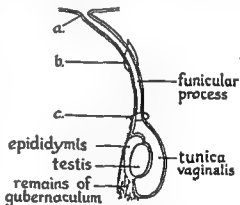


FIG. 108. Showing the formation of the processus vaginalis before the funicular process has become obliterated. (a) Represents the level of the internal abdominal ring. (b) The external abdominal ring. (c) The neck of the scrotum. (After Keith.)

**Symptoms and Signs.** An inguinal hernia may appear at or soon after birth following a strain such as crying or coughing ; the protrusion, which is slight at first, may not be apparent at the initial clinical examination. The rupture, which is seen best in babies during a bout of crying and in

older children when they are standing, appears as a soft smooth swelling in the groin, emerging from the external abdominal ring (Fig. 109); the tips of the examining thumb and finger cannot be brought close together above the swelling as with hydroceles. The hernia contents (commonly bowel or omentum) can usually be reduced by gentle pressure on the rupture in the direction of the inguinal canal. When the child is straining, pressure over the internal abdominal ring controls the rupture. Discomfort and vomiting may occur when a hernia is "down," but not



FIG. 109. Showing a left indirect inguinal hernia in an infant.

actually strangulated (*vide infra*); crying aggravates the symptoms. Most hernias become reduced when the patients lie down. If the rupture is not easily reducible, the loop of incarcerated intestine becomes swollen and congested, and later gangrenous (*strangulated hernia*); inflammation of the adjacent hernial sac causes redness, oedema and tenderness of the surrounding skin, and may thus lead to confusion with lymphadenitis. In addition to these local manifestations, signs of intestinal obstruction develop, except when only part of the circumference of the bowel is trapped in the sac (*Richter's hernia*); diarrhoea and melæna may then occur.

Fortunately, the majority of infantile hernias are easily reducible, although under the age of one year the chances of easy reduction are much less than at any other age. *Therefore failure to control an inguinal hernia in the first year is liable to lead to intestinal obstruction.*

**Treatment.** Single or bilateral reducible hernias, seen soon after birth, are best treated by means of a truss, which will relieve any discomfort, give a chance of cure and prevent strangulation of the contents. A rubber-covered horse-shoe shaped truss is the most suitable (Fig. 110). The size required is equivalent to the measurement of the girth at the level of the umbilicus. The child's mother is shown how to fit the support: after reduction of the hernia by taxis,\* the limbs of the U-shaped pad are placed over the internal abdominal rings and held in position by the belt around the waist and the straps between the legs. It is advisable to apply dusting powder to the skin in contact with the truss to avoid soreness. The truss, which is kept in place at all times even when the baby is washed, is worn up to the end of the first year and then discarded; in a few instances the hernia will then be cured, but usually surgical repair is necessary. Operation is best carried out after the age of one year, although some surgeons advocate repair in the first few weeks of life. Postponement until a later age is preferable, for the sac will then be

*Taxis* = manual pressure applied to restore parts to their place.

stronger and repair therefore easier. Occasionally, early operation is necessary if the hernia cannot be controlled by the truss, or the rupture is associated with an ectopic or undescended testicle.

An irreducible hernia, which is usually obstructed at the *external abdominal ring*, is treated first by gentle manipulation; the contents of the hernia are guided through the narrow ring with the index finger and thumb of one hand and gentle pressure in the direction of the inguinal canal over the fundus with the other hand until complete reduction has occurred. If the baby is straining, a sedative, such as chloral hydrate is given; 1 grain per year up to the age of five years is a simple working rule. If the attempt is still unsuccessful, the infant is suspended with the head down, either by the legs attached to a gallows splint or by raising the foot of the bed, and cold compresses are applied to the inguinal region. In about three-quarters of cases treated by suspension, reduction is successful, but if, after three hours, this method fails, immediate operation is necessary. Taxis should not be attempted if the skin over the hernia is red or swollen, for this indicates that the underlying contents are adherent to the sac and have become strangulated. Immediate operation is necessary. All inguinal hernias occurring after the age of one year are treated by operation, for the chances of cure by wearing a truss, in older children, are remote. A temporary support is, however, applied if any undue delay is anticipated before operation can be carried out.

*Operation.* This is carried out under general anæsthesia. A curved transverse incision in the skin crease over the external abdominal ring is made. After division of the fatty subcutaneous tissue the cord and its covering are exposed (Fig. 111a); by blunt dissection through the cremasteric fascia the white hernial sac is exposed. The spermatic vessels and vas deferens, being intimately adherent to its under surface, must be carefully freed by dissection (Fig. 111b). The isolated sac is then opened, or in the case of a complete scrotal hernia, divided across, thus allowing the cut distal portion of the sac to recede downwards. By traction on the proximal end of the open sac, the abdominal opening of the hernia is defined as far as the internal abdominal ring. When the hernial contents have been reduced, the neck of the sac is closed by a transfixion stitch inserted with a round bodied needle (Fig. 111c), care being taken to avoid splitting the thin wall of the sac. When the redundant portion of the sac is cut away, the ligated stump recedes upwards under cover of the external oblique muscle as far as the internal abdominal ring.

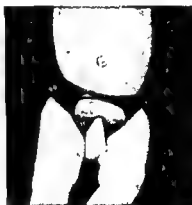


FIG. 110. Rubber horse-shoe truss in position.



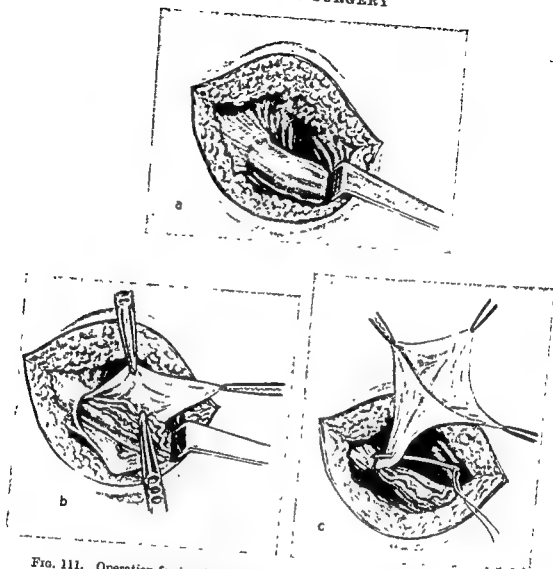


FIG. 111. Operation for inguinal hernia. (a) Showing the surface of the external abdominal ring and the coverings of the cord. (b) The hernial sac is being separated from the underlying cord. The vas deferens and spermatic vessels are shown. (c) The neck of the hernial sac has been exposed and transfixed before its ligation.

After ensuring that the testicle lies in its natural position, the surgeon approximates the covering of the cord with one or two stitches. If the external abdominal ring is large, it is narrowed by sutures, but compression of the underlying spermatic cord is avoided. The wound is then closed. In females, repair of an inguinal hernia is similar but easier than in males, for the delicate spermatic vessels and the vas are replaced by the strong fibres of the round ligament.

In the operation for irreducible inguinal hernia the same surgical approach is used. When the thickened sac wall is opened blood-stained fluid escapes. After division of the constricting margin of the external ring, the hernial contents can be reduced easily in most instances; but

before the bowel is returned into the abdominal cavity its viability is assessed. The intestine is considered to be non-viable if, when it is dull, dark and purplish, its appearance cannot be improved by the application of hot packs, or if the bowel is necrotic or non-contractile. If the bowel is judged to be non-viable resection is necessary. The continuity of the intestine is best restored by a single layer end-to-end junction. It may be possible to do this through the hernial opening, although a separate paramedian incision is preferred, in order to obtain better mobilisation of the gut, easier entry of the anastomosis into the abdomen and better repair of the hernia. Gangrene of the testicle may sometimes occur with a strangulated hernia as the result of interference with the spermatic vessels; the testicle is purplish black. Orchidectomy is advised, for if the ischaemic testicle is retained it is most likely to be extruded later from the wound as a slough. After reduction of the intestine, as in uncomplicated cases, separation of the sac and its ligation at the internal abdominal ring is carried out. This step may be difficult, for the peritoneal lining, although thickened by oedema, is often friable. After repair of the cut margin of the external abdominal ring the wound is closed.

Following operation for a simple hernia, the child is able to resume normal activities after a few days. In complicated cases, however, post-operative management is dictated by the findings at operation. Thus in obstructed cases, gastric suction, parenteral fluids and gradual introduction of feeding is necessary (see under Intestinal Obstruction).

**Femoral Hernia.** This type of hernia, which is uncommon in children, is confined mostly to the female sex. The clinical features are similar to those in adults and the treatment is the same. At operation the sac, which is usually surrounded by fatty tissue, is often poorly developed. Occasionally the close proximity of the hernia to the bladder may cause increased frequency of micturition.

### HYDROCELES IN INFANCY AND CHILDHOOD

The term hydrocele (literally a collection of fluid in an enclosed sac) is usually applied to excess of fluid within the tunica vaginalis of the testis (Fig. 112C). This type of hydrocele, which occurs commonly in children, may appear soon after birth without obvious cause. Occasionally a hydrocele may arise secondary to injury or disease of the testicle. The idiopathic variety is more common. One or both sides of the scrotum become swollen as the result of accumulation of straw-coloured fluid within the tunica vaginalis. Pressure of the encysted fluid, which lies anterior to the testicle, widens the sulcus between the epididymis and testis. If the whole length of the processus vaginalis is patent, the fluid in the hydrocele is able to enter the peritoneal cavity (Fig. 112A). Thus, this communicating type of hydrocele can vary in size by change of posture, emptying on lying down and filling on standing. Occasionally,

when the processus vaginalis is occluded at the internal abdominal ring as well as at the upper level of the scrotum, a fluid collection in the funicular process forms an encysted hydrocele of the cord (Fig. 112B). The last variety occurs also in girls, in which case the encysted fluid collects in the canal of Nuck.

**Symptoms and Signs.** Idiopathic hydroceles in infancy are usually symptomless. A gradual increase in size of one side of the scrotum, more commonly on the right, is first noticed. The tense, soft, smooth swelling,

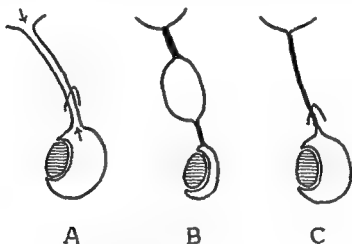


FIG. 112. The various types of hydrocele. A. Communicating type of hydrocele in which the processus vaginalis remains patent. B. Encysted hydrocele of the cord. C. Hydrocele of the tunica vaginalis.

which is covered with normal skin, is dull to percussion and translucent to light : the testicle can be felt lying posteriorly (Fig. 113). If the hydrocele is confined to the tunica vaginalis, the texture of the cord above the swelling is normal. If the funicular process is patent, the hydrocele extends into the inguinal region and occasionally pressure on the scrotum below causes the hydrocele to empty, thus demonstrating its connection with the abdominal cavity. This communicating type of hydrocele recurs on standing, and its rate of filling depends on the size of the communication with the peritoneal cavity. If large enough to admit bowel or omentum, a complete hernia develops. In ascites a communicating hydrocele may occur as a secondary phenomenon and is then usually bilateral. An encysted hydrocele of the cord lies within the covering of the cord and presents as an oval tense translucent swelling.

**Treatment.** Hydroceles of the tunica vaginalis occurring in infancy should be kept under observation, for many disappear gradually without any treatment. If spontaneous recovery does not occur operation is necessary. In children after the age of two years there is no natural tendency for the hydrocele to disappear. Communicating hydroceles do not close naturally at any age, and thus always need operation. Aspira-

tion is not so beneficial for hydroceles in children as in adults, for the fluid soon reforms after this procedure.

At operation, an incision is made transversely in the region of the external abdominal ring. By separation of its superficial coverings, the innermost sac lining the hydrocele is exposed and carefully dissected free from the spermatic cord proximally and as far as the margin of the epididymis distally. Care is taken not to damage the blood supply of the testicle. When the tunica vaginalis is opened the hydrocele is emptied and the whole sac wall, which has been already defined, is removed. Any oozing from the raw edge is controlled by electrocoagulation or ligation of the vessels. *In all cases, the proximal end of the hydrocele should be traced into the inguinal region and any connection with the processus vaginalis is tied and divided at the level of the internal abdominal ring; here a hernial sac should be sought for and dealt with.* In the case of an encysted hydrocele of the cord, the skin over the swelling is incised and the cyst is separated from the ensheathing layers of the spermatic cord and removed. Careful hæmostasis is necessary to avoid a hæmatoma.



FIG. 113. Hydrocele of the tunica vaginalis (left) in an infant.

#### IMPERFECT DESCENT OF THE TESTICLE

At the third month of intra-uterine life, the foetal testis lies in the iliac fossa at the brim of the pelvis. The caudal end of the testis becomes connected to the scrotum by mesenchymatous bands, which later, by becoming condensed into fibrous and muscular tissue, form the gubernaculum testis; the development of this structure is complex, being derived from components of the mesonephric and urogenital folds proximally, and the gubernacular cord and ligament of the scrotum below (Wakeley, 1953). The gubernaculum is thought to have a guiding influence on the descent of the testis. At the fourth month of foetal life evagination of the parietal peritoneum gives rise to the processus vaginalis which reaches the newly formed scrotum, lying anterior to the line of descent of the testis. During the seventh month, the gonad traverses the inguinal canal and reaches the scrotum at birth (Fig. 114). However, the exact mechanism of the migration of the testis into the scrotum is not known. The gubernaculum, which was described by John Hunter (1786), is believed to have a controlling effect, and the testis may even be misguided by accessory gubernacular bands into ectopic positions, such as the superficial inguinal, femoral or perineal regions (Lockwood,

1887); or the existence of congenital fascial pouches may impede the downward progress of the testis (McGregor, 1929). Hormones, which influence the development of the genitalia, can accelerate natural descent of the testis.

Spermatogenesis is impaired if the testis fails to reach the scrotum before puberty, but its hormone production is unaffected. The former function is thought to be influenced by the temperature of the surroundings; thus the migration of the gonads to an extra-abdominal position of a lower temperature than that of the inside of the abdomen favours normal sperm formation. There are three conditions to be considered

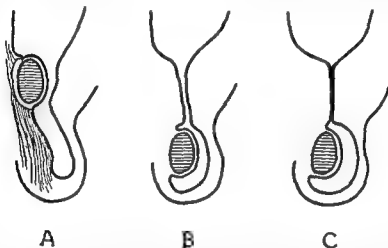


FIG. 114. Descent of the testicle. A. The testicle is lying on the posterior wall of the processus vaginalis at the level of the internal abdominal ring. The gubernaculum is represented below the testis. B. The testis has reached the scrotum, but the processus vaginalis remains patent. C. The processus vaginalis has become obliterated.

under "imperfect descent": retractile, ectopic and undescended testicle.

**Retractile Testis.** The cremasteric reflex, which is well developed in early childhood, often causes the testicle to retract into the superficial inguinal pouch when the region of the scrotum is stimulated by touch or cold. Knowledge of this fact should prevent the common error of mistaking a retractile for an incompletely descended testicle. All suspected cases should be examined in a warm atmosphere, first without touching the scrotum. The retractile testicle is commonly found in the lower inguinal region lying in the superficial inguinal pouch, and from this extra-abdominal position between the external oblique muscle and Scarpa's fascia, the testis can be coaxed into the scrotum by gentle downward pressure. No special treatment is required for the retractile testicle. The parents of the child can be reassured that the gonads will develop and function normally and will ultimately reach their natural position in the scrotum.

**Ectopic Testis.** When it is situated *outside* the line of normal descent,

the testicle is considered to be ectopic. It is commonly found lying on the surface of the external oblique muscle near the anterior superior iliac spine. Contraction of the anterior abdominal wall causes the testicle, which is usually of normal size and consistency, to become more prominent; thus its superficial position is confirmed. Less commonly, the gonad lies in the pubic, femoral or perineal position. In each instance the scrotum, which is well developed, is usually empty, although sometimes a hernia or a hydrocele may be present. The testis, lying in an unnatural position, is prevented from entering the scrotum. Therefore operation is necessary. This should be performed between the ages of six to ten years, but when an ectopic testis is associated with a hernia, earlier operation is necessary for repair of the latter. Orchidopexy, for ectopic testis, is a similar procedure to that used for the undescended testicle (*vide infra*).

**Undescended Testis.** The testicle may become arrested at any point on the path of descent into the scrotum; it may remain inside the abdomen, but more often it lies at the external abdominal ring. The condition may be unilateral or bilateral. It is usually symptomless unless complicated by injury or hernia. The empty scrotum is often ill developed (cf. ectopic testis). If lying in the intra-abdominal position, the testicle cannot be felt, but it may become evident on coughing or straining, especially if accompanied by a hernia. In other situations the elusive testis can be coaxed towards the scrotum by gentle pressure directed along the line of the inguinal canal. The frequency of undescended testicle varies with age; thus at birth about 10 per cent. of males are affected whereas between the age of seven to seventeen years the incidence is reduced to less than 2 per cent. With growth there is an increasing chance of the testis reaching the scrotum. The undescended gonad is often softer and smaller than normal. Although its internal secretion is unaffected, spermatogenesis is impaired and in bilateral cases sterility is found.

**Treatment.** As there is a natural tendency for the majority of undescended testicles ultimately to reach the scrotum, operation is postponed till the ages of eight to ten years, when those patients in whom descent has not occurred, should be treated by orchidopexy; but earlier operation is necessary if an associated hernia supervenes. A truss, in the unlikely event of its being tolerated, may be harmful. Hormone treatment (intramuscular injection of 600 I.U. of gonotrophic hormone twice a week for six weeks) is reserved for cases of bilateral cryptorchidism, in which the genitalia are poorly developed. This treatment may cause the genitalia to develop and the testes to descend.

**Orchidopexy.** Many different procedures have been devised, but the essential principles are to free the testicle adequately, and to avoid tension or pressure on the cord. The covering of the testicle is exposed by an inguinal incision and division of the external oblique muscle. The spermatic cord is dissected from its underlying anchoring fascial bands—including as much as possible of the cord internal to the internal abdominal

ring. Next, the patent processus vaginalis, which is usually present with undescended testicles, is carefully separated from the spermatic vessels and vas (lying on its deep aspect) up to the internal abdominal ring where it is tied and divided. Further lengthening of the spermatic cord is gained by stripping with the finger all adventitious bands binding the vas and testicular vessels to the parietal peritoneum on the posterior abdominal wall. The testis can then be placed in the scrotum without tension. Fixation of the gonad is best carried out by passing a long unabsorbable stitch through its distal pole and bringing out the ends of the suture through the bottom of the scrotum, after coaxing the testicle down into position. Next, the external abdominal ring is reconstituted and the wound closed. Finally the long ends of the anchor stitch are tied over a small gauze roll on the scrotal skin, and attached with slight tension to a piece of elastic strapping applied to the thigh. The fixation stitch is maintained for about ten days. The above method is the simplest, but other devices are used to keep the testicle *in situ*, such as (a) manipulation of the isolated testis through a tunnel fashioned under the firm band of fibrous tissue on the posterior wall of the neck of the scrotum where it lies anterior to the origin of pectineus (D. Browne, 1949), and (b) temporarily anchoring the freed testicle to the adjacent deep fascia of the thigh through separate incisions in the scrotal skin and thigh. When released from the thigh after an interval of ten weeks, the testicle remains in the scrotum (Kectley and Torek).

**Results.** Atrophy of the testicle occurs post-operatively in about 10 per cent. of cases, and is more liable to develop in cases when there has been tension on or over the spermatic cord. Thus the best results of orchidopexy are obtained when at operation the testicle can be made to lie easily in the scrotum. When the testicle cannot be made to reach its normal position, a second operation should be carried out some months later in order to mobilise the gonad further. The internal secretion of the testis is unaffected by malposition, but spermatogenesis is often impaired; the immature development of the spermatozoa leads to subfertility, or sterility in bilateral cases. As malignant tumours are more likely to develop in undescended testicles than those normally placed, orchidopexy, even in adult life, is advisable. The reason for this neoplastic tendency is unknown.

**Torsion of the Testis.** This is an uncommon condition, which may occur in childhood, adolescence or adult life. It usually follows a sudden strain or injury, especially if the testicle is abnormally mobile. Thus, a persistent mesorchium or incomplete descent of the testis predispose to torsion. Unless the torsion is untwisted early, congestion, gangrene and later atrophy of the testis follow. The whole testis rotates on its axis within the tunica vaginalis. The initial sudden onset of severe lower abdominal and testicular pain, which is often accompanied by vomiting and shock, slowly subsides after a few days. Pyrexia (99 to 100° F.) may occur. Within the first few hours, the scrotal skin becomes red,

shiny and oedematous, and a tense tender testicular swelling develops with thickening of the cord. Because of the accompanying hydrocele the enclosed testis and epididymis cannot be distinguished separately. If an undescended testis becomes twisted, the appearances of this tender swelling in the inguinal region closely resemble a strangulated hernia, but the scrotum is empty.

*Treatment* should be prompt in all cases, for, if delayed beyond a few hours after the onset, gangrene of the testis ensues. Occasionally the torsion untwists itself, or manipulation may succeed. In the very occasional case early operation saves the testicle. At operation, the tunica vaginalis is opened, the pedicle is untwisted and the testis is anchored to the adjacent scrotal wall with stitches. Decompression of the testis and epididymis by incising their inelastic covering may be tried in cases of doubtful viability, but if the organ is obviously gangrenous, orchidectomy should be performed, for by leaving a necrotic testicle *in situ*, wound healing and relief of the symptoms are delayed. Fixation of the normal contralateral testis, at the same operation or later, is recommended, for subsequent torsion of the other testicle is not uncommon.

**Torsion of the Appendix Testis.** Torsion of the hydatid of Morgagni—the appendix testis—presents with the same symptoms and signs as torsion of the testicle. At operation, incision of the accompanying tense hydrocele, which contains blood-stained fluid, reveals the dusky red hydatid twisted at its base, or lying detached like a purple bead at the bottom of the scrotum. The epididymis is usually secondarily inflamed and the spermatic cord is also thickened; the appendix testis, if still attached, is removed after ligation of its base. There is no danger of atrophy of the testicle.

**Orchitis.** Most painful swellings of the testicle in children are, in fact, cases of torsion of the testis or its appendix. Orchitis occurs occasionally in children as a complication of infective parotitis (mumps) or other virus diseases. In the absence of parotitis, the inflammatory condition cannot be distinguished from torsion of the testis, for the symptoms and signs are very similar, and furthermore atrophy of the testicle may occur in either condition. The inflammatory skin changes seen in both conditions may be misleading. Therefore, all patients with the above syndrome should be operated on within twenty-four hours, for the untwisted or inflamed testis can occasionally be effectively decompressed by incision of the tunica albuginea.

*Orchitis* due to congenital syphilis now rarely occurs. It presents as a painless swelling and ultimately causes atrophy of the testis. The child shows other evidence of congenital syphilis, and specific serological tests are positive.

**Infarction of the Testis,** due to thrombosis of a testicular vessel, is indistinguishable from torsion but, at operation, the absence of torsion suggests the diagnosis. The condition is rare.



## CHAPTER 15

### ABNORMALITIES OF THE ANTERIOR ABDOMINAL WALL

**Development of the Abdominal Wall.** The ventrolateral walls of the abdomen are derived from the somatopleure\* which first becomes segmented and later develops myotomes which give rise to the various muscle layers; these spread forwards towards the midline and blend with the margins of the umbilical ring which, at the third week of intra-uterine life, occupies most of the ventral aspect of the abdomen. Above and below this central opening the lateral muscle layers fuse with a median band of mesoblastic tissue to form the supra- and infra-umbilical parts of the linea alba. As the embryo elongates the umbilical ring alters little in size; thus a relatively small part of the midline of the abdomen is eventually occupied by the umbilicus. The umbilical cord of the early embryo contains the vitello-enteric canal and the allantois, both of which become obliterated just before birth when the development of the alimentary and urinary tract is completed. The umbilicus becomes sealed by scar tissue after the separation of cord.

**Umbilical Hernia.** This common deformity, which appears as a protrusion of the umbilicus of variable size, is easily recognised at birth. It becomes tense with crying and straining. Occasionally the presence of bowel or omentum in the hernial sac gives rise to bouts of pain and vomiting, but the contents are usually easily reducible. When the rupture is indented by the finger the underlying gap in the umbilical ring can be felt. In a *true* umbilical hernia as defined above, this opening is round (Fig. 115), whereas in the *supra-umbilical* or *semi-umbilical* type, there is also a gap in the linea alba in the upper margin of the umbilical ring (Browne, 1952) (Fig. 116). It is important to distinguish between these two varieties of hernia, for their prognosis and treatment differ. The true umbilical hernia has a great tendency to spontaneous cure as the result of gradual contraction of the umbilical opening, whereas the semi-umbilical hernia needs operative repair. Natural cure of the former type can be assisted by the application of waterproof adhesive strapping over the hernia when reduced. This support should be applied to the infant soon after birth and maintained for several weeks to be effective. When such a hernia has been present for several months it is unlikely to benefit from this treatment, but if left unsupported the hernia may still disappear with development of the abdominal muscles. Conservative treatment may also be tried for the *semi-umbilical* type of hernia, but as complete

\* Somatopleure = layer formed by union of the somatic mesoblast and epiblast.

closure is unlikely, operation is usually necessary. Abdominal belts have little or no curative value but may be used pending operation (when the protrusion is large enough to alarm the parents). A flat rubber type of support with adjustable studs is usually employed. The measurement of the girth at the umbilical level is all that is required when the appliance is ordered.

Operative treatment is necessary for all umbilical hernias when spontaneous closure of the opening does not occur. Thus almost all semi-umbilical hernias need operative repair; it is best carried out between the ages of two to three years. In persistent true umbilical hernias, operation may be postponed for several years, in the hope that natural contracture of the opening may still occur. It is especially important in girls that the umbilical ring should be repaired, for subsequent childbirth causes the opening to enlarge and in later years there is the risk of strangulation which is particularly common in elderly fat multiparous women.



FIG. 115. A true umbilical hernia in an infant.



FIG. 116. A semi-umbilical hernia in an infant.

*Operation* (Fig. 117). A curved incision is made along the lower margin of the umbilicus and the inferior aspect of the hernia is exposed. The fundus of the sac is opened; the adherent umbilical skin is reflected and the rest of the hernia is defined. An elliptical incision is then made around its base through the linea alba without damaging the peritoneum. After reduction of the contents of the hernia—commonly a piece of slightly adherent omentum—the neck of the sac is transfixed and ligated with chromicised catgut or thread and the sac removed. Next, the edges of the linea alba are sewn together transversely with several mattress sutures. In closure of the skin, the centre of umbilicus is anchored with a stitch

to preserve the normal appearance of the umbilicus. Recurrence of the hernia after operation is rare and the scar is barely visible.

**Epigastric Hernia.** This type of hernia may occur in children between the ages of six to ten years. It is not a true hernia for there is a protrusion of fatty tissue only through a gap in the supra-umbilical part of the linea alba with no peritoneal sac. There is a small, soft and sometimes tender swelling in the midline of the epigastrium. Occasionally operation is carried out for pain; the fatty swelling is removed and the gap in the linea alba closed.

**Exomphalos (Fig. 118)** is a rare condition seen in new-born infants. The umbilical ring has remained patent at birth as a large deficiency in the anterior abdominal wall. Abdominal organs protrude through the opening covered by a thin opalescent membrane. Coils of intestine and the liver can usually be seen. The umbilical cord, with its two arteries and vein, is attached to the surface of the sac. Often the covering is torn and the abdominal organs are exposed. There is then a serious risk of peritonitis. Other congenital abnormalities are commonly present. Pending urgent operation, the omphocele is covered with a gauze pack thoroughly moistened with saline and bound to the abdomen to prevent further prolapse. Success of the operation depends to a great extent on the size of the abdominal deficiency. The stomach is first deflated by means of a catheter passed through

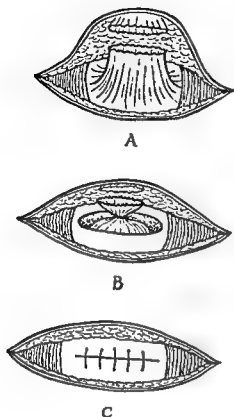


FIG. 117. Repair of umbilical hernia. A. The sac has been defined and separated from the under surface of the umbilicus. B. The linea alba has been divided around the sac which is ligated at its neck. C. The stump of the ligated sac has been buried and the linea alba repaired.

the mouth. The edges of the opening are defined by cutting along the junction of the skin and the membranous sac. The abdominal organs are carefully examined for any associated intestinal abnormality, such as atresia or malrotation of the gut. These abnormalities are treated first (see under Congenital Defects of the Jejunum, Ileum and Colon). The extruded organs are then placed in the abdomen—often with great difficulty—and the edges of the opening are brought together, if possible, by two layers of continuous sutures, one through the peritoneum and the other through the fascial layer. If the exomphalos is large and it is impossible to close the deep layers around the gap, the membranous covering is left intact and extensive under-cutting of the surrounding skin

is carried out to cover the protrusion (Fig. 119). A secondary repair of the deep layers is carried out after an interval of several months. By then the gap is relatively smaller because of the growth of the child (Gross, 1953). The mortality of this serious deformity is decreasing with prompt treatment. The prognosis is bad when the opening is very large, when there are associated anomalies of the gut or the bladder, or when repair is unduly delayed.

**Agenesis of the Abdominal Muscles.** This is very rare. It results from the failure of development of the myotomes. The abdomen is covered with flaccid skin through which the outline of the abdominal organs can be seen and easily felt. Co-existent anomalies of the urinary tract, such as hydronephrosis and hydroureters, are common. These conditions are



FIG. 118. A case of exomphalos which was successfully repaired.

usually secondary to obstruction by a urethral valve. There is no treatment for agenesis of the abdominal muscles. Some of the affected children survive for a few years before dying of intercurrent infection. The prognosis is much worse when there are associated anomalies of the urinary tract.

**Discharge from the Umbilicus.** A "moist" navel occasionally occurs a few days after birth, and is caused by infection at the line of separation of the umbilical cord. Its occurrence indicates lack of proper care of the umbilical area at birth. It may lead to severe neonatal infection. It is avoided by keeping the cord dry and clean. Established infection should be promptly treated by antibiotics appropriate to the causal organism. Chronic infection is associated with the formation of granulation tissue and polypi, and it gives rise to a persistent purulent or blood-stained discharge. The best treatment is cauterisation of the granulomatous area or removal of any polypus (Fig. 120). An intractable umbilical discharge may come from a patent urachus or a persistent vitello-enteric duct. The fluid from the former is urine, whereas that from the latter is mucoid or faecal. The introduction of a probe or lipiodol into the sinus,



FIG. 119. The appearance after skin closure only in a case of exomphalos. The outline of the rectus abdominal muscle is well formed on both sides of the bulge; the central area of skin was adherent to the liver, but a sound secondary repair was possible.

at the base of the bladder with a gaping urethral gutter (Fig. 121). This deformity is found in one in fifty thousand births and affects males predominantly. The sensitive bladder mucosa lies exposed on the lower anterior abdominal wall (Fig. 122). Urine is seen flowing over its surface after emerging from the pouting ureteric orifices. Crying and straining increases the protrusion. When the end of the penis is drawn downward the floor of the urethra can be seen. The infant is constantly wet. Epispadias may also occur with most of the bladder wall intact. At the level of the symphysis pubis an orifice opening into the bladder is visible, revealing the bladder neck and the floor of the urethra. As in ectopia vesicæ, there is no control of the urine.

Associated defects, which commonly occur with the above two conditions, are separation of the symphysis pubis with divarication of the rectus abdominis muscles, and laxity of the anal sphincter with prolapse of the rectum. In the male the cleft penis, which is usually upturned, is ill-developed, and bilateral inguinal hernias or cryptorchidism

followed by X-ray examination, shows whether the fistula communicates with the bladder or the intestine. Occasionally a small fistula heals spontaneously, but usually total excision of the fistulous tract from the umbilicus to the affected viscus is necessary. (See also under Meckel's diverticulum.)

Rarely, an area of heterotopic gastric mucosa, causing a continuous acid secretion, may be found at the umbilicus. It should be excised.

**Ectopia Vesicæ and Epispadias.** These two conditions are nearly always associated and are the result of failure of developmental closure of the infra-umbilical part of the abdominal wall, bladder and roof of the urethra. The deficiency can extend from the umbilicus above to the end of the urethra below, but more often it occurs as an anterior opening



FIG. 120. A granulomatous polyp at the umbilicus caused by chronic infection.

are found. In females, the clitoris is bifid and the urethral groove is visible between the two halves.

In most cases, the defect is obvious on external examination, but minor degrees of the deformity, especially in girls, may escape notice, as the cleft may lie buried in the pubic region. All affected patients are incontinent of urine. The constant soaking of the clothes and soreness of the skin makes the patients miserable and reclusive. Some have little control of feces. Those with a widely separated symphysis pubis walk with a waddling gait.

**Treatment.** In the first few months of life little can be done except to keep the child as dry as possible with frequent changes of absorbent dressings, and to protect the skin with barrier cream\* and regulate the fluid intake. Appliances are of little value and are not tolerated owing to the sensitivity of the bladder mucosa, which is often inflamed and ulcerated.



FIG. 122. A case of ectopia vesicae; the posterior bladder wall is seen bulging from the lower abdomen. Its upper aspect has become partially covered with squamous epithelium. The pointing ureteric orifices can be seen below. The umbilicus is absent. The child was successfully treated by transplantation of the ureters into the pelvic colon followed by removal of the bladder wall and repair of the abdominal wall defect.

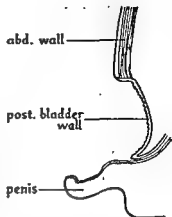


FIG. 121. A diagram of ectopia vesicae. A ureter can be seen entering the lower posterior margin of the bladder wall. (After Keith.)

Reconstructive operations rarely produce a good functioning bladder so that lack of control of urine persists. Attempts to reform the bladder sphincter rarely succeed. Nevertheless, repair is worth trying for minor degrees of epispadias. The muscle of the bladder-neck sphincter is reconstructed over a catheter and the penile deformity, which is difficult to correct, is best dealt with by using the principle of the buried strip method (see under Hypospadias). When reconstructive operations fail, or when they are not indicated because of the extent of the deformity, the urinary flow may be diverted. Provided rectal control is not impaired, bilateral transplantation of the ureters into the sigmoid colon, followed by removal of the bladder and closure of the abdominal defect, is

\* A barrier cream is one which contains water-repellant substances, e.g. silicone.

performed. It is safer to transplant one ureter on one occasion and the other two to three weeks later. Nowadays, the operation is being performed earlier than formerly and, provided the child's general condition is good, the operation is best carried out between the ages of eighteen months and two years. The bladder wall is removed later when convenient. Following the diversion of urine into the bowel, control of the fluid rectal contents is acquired in a few weeks. The child is then able to hold urine for a few hours and can therefore lead a happier and fuller life. With improved technique and better means of treating urinary infections the operative mortality has diminished to about 10 per cent. The majority of patients who survive operation reach adult life but there is a tendency to recurrent urinary infection, which, however, can usually be controlled by drugs.

## CHAPTER 16

### CLEFT LIP AND CLEFT PALATE

THESE deformities develop during the first six to eight weeks of intra-uterine life, and are the result of failure of fusion of the fronto-nasal and maxillary processes of the developing embryo. The fronto-nasal element gives rise to five processes (Fig. 123A) : two lateral nasal processes and one median nasal process, eventually forming the sides and the septal portion of the nose respectively, and two globular processes which form the philtrum of the lip and premaxilla (Fig. 123). The maxillary processes develop into the cheeks and lateral portions of the upper lip, the floor of the nose, the sides of the hard palate and most of the upper jaw (Fig. 123C and D).

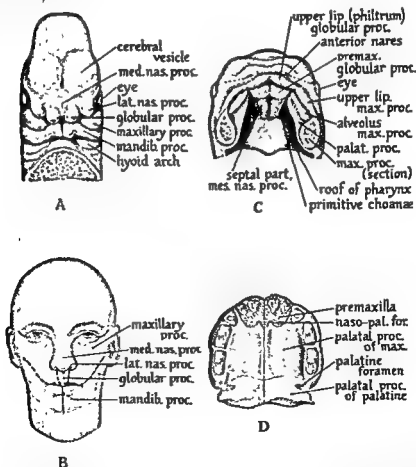


FIG. 123. A. Processes which form the face at the sixth week. B. Lines of fusion in the formation of the face. C. The processes involved in the formation of the upper lip and the roof of the mouth. D. The hard palate at birth. (After Keith.)



**Incidence.** Haro lip and cleft palate occur in about 1 in 700 births (Corsin, 1950) and predominantly affect males more than females in the proportion of three to two. Almost half of the affected infants have defects of the palate and the lip. The remainder have defects of one or the other alone, in about equal proportions. In about a third of the cases, the deformity is inherited either as a Mendelian recessive or dominant. The chances of normal parents having a second child with a cleft deformity is about 5 per cent., and the risk of parents, one of whom has



FIG. 124. Pre-alveolar cleft, Group Ia. (Mr. R. J. V. Battle's case.)



FIG. 125. Bilateral pre-alveolar cleft, Group Ic. (Mr. R. J. V. Battle's case.)

a cleft palate or lip, having a similarly affected child is 2 per cent. (Fogh Anderson, 1942).

**Types of Deformity.** Cleft lip and cleft palate are best considered together in relation to the alveolus. The classification suggested by Davis and Ritchie (1922) is used.

*Group I*  
*Pre-alveolar Cleft*

- (a) Unilateral
- (b) Median (rare)
- (c) Bilateral

*Group II*  
*Post-alveolar Cleft*

- (a) Soft palate only
- (b) Soft and hard palate
- (c) Submucous cleft

*Group III*  
*Alveolar Cleft*

- (a) Unilateral
- (b) Median
- (c) Bilateral

**Group I—Pre-alveolar Cleft.** This involves the lip elements only and may be a simple notch in the mobile part of the upper lip, or a more complicated cleft extending upwards involving the floor of the nose. The

nostril is generally wide and flattened on the affected side (Fig. 124). The underlying alveolus is usually intact but may be irregularly joined. The unilateral type (a) may affect either side. The median cleft (b) is rare and is due to failure of development of the median nasal process (Fig. 123A); therefore the philtrum is absent. In the bilateral type (c) the

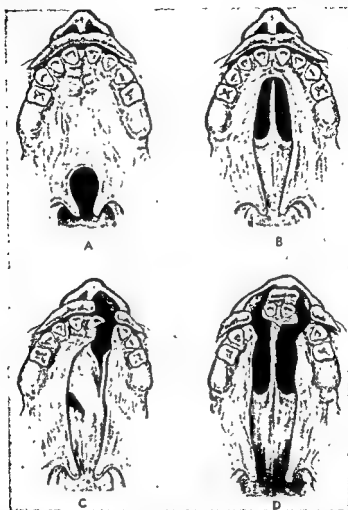


FIG. 126. *Types of deformity of palate and lip.* A. Post-alveolar cleft (soft palate only), Group IIa. B. Post-alveolar cleft (soft and hard palate), Group IIb. C. Alveolar cleft, unilateral, Group IIIa. D. Alveolar cleft, bilateral, Group IIIc. (Oldfield, *Brit. Journ. Surg.*, 1949.)

philtrum is present but has failed to fuse with the lateral elements of the lip (Fig. 125). A post-alveolar cleft may coexist with all these types.

**Group II—Post-alveolar Cleft.** In this group there is a failure of fusion of the palatal elements of the maxillary process behind the premaxilla. Thus the median cleft may involve the soft palate only, type (a) (Fig. 126A), or extend through the hard palate, type (b) (Fig. 126B). The

**Incidence.** Hare lip and cleft palate occur in about 1 in 700 births (Corsin, 1950) and predominantly affect males more than females in the proportion of three to two. Almost half of the affected infants have defects of the palate and the lip. The remainder have defects of one or the other alone, in about equal proportions. In about a third of the cases, the deformity is inherited either as a Mendelian recessive or dominant. The chances of normal parents having a second child with a cleft deformity is about 5 per cent., and the risk of parents, one of whom has



FIG. 124. Pre-alveolar cleft, Group Ia.  
(Mr. R. J. V. Battle's case.)



FIG. 125. Bilateral pre-alveolar cleft,  
Group Ic. (Mr. R. J. V. Battle's case.)

a cleft palate or lip, having a similarly affected child is 2 per cent. (Fogh Anderson, 1942).

**Types of Deformity.** Cleft lip and cleft palate are best considered together in relation to the alveolus. The classification suggested by Davis and Ritchie (1922) is used.

Group I Pre-alveolar Cleft	Group II Post-alveolar Cleft	Group III Alveolar Cleft
(a) Unilateral	(a) Soft palate only	(a) Unilateral
(b) Median (rare)	(b) Soft and hard palate	(b) Median
(c) Bilateral	(c) Submucous cleft	(c) Bilateral

**Group I—Pre-alveolar Cleft.** This involves the lip elements only and may be a simple notch in the mobile part of the upper lip, or a more complicated cleft extending upwards involving the floor of the nose. The

when the infant is three months old, or when it has reached the weight of ten pounds (4.6 kilos). Early closure not only facilitates bottle feeding but tends to correct any underlying malformation of the alveolus. The age of twelve to eighteen months, before speech develops, is the optimum time for repair of the palate, providing the infant is otherwise healthy and not anæmic. Infected tonsils and carious teeth are removed before this operation. Search for the best method of closure of the defect has exercised the ingenuity of surgeons for generations. Many methods have

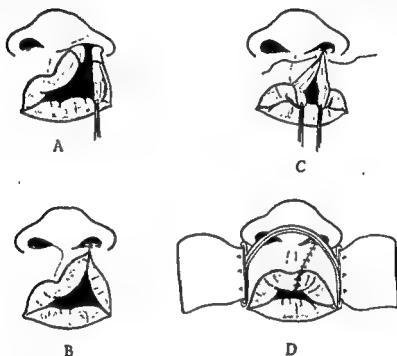


FIG. 129. Repair of unilateral pre-alveolar cleft. (After Rose.) A. and B. Repair of nasal floor. C. Incisions preparing lip for suture. D. Suture complete and Logan's bow (strapped to the face) in position.

contributed to the present-day procedure. The aim, in addition to correction of the deformities, is to produce a mobile lip and a mobile soft palate, in order to obtain the best functional results. Only methods in common use are described here.

*Pre-alveolar Cleft—Group I.* Unilateral hare lip (type *a*) is repaired by simple closure (Fig. 129). The nasal side of the cleft is freed from the underlying bone; the edges are cut; the floor of the nostril is reconstructed to look symmetrical. Then the labial flaps are fashioned and approximated so that the red margin of the lip is exactly in line (Fig. 129). The median cleft (type *b*) is similarly treated. For a bilateral cleft (type *c*) the lip is mobilised and repaired in the manner shown in Fig. 130. After all these operations on the lip a modified Logan's bow is applied to

submucous variety is rare ; in this deformity the soft tissue of the palate, being intact, bridges the gap in the hard palate.

**Group III—Alveolar Cleft.** This results from failure of union of the premaxillary and maxillary parts of the alveolus and is associated with a pre-alveolar and/or post-alveolar cleft (Fig. 126C and D). Unilateral types (a) appear to one side of the midline (Figs. 126C and 127) ; the nasal septum is joined to only one side of the cleft. The median type (b) is rare. The premaxilla fails to develop leaving an anterior gap in which the nasal septum lies free. In the bilateral type (c) the premaxilla is



FIG. 127. Alveolar cleft, Group IIIa.  
(Mr. R. J. V. Battle's case.)



FIG. 128. Alveolar cleft, Group IIIc.  
(Mr. R. J. V. Battle's case.)

isolated, protruding forward in the midline from the anterior end of the vomerine portion of the nasal septum (Figs. 126D and 128). The buccal-gingival sulcus is absent, for the central isolated part of the lip is adherent to the bony protrusion (Figs. 126D and 128).

**Clinical Manifestations.** The presence of a pre-alveolar cleft is obvious at birth. The other types are seen on examination of the mouth. Feeding may be difficult, for milk regurgitates into the nose and may then be aspirated and cause pneumonia. Infants with cleft deformities are liable to upper respiratory infections. A small proportion of them are mentally retarded and may have other congenital abnormalities. If clefts are left untreated speech defects occur and dental development is deranged.

**Management.** Breast feeding should be tried, but, except in the case of minor clefts, it may be impossible. Feeds of expressed breast milk are then given from a bottle with a rubber teat, with the child in the upright position. The baby's power of suction is poor ; therefore feeding is slow and requires much patience. Operative repair of the lip is carried out

when the infant is three months old, or when it has reached the weight of ten pounds (4.6 kilos). Early closure not only facilitates bottle feeding but tends to correct any underlying malformation of the alveolus. The age of twelve to eighteen months, before speech develops, is the optimum time for repair of the palate, providing the infant is otherwise healthy and not anæmic. Infected tonsils and carious teeth are removed before this operation. Search for the best method of closure of the defect has exercised the ingenuity of surgeons for generations. Many methods have

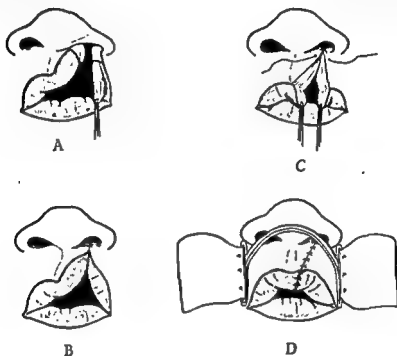


FIG. 129. *Repair of unilateral pre-alveolar cleft. (After Rose.)* A. and B. Repair of nasal floor. C. Incisions preparing lip for suture. D. Suture complete and Logan's bow (strapped to the face) in position.

contributed to the present-day procedure. The aim, in addition to correction of the deformities, is to produce a mobile lip and a mobile soft palate, in order to obtain the best functional results. Only methods in common use are described here.

**Pre-alveolar Cleft—Group I.** Unilateral hare lip (type *a*) is repaired by simple closure (Fig. 129). The nasal side of the cleft is freed from the underlying bone; the edges are cut; the floor of the nostril is reconstructed to look symmetrical. Then the labial flaps are fashioned and approximated so that the red margin of the lip is exactly in line (Fig. 129). The median cleft (type *b*) is similarly treated. For a bilateral cleft (type *c*) the lip is mobilised and repaired in the manner shown in Fig. 130. After all these operations on the lip a modified Logan's bow is applied to

submucous variety is rare ; in this deformity the soft tissue of the palate, being intact, bridges the gap in the hard palate.

**Group III—Alveolar Cleft.** This results from failure of union of the premaxillary and maxillary parts of the alveolus and is associated with a pre-alveolar and/or post-alveolar cleft (Fig. 126C and D). Unilateral types (a) appear to one side of the midline (Figs. 126C and 127) ; the nasal septum is joined to only one side of the cleft. The median type (b) is rare. The premaxilla fails to develop leaving an anterior gap in which the nasal septum lies free. In the bilateral type (c) the premaxilla is



FIG. 127. Alveolar cleft, Group IIIa.  
(Mr. R. J. V. Battle's case.)



FIG. 128. Alveolar cleft, Group IIIc.  
(Mr. R. J. V. Battle's case.)

isolated, protruding forward in the midline from the anterior end of the vomerine portion of the nasal septum (Figs. 126D and 128). The buccal-lingual sulcus is absent, for the central isolated part of the lip is adherent to the bony protrusion (Figs. 126D and 128).

**Clinical Manifestations.** The presence of a pre-alveolar cleft is obvious at birth. The other types are seen on examination of the mouth. Feeding may be difficult, for milk regurgitates into the nose and may then be aspirated and cause pneumonia. Infants with cleft deformities are liable to upper respiratory infections. A small proportion of them are mentally retarded and may have other congenital abnormalities. If clefts are left untreated speech defects occur and dental development is deranged.

**Management.** Breast feeding should be tried, but, except in the case of minor clefts, it may be impossible. Feeds of expressed breast milk are then given from a bottle with a rubber teat, with the child in the upright position. The baby's power of suction is poor ; therefore feeding is slow and requires much patience. Operative repair of the lip is carried out

opposite the canine tooth back as far as the pterygomandibular ligament. (This ligament, which can be felt by the finger introduced into the mouth, extends from the pterygoid hamulus to the posterior part of the mylohyoid line on the medial aspect of the mandible.) The medial limb of the V runs obliquely back to reach the margin of the cleft in the hard palate (Fig.

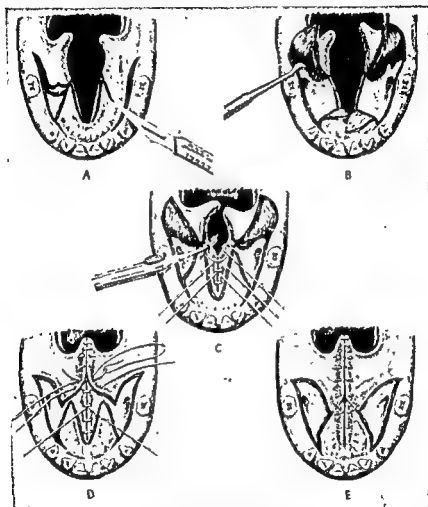


FIG. 131. Repair of cleft palate by the four-flap V-Y procedure. A. The palatal incisions through mucoperiosteum. B. Raised flaps. C. Suturing the nasal mucosa. D. Suturing the oral palatal flaps. E. Completion of repair. The dotted line indicates the site of the cleft. (Wardill, *Brit. Journ. Surg.*, 1937.)

131A). The edges of the cleft are trimmed. The anterior and posterior buccal flaps are raised (Fig. 131B), and the latter are separated with scissors from the posterior margin of the hard palate (Fig. 131B). Further relaxation of the soft palate is obtained by freeing the lateral pharyngeal wall on each side and fracturing the hamular processes, thereby slackening the tensor palati muscles. Lateral mucosal flaps are elevated on the



allow healing without tension. No skin dressing is required, but any nasal discharge is soaked up by a pledget of cotton-wool placed in the nostrils and changed frequently. The arms are splinted to prevent the infant interfering with the wound.



FIG. 130. Repair of double hare-lip. A. Stitching of the floor of the nostril. B. Incisions in the lip. C. Insertion of crucial stitch halfway along the side of the globular process and through the mucocutaneous junction. D. This stitch tied on both sides fashions Cupid's bow. (From D. Broune, 1949, *Annals of Royal Coll. Surgs. Eng.*)

*Post-alveolar Cleft—Group II.* Sub-groups (a) and (b) (clefts of soft and/or hard palate) are considered together. The Veau-Wardill (1937) type of operation is the most popular; it is a four-flap procedure designed to relax tension on the median suture line and to produce a mobile soft palate which, after pharyngoplasty, closes the nasopharyngeal isthmus. A V-shaped incision is made in the hard palate on both sides (Fig. 131A). The lateral limb of the V extends along the alveolar border from a point

Gillies-Fry type can be used. The soft palate is repaired at the expense of enlarging the opening in it in front. Subsequently the gap is closed with an obturator. By this method a fairly mobile soft palate is obtained posteriorly. This procedure is now rarely performed.

Infants stand operations for cleft palate well. Occasional blood transfusion is necessary if bleeding has been troublesome. Otherwise no special after-treatment is required, for soon after the operation the child is able to swallow soft foods. The mortality of the operation is negligible.

**Speech Training.** If operation for cleft palate is performed before the age of two years phonation is little impaired. A co-operative mother is the best teacher for slight speech defects; from her the intelligent child can imitate correct articulation. When operation is delayed, speech has to be corrected by long training carried out by a speech therapist. If there is any malformation of the alveolus and teeth constant orthodontic care is required, and in some cases corrective intra-oral moulds are necessary.

**Results.** Investigations of speech function after cleft palate operations reveal that those performed before the age of eighteen months give the best results; 90 per cent. of children so treated are able to talk well; just under a half of these have only minor speech defects.

nasal (superior) aspect of the margin of the cleft and closed in a continuous layer (Fig. 131C). The medial sides of the soft palate are now sutured, beginning at the uvula and continuing forward along the posterior buccal flap. This causes the velum palati to fall back towards the posterior wall of the nasopharynx (Fig. 131D). Further narrowing of the nasopharyngeal isthmus can be obtained, if desired, either by means of pharyngoplasty (Wardill) or a purse-string suture (Denis Browne). In the former a *transverse* opening is made along the line of Passavant's ridge\* on the posterior pharyngeal wall and closed *longitudinally*. In the latter a continuous stitch is inserted encircling the isthmus at the same level and tied. Thereby the muscles of the palate are splinted during healing and the nasopharynx is narrowed.

*Alveolar Cleft—Group III.* A unilateral alveolar cleft (*a*) is usually combined with a hare lip or cleft palate which need surgical treatment as outlined above. In some cases repair of the lip assists in correcting the malalignment of the underlying alveolus. In many cases, however, there is gross malposition of the ridge and irregular development of the teeth. In this condition, an adjustable palatal mould is of great help (the orthodontist is playing an increasingly important role in this branch of surgery). The rare median cleft (type *b*) is treated similarly. A bilateral cleft (type *c*) requires surgical and orthodontic treatment. The two-stage operation, as described by Denis Browne (1949), is recommended. At the first stage, performed when the infant is three months old, the lip is raised from the underlying protruding premaxilla, and the "prevomerine bone" † is resected to allow the premaxilla to be set back almost in line with the gap in the alveolar ridge. Here it is held in place by a spiked bar applied to its anterior surface. The adjacent margins of the mucosa of the hard palate are refashioned over the replaced bone. The spiked bar is removed after two weeks. The second stage is performed a month later, and consists of freeing the cheek and alæ nasi in the region of the cleft from the underlying bone, and closing the wide gap of the nasal floor on each side. This is done by sewing the raw edge of the lip, just to the outer side of the nostril, to a corresponding raw edge on the nasal septum just inside the nose (Fig. 130A). Next, the margins of the central part of the lip are cut to a V shape (Fig. 130B). The sides of the central part are joined to the edges of the lateral part of the lip after turning down equal lengths of red margin. The ends of the latter are then joined to complete the shape of a Cupid's bow (Fig. 130C and D). Logan's bow is then applied (Fig. 129D). When there has been much delay in carrying out the repair of the palatal defect, or operation has been followed by disruption of the sutures—a rare occurrence—secondary repair of the

\* Passavant's ridge is produced by the sphincteric fibres of the palatopharyngeal muscle; contraction of this specialised portion of the superior constrictor normally assists in closure of the nasopharynx.

† The "prevomerine bone" is that part of the newly formed bone which develops with this deformity between the premaxilla and the vomer.

held up in the upper blind segment, usually at 10 to 11 cm. from the alveolar margin. The position of the tube can be shown radiographically (Fig. 133). It is unwise to outline the upper blind segment with radio-opaque fluid, as practised formerly, for there is the risk of its overflowing into the lungs and causing further respiratory embarrassment. When the condition is suspected, oral feeding is strictly withheld, for otherwise there is a grave risk of the baby being drowned by its first feed. The blind

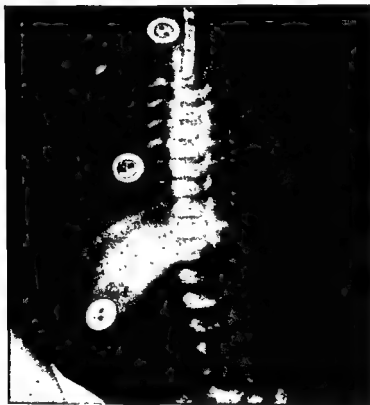


FIG. 133. Showing an oblique view of an opaque rubber catheter held up in the blind segment. Gas in the stomach and the intestine indicates that there is an oesophageotracheal fistula in the lower segment of the oesophagus.

pouch is kept empty by suction through an indwelling soft rubber catheter. Occasionally the recurring attacks of cyanosis lead to the mistaken diagnosis of congenital heart disease or a diaphragmatic hernia. All cases of oesophageal atresia require early operation. It is performed under general endotracheal anaesthesia with the infant lying on its left side. The right pleural cavity is opened along the line of the fourth intercostal space. After forward retraction of the lung the pleura in the region of the arch of the vena azagosa is incised and this vessel, which corresponds to the level of the deformity, is divided between ligatures. The upper oesophageal pouch and the communication between the

## CHAPTER 17

# CONGENITAL ABNORMALITIES OF THE ŒSOPHAGUS AND DIAPHRAGM

## THE ŒSOPHAGUS

**Congenital Atresia.** The œsophagus, which is that part of the foregut connecting the primitive pharynx to the gastric dilatation, gradually lengthens with the development of the lungs and the consequent recession of the diaphragm. Two lateral ridges appear in this primitive tube and ultimately, by fusion in the midline, separate the trachea from the œsophagus. At about the sixth week of intra-uterine life this division has been completed. If the plane of fusion is oblique the upper part of the œsophagus becomes divided from the lower part which remains in communication with the trachea. Thus arises the commonest anomaly in this region (R. H. Franklin, 1952). Vogt (1929) classified the various abnormalities of the œsophagus into three types, namely :

- (1) Absence of the œsophagus.
- (2) Atresia of the œsophagus with the upper and lower segments ending as blind pouches.
- (3) Atresia of the œsophagus with tracheo-œsophageal fistula : (a) With the fistula between the upper segment and the trachea ; (b) with the fistula between the lower segment and the trachea ; (c) with the fistula between both segments and the trachea. Type 3 is the commonest of these deformities, comprising about 80 per cent.

of all the types (Fig. 132). It is estimated that the incidence of congenital atresia of the œsophagus occurs in 1 in 1,500 births.

**Diagnosis and Management.** This condition must be diagnosed very soon after birth. The upper blind pouch becoming filled with saliva overflows and causes respiratory embarrassment and finally choking. The lower segment of the œsophagus, being in communication with the trachea, allows inspired air to fill the gastro-intestinal tract. Failure to appreciate this fact frequently accounts for delay in the diagnosis, for it is thought that the intestinal air must have come through a normally patent œsophagus ; œsophageal atresia is demonstrated by inability to pass a small rubber catheter into the stomach. An œsophageal tube is



FIG. 132. Showing type III deformity of congenital atresia of the œsophagus with tracheo-œsophageal fistula.

is frequently blood-stained and the baby fails to thrive. The symptoms may be slight at first but, if later they become severe, ulceration and stricture formation may develop at the lower end of the Œsophagus. Examination of the infant shows some degree of emaciation and anæmia. Visible gastric peristalsis similar to that seen in congenital pyloric stenosis may also be evident. Radiologically, there is great variation in the pattern of the cardio-Œsophageal junction in infants, and expert judgement is required to detect abnormal reflux and its associated changes. Barium sulphate is mixed with a feed and its progress is observed. In reflux Œsophagitis, when the stomach is full, regurgitation occurs freely on inspiration and, in some cases, there is rigidity of the wall of the Œsophagus, increased peristalsis, widening of the Œsophageal lumen or irregularity of the mucosa. These appearances, which may occur singly or together, indicate the presence of Œsophagitis; pressure on the full stomach demonstrates herniation at the hiatus of the diaphragm. In doubtful cases emptying of the stomach through the pylorus should be observed to exclude stenosis or spasm in this region, for reflux Œsophagitis clinically resembles congenital pyloric stenosis and furthermore the symptoms of the former condition may possibly be initiated by pyloric spasm or a pyloric "tumour" (syn. hypertrophic pyloric stenosis). Œsophagoscopy reveals laxity of the cardia and reflux; the mucosa may be ulcerated.



FIG. 134. Showing infant suffering from reflux Œsophagitis being treated in the upright position. (Courtesy of Dr. Helen Makay.)

*Treatment.* Reflux Œsophagitis in infancy due to a lax cardia is treated conservatively by feeding the baby in a semi-upright position (Fig. 134). The feeds are thickened and given frequently. Good nursing care is of prime importance, as feeding is often slow and tedious. When ulceration of the Œsophagus is present an anti-acid mixture is given. The majority of cases respond well to medical measures, and the symptoms tend to disappear when the child begins to walk. A very small proportion of patients require operation because of stricture formation (assessed periodically by X-ray examination). A sliding hernia does not respond very well to conservative treatment, therefore operation on similar lines to that used in adults is employed (see Chap. 43). Operative treatment is

trachea and lower segment of the œsophagus are both freed by gentle dissection. After ligation and division of the fistulous track the continuity of the œsophagus is restored by end-to-end anastomosis without tension. If the two parts of the œsophagus are widely separated, the lower end, and if necessary the stomach, are mobilised by enlarging the diaphragmatic hiatus. Interrupted sutures of fine silk are used for the anastomosis; after stitching the posterior layer the œsophageal catheter is passed down into the distal end so that the junction can be completed over this tube. If the gap between the upper and lower segments is too wide to bridge, the blind end above is exteriorised to the side of the neck, and in addition a gastrostomy is performed, so that a restorative reconstruction can be carried out later on similar lines to that adopted in adults. After closure of the chest wound the lung is kept inflated by means of an under-water closed intercostal drainage system. Immediately after operation hydration is maintained by intravenous therapy, and fluids are given by mouth after forty-eight hours. The infant is nursed in an oxygen cradle and the airway is kept clear by suction. Since 1941, when the first successful primary anastomosis for congenital œsophageal atresia was done (Haight and Townsley, 1943), the survival rate after operation has steadily increased; at special centres it has reached as high as 75 per cent. The early recognition of the condition before pneumonia and dehydration have occurred is vital. Other congenital abnormalities, such as malformation of the heart or rectum, occur in 1 in 10 of these cases. Therefore the possible coexistence of an imperforate anus warrants a careful rectal examination in every case.

**Reflux Œsophagitis in Infancy and Childhood.** According to Forshall (1954), approximately one in every thousand infants has reflux œsophagitis. Without treatment there is a risk of permanent damage to the lower end of the œsophagus. The presence of gastric juice in the œsophagus can be due to (a) a lax opening at the cardiac end of the stomach (below the diaphragm), or (b) to a sliding hernia in which the cardia lies above the diaphragmatic opening. The nature of these lesions corresponds to that occurring in adults, but, in infancy, pyloric spasm may further aggravate the reflux of gastric contents. Persistent regurgitation of acid leads to ulceration, scarring, and subsequent narrowing of the lumen of the lower end of the œsophagus; in some cases inflammation outside the lumen may occur. Marked peri-œsophagitis produces thickening of the wall of the gullet which becomes fixed to the surrounding structures. Shortening of the œsophagus by cicatrisation may also develop; if this occurs *in utero* the so-called congenital short œsophagus may result (Waterston, 1954). Rarely gastric mucosa may extend upwards into the œsophagus for a variable distance above the diaphragmatic opening and give rise to symptoms of reflux œsophagitis.

*Symptoms* may occur at any age, but often appear in the first few days of life. There is regurgitation of the feeds, and vomiting. The vomitus

## CHAPTER 18

### THE STOMACH, DUODENUM AND BILIARY TRACT IN CHILDREN

**Infantile Hypertrophic Pyloric Stenosis.** It is estimated that this lesion occurs in 1 in 250 infants and affects males more than females in the proportion of 4 to 1. Although there is a familial tendency, and about half the affected infants are first-born, the genetic mechanism is uncertain. The term "infantile" is more accurate than "congenital," for



FIG. 135. A specimen of infantile hypertrophic pyloric stenosis. The pyloric canal is narrowed and surrounded by marked thickening of the muscular wall which blends proximally with the hypertrophied wall of the stomach; distally the fornix of the duodenal mucosa can be seen close to the distal end of the tumour.

this condition is not present at birth but develops at the end of the neonatal period. It is thought that previous spasm of the pylorus leads to hypertrophy of its circular muscle coat; the enlargement of the pylorus becomes evident in three to ten weeks after birth. Histologically, the nerve and muscle cells in the region are normal. This hypertrophic swelling of the pyloric region appears as an olive-shaped "tumour" of firm consistency. It fades gradually at its proximal end into thickened gastric muscle, whereas at its distal end it terminates abruptly and lies very close to the fornix of the surrounding duodenal mucosa (Fig. 135).



indicated when the child (*a*) fails to gain weight, (*b*) vomits repeatedly, or (*c*) shows evidence of peri-oesophagitis. Through a transthoracic approach, the oesophageal gastric region is freed from the posterior mediastinum and replaced below the diaphragm ; it is held in place by suturing the fundus of the stomach to the under surface of the diaphragm, and fashioning the hiatus closely around the cardia with interrupted sutures.

### THE DIAPHRAGM

*Eventration of the diaphragm* may be seen in infancy. The ascent of the intact diaphragm high into the chest causes cyanosis. The diaphragmatic muscle is poorly developed in most of such cases ; in some there may be phrenic nerve paralysis. When respiratory symptoms are severe urgent operation may be required. Plication of the diaphragm with interrupted sutures is often successful.

*Congenital Diaphragmatic Hernia.* This subject is discussed in Chap. 43.

admission the child is dehydrated, correction by subcutaneous, or less commonly and with due caution, intravenous fluids is necessary. Up to 400 ml. of *half-strength* isotonic saline with hyaluronidase (1,000 units per 500 to 1,000 ml. fluid) are given subcutaneously, or 100 ml. of half-strength isotonic saline per pound weight is given in the first twenty-four hours intravenously. If dehydration is extreme, *isotonic* saline should be given followed by plasma or blood. The fluid requirements are calculated as suggested in Chap. 23.

Before operation, the stomach residue is washed out with saline solution and the intragastric tube is left in place. The choice between local or general anaesthesia is a matter of the surgeon's preference, and sometimes depends on whether or not a skilled anaesthetist is available. Before general anaesthesia, premedication with atropine, 0.15 mg. (1/400 gr.), is necessary, but care should be taken to avoid an overdose of this drug, *especially if medical treatment by an atropine derivative has been employed previously* (in these circumstances it is wise to withdraw antispasmodics at least twenty-four hours before operation and to halve the dose of atropine recommended above). When local anaesthesia ( $\frac{1}{2}$  per cent. procaine) is used about 10 to 15 ml. are required to infiltrate the layers of the abdomen in the area of the incision; preoperative chloral hydrate, 60 to 120 mg. (1 to 2 gr.), is used to control restlessness.

A right upper rectus muscle-splitting incision is made. When the peritoneum is opened, the anterior surface of the liver is first seen. Its lower border is retracted upwards and the free margin of falciform ligament is drawn aside, the stomach wall is then picked up with bowel holding forceps and withdrawn from the wound. The pyloric "tumour" comes into view without disturbing the adjacent bowel. A 2 to 3 cm. longitudinal incision is made in an avascular area of the tumour, beginning just proximal to the pyloroduodenal junction. The margins of the incision are separated by inserting a blunt-ended forceps into its depth and separating the blades (Fig. 136A). The hypertrophic muscle is thus widely split and the underlying pyloric mucosa exposed (Fig. 136B). Special care is necessary when splitting the muscle distally to avoid opening the duodenum, for the superficial fornix of its mucosa lies very close to the distal end of the incision (Fig. 135). Provided a blunt-ended forceps is used, this risk is minimised, but in the rare event of a duodenal perforation being produced, the hole is closed with an atraumatic suture.\* Bleeding from the pyloric incision is controlled with hot packs. The stomach is replaced into the abdomen and the wound closed accurately in layers.

After operation 10 per cent. dextrose in half-strength isotonic saline is given by mouth, 5 to 10 ml. (1 to 2 dr.) hourly, followed by the addition of milk after about six hours. At first, expressed breast milk is given in increasing amounts until full feeds are tolerated—usually within three or

\* An eyeless needle swaged to the suture material.

Vomiting after feeds is the prominent symptom ; this is slight at first, but later becomes projectile. Occasionally the vomitus contains altered blood but never bile. Constipation and loss of weight occur. The physical signs of hypertrophic pyloric stenosis are visible gastric peristalsis and a palpable pyloric "tumour." Varying degrees of dehydration are evident. The diagnosis depends on the ease with which the pyloric swelling can be felt ; in most cases this presents no difficulty. The abdomen is best examined from the left side with the baby lying on the mother's or nurse's lap. The index finger of the left hand is gently pressed into the right upper quadrant of the abdomen just below the liver. A bottle feed is given to the baby during the examination to prevent crying and to stimulate gastric peristalsis ; the tumour is also easier to feel when the infant is occupied with a feed. In the absence of a palpable tumour such conditions as overfeeding, pylorospasm, œsophageal hiatus hernia or possibly duodenal obstruction have to be considered. Pylorospasm closely simulates hypertrophic pyloric stenosis, and similarly is relieved by antispasmodics. This response, in the absence of a palpable tumour, suggests the diagnosis of spasm. Hiatus hernia, if accompanied by œsophagitis, is more likely to cause hæmatemesis than is pyloric obstruction. Screening during a barium swallow examination differentiates these two conditions, but some laxity of the cardiac sphincter may occur as a secondary effect of pyloric obstruction. Radiography after a barium meal is required *only* when the diagnosis of hypertrophic pyloric stenosis is in doubt ; radiologically this condition shows marked delay in emptying of the stomach and the pyloric canal appears narrower and more elongated than normal.

*Treatment.* Infantile pyloric stenosis was first treated successfully in 1911 by Rammstedt, who divided the thickened muscle around the pylorus longitudinally (pylorotomy), thus relieving the obstruction. This operation, which bears the name of its originator, is still the standard procedure for this condition. Opinions differ as to whether surgical or medical treatment should be adopted ; however, surgery is always successful, whereas antispasmodic drugs relieve the condition only in selected cases, and even then operation is required for a small proportion of cases in this group when medical treatment fails. The decision regarding treatment usually rests with the pædiatrician, but nevertheless it should be pointed out that operation is definitely indicated when : (1) vomiting begins early after birth, i.e. during the second week ; (2) the baby is severely dehydrated ; (3) hæmatemesis has occurred ; or (4) the infant is below its birth weight. These criteria are those suggested by Jacoby (1946).

*Surgical Management.* Owing to the danger of cross-infection in hospitals, the baby should be treated in isolation in a hospital suitably equipped for dealing with infants, and the mother should be accommodated as well ; trained pædiatric medical and nursing staff are essential. If on

denum, usually involving the duodenojejunal junction, is reduced to a thin fibrous cord. The obstructive duodenal lesion is almost invariably single, in contrast with the multiple sites of occlusion characteristic of this type of obstruction in the intestine. Duodenal *atresia* causes gross distension of the proximal part of the duodenum and the pylorus becomes dilated as a thickened rim. The transverse colon is usually in its normal situation with its mesocolon stretched over the distended duodenum (Fig. 137). In *stenosis* of the duodenum, these appearances are less marked, but usually the lumen of the bowel above is larger in contrast with the jejunum. In infantile occlusion there is a high incidence of *mongolism* (one-third of the cases).

**Extrinsic Obstruction.** This is commonly associated with malrotation of the intestine. A well-defined peritoneal band stretches from the ileocaecal region (lying high up in the abdomen in these cases) across the duodenum to the right posterolateral region of the abdominal wall (Fig. 138). The band obstructs the malrotated duodenojejunal junction (Ladd, 1932). Volvulus (twisting) of the intestine may also occur around the axis of the superior mesenteric vessels (Fig. 138). In the condition known as *annular pancreas* a ring of pancreatic tissue encircles the second part of the duodenum, thereby constricting its lumen. This rare anomaly is

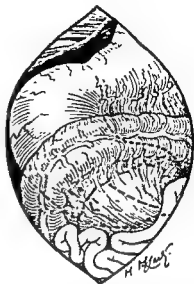


FIG. 137. Duodenal atresia. The distended duodenum is outlined; the interrupted straight line indicates the site for duodenojejunostomy.



FIG. 138. Extensive duodenal obstruction caused by a congenital band, seen after the associated volvulus of the small intestine had been untwisted (right hand diagram). The volvulus, before being untwisted is shown in the left-hand diagram. The gangrenous jejunum was resected and the congenital band divided. The patient, a three-day-old baby, recovered.

four days after operation. Apart from occasional regurgitation of feeds at first, progress including gain in weight is usually good.

**Complications.** Rupture of the wound may occur if the peritoneum has not been closed accurately. Occasionally infection of the abdominal incision occurs—especially in undernourished infants. The incidence of wound sepsis is minimised by thorough cleansing of the abdominal skin before operation and the discriminate use of antibiotics after operation. Perforation of the duodenum may occur during operation but, if recognised and sutured, no ill effect results. The mortality is decreasing and at present is below 2 per cent. of all the cases treated by operation.

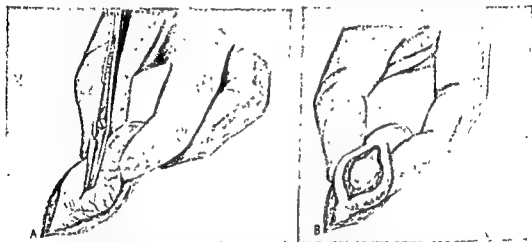


FIG. 136. A. Showing the pyloric "tumour" at operation. The adjacent stomach wall is being held steady whilst the hypertrophied muscle is being split with forceps. Note the relatively avascular area in the region of the incision. B. Appearance after the Rammstedt operation has been completed. The pyloric mucosa can be seen pouting through the line of the incision, after separation of its margins.

**Pyloric Obstruction in Children Following Ferrous Sulphate Poisoning.** The accidental swallowing of ferrous sulphate tablets by children is being recorded with increasing frequency. The poisoning is characterised by circulatory collapse and hæmatemesis. Symptoms vary in severity with the number of tablets taken, and may be delayed for several hours after the ingestion. Hæmorrhagic gastritis with ulceration develops and is followed in a few months by fibrosis and consequent obstruction of the pylorus. Gastro-enterostomy or occasionally a pyloroplasty, if the narrowing is limited, is usually required (Forshall and Rickham, 1954). The results of operation are satisfactory.

**Duodenal Obstruction. Duodenal Atresia and Stenosis.** Atresia or stenosis of the duodenum (intrinsic obstruction) usually occurs in the vicinity of the ampulla of Vater. Incomplete thin septa of mucous membrane are most frequently found, but sometimes a diaphragm completely occludes the lumen, and more rarely a portion of the duo-

cases of duodenal obstruction. Duodenal atresia and stenosis are best treated by a by-pass procedure, preferably a duodenojejunostomy. A single layer anastomosis is sufficient. If this operation is not anatomically possible a gastro-enterostomy is performed. Decompression of the proximal part of the duodenum is attained by suction through an indwelling gastric catheter until the functioning of the anastomosis is assured. For the rare annular pancreas it is generally agreed that a short-circuiting operation is preferable to excision of the constricting ring of pancreatic tissue, for the latter procedure does not relieve any underlying narrowing of the duodenal lumen. For extrinsic adhesions the procedure suggested by Ladd is usually successful. The avascular peritoneal band is divided, thus relaxing the occluded duodenum; also the ileocaecal region is sufficiently freed to prevent subsequent volvulus of the duodenocolic isthmus (Fig. 138).

### CONGENITAL ABNORMALITIES OF THE BILIARY TRACT IN INFANTS

**Atresia** is the commonest congenital lesion of the biliary tract. The common bile duct may be occluded above or below the entry of the cystic duct, and thus the gall bladder may be empty or distended. The part of the duct which has no lumen may be represented by a fibrous cord. The prominent feature is obstructive jaundice, which is usually evident at birth, although occasionally it may not become obvious until a few days later. The jaundice increases gradually, and after a few weeks develops a greenish-yellow tinge. Enlargement of the liver occurs and its surface eventually becomes irregular as the result of marked cirrhosis. The urine throughout is darkly coloured with bile pigments, and the stools are pale, resembling putty. This condition should be distinguished as early as possible from other causes of jaundice in the new-born, so that operative treatment can be carried out before the gall bladder and biliary ducts are "buried" by the enlarging liver. Such conditions as icterus neonatorum, erythroblastosis foetalis, congenital syphilis and various infections have to be considered in the differential diagnosis.

**Treatment.** Operative treatment should be carried out, for it holds the only hope of recovery; but successes are rare. Before operation a blood transfusion is given and administration of vitamin K (menaphthone B.P.C. 0.5 to 1 mg.) is necessary to guard against the supposed increased tendency to bleeding in these cases. The gall bladder area is explored through a generous right paramedian incision. If this organ is enlarged its contents are aspirated and examined. The presence of bile indicates that the obstruction is *below* the opening of the cystic duct, whereas the presence of clear mucus indicates that the obstruction is *above* the opening of the cystic duct. The peritoneum over the line of the common bile duct is opened. Dissection may reveal a distended duct, or more often a solid

caused by failure of the ventral anlage of the embryonic pancreas to rotate with the duodenum.

*Symptomatology.* Complete duodenal obstruction gives rise to vomiting beginning two or three days after birth. The vomitus is often copious and green. This colour indicates that the obstruction is below the ampulla of Vater. Distension and peristalsis in the gastric area are evident ; the rest of the abdomen appears normal, for the bowel beyond the obstruction

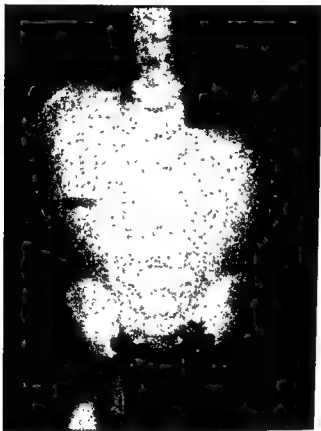


FIG. 139. Radiograph of the abdomen in a new-born infant suffering from duodenal atresia. The gastric shadow can be seen on the reader's right and a small bubble of gas is visible in the duodenum on the left. The rest of the abdomen is devoid of gas shadows.

is not distended. Occasionally pellets of inspissated meconium may be passed. Trypsin and bile are present in the faeces only if the obstruction is above the ampulla of Vater. Radiography of the abdomen demonstrates complete obstruction ; gas is seen only in the stomach and duodenum ; there is none in the intestine (Fig. 139). Incomplete obstruction due to stenosis or extrinsic causes gives rise to indefinite obstructive symptoms, which usually arise later in life and are often intermittent in character. A barium meal X-ray is necessary to elucidate incomplete duodenal occlusion.

*Treatment of Duodenal Obstruction.* Early operation is necessary for

cases of duodenal obstruction. Duodenal atresia and stenosis are best treated by a by-pass procedure, preferably a duodenojejunostomy. A single layer anastomosis is sufficient. If this operation is not anatomically possible a gastro-enterostomy is performed. Decompression of the proximal part of the duodenum is attained by suction through an indwelling gastric catheter until the functioning of the anastomosis is assured. For the rare annular pancreas it is generally agreed that a short-circuiting operation is preferable to excision of the constricting ring of pancreatic tissue, for the latter procedure does not relieve any underlying narrowing of the duodenal lumen. For extrinsic adhesions the procedure suggested by Ladd is usually successful. The avascular peritoneal band is divided, thus relaxing the occluded duodenum; also the ileocecal region is sufficiently freed to prevent subsequent volvulus of the duodenocolic isthmus (Fig. 138).

#### CONGENITAL ABNORMALITIES OF THE BILIARY TRACT IN INFANTS

Atresia is the commonest congenital lesion of the biliary tract. The common bile duct may be occluded above or below the entry of the cystic duct, and thus the gall bladder may be empty or distended. The part of the duct which has no lumen may be represented by a fibrous cord. The prominent feature is obstructive jaundice, which is usually evident at birth, although occasionally it may not become obvious until a few days later. The jaundice increases gradually, and after a few weeks develops a greenish-yellow tinge. Enlargement of the liver occurs and its surface eventually becomes irregular as the result of marked cirrhosis. The urine throughout is darkly coloured with bile pigments, and the stools are pale, resembling putty. This condition should be distinguished as early as possible from other causes of jaundice in the new-born, so that operative treatment can be carried out before the gall bladder and biliary ducts are "buried" by the enlarging liver. Such conditions as *icterus neonatorum*, *erythroblastosis foetalis*, congenital syphilis and various infections have to be considered in the differential diagnosis.

*Treatment.* Operative treatment should be carried out, for it holds the only hope of recovery; but successes are rare. Before operation a blood transfusion is given and administration of vitamin K (menaphthone B.P.C. 0.5 to 1 mg.) is necessary to guard against the supposed increased tendency to bleeding in these cases. The gall bladder area is explored through a generous right paramedian incision. If this organ is enlarged its contents are aspirated and examined. The presence of bile indicates that the obstruction is *below* the opening of the cystic duct, whereas the presence of clear mucus indicates that the obstruction is *above* the opening of the cystic duct. The peritoneum over the line of the common bile duct is opened. Dissection may reveal a distended duct, or more often a solid



fibrous cord. The procedure to be adopted depends upon the site of the obstruction, demonstrated by injection and distension of the patent duct with saline. If the proximal part of the hepatic duct is patent it is anastomosed to the adjacent duodenum or a loop of jejunum. This is best carried out over a tube by a single-layer junction. When the jejunum is



FIG. 140. Radiograph of the abdomen in a new-born infant suffering from cystic dilatation of the bile ducts. A smooth swelling which occupied the upper abdomen is outlined below by gas in the downwardly displaced large intestine.

used, the adjacent afferent and efferent portion of jejunum are anastomosed as well to lessen the chance of digestive juices entering the biliary tract (Maingot, 1955). If the obstruction is below the cystic duct the gall bladder is anastomosed, preferably to the duodenum or, if practicable, to the stomach. Obstruction above the junction of the hepatic ducts is fatal, for the obstruction cannot be relieved. Those infants in whom the lesion is inoperable survive for many months before eventually dying from cholæmia. Only about 20 per cent. of these obstructive lesions are operable, for extra hepatic obstruction is found in only a small proportion of cases.

#### Obstruction Due to Biliary Sand.

Rarely the ducts are obstructed by debris, known as biliary sand or mud. The condition closely simulates congenital biliary obstruction. When the abdomen has been opened, gentle pressure on the gall bladder, or the instillation of saline under pressure, may be sufficient to relieve

the obstruction. This type of biliary obstruction is rare compared with biliary atresia. It may account for the occasional case of cure in a seemingly fatal case of obstructive jaundice.

**Biliary Peritonitis.** This curious condition may be found in children or adults. In the former, fullness of the abdomen, slight jaundice and upper abdominal discomfort lead to operation. Biliary fluid is found in the abdomen. Leakage of bile most likely comes from a perforation of the biliary duct, but usually no hole can be found, although the region appears œdematous. Providing the biliary tract is patent, recovery usually follows.

**Congenital Cystic Dilatation of the Common Bile Duct.** This curious

anomaly, the cause of which is unknown, presents as a cystic swelling, arising in the bile duct and lying in the upper abdomen. The symptoms, which occur in infancy or early childhood, consist of abdominal discomfort and vomiting. Obstructive jaundice is also present and is occasionally accompanied by intermittent fever. On examination, the swelling is usually readily palpable in the epigastric region, and is of variable size. Radiologically the stomach and the colon can be seen to be displaced by the cyst (Fig. 140). The swelling, which may be the size of a large orange, lies in the region of the common bile duct and contains bile-stained fluid. Above it the gall bladder and cystic duct may be dilated and the liver may be cirrhotic. The cyst is anastomosed to the adjacent duodenum and so drained internally. External drainage is usually fatal. The obstruction of the bile ducts is usually not obvious in post-mortem specimens, but probably arises during foetal life as the result of occlusion of the lower end of the bile duct by incomplete canalisation, by inspissated bile, or by inflammation.

**Other Abnormalities.** Various rare anomalies of the gall bladder may occur. It may be double, bilobed, or lie on the left side. It may be absent or lie buried within the liver. The bile duct and cystic duct may be duplicated.

## CHAPTER 10

### CONGENITAL DEFECTS OF THE JEJUNUM, ILEUM AND COLON

**Malrotation and Volvulus of the Intestine.** Volvulus is usually associated with malrotation of the intestine, for the resulting incomplete fixation allows the free loops of bowel to rotate on a narrow attachment. This occurs most commonly at the duodeno-colic isthmus around the axis of the superior mesenteric artery (Fig. 141). In exomphalos, malrotation

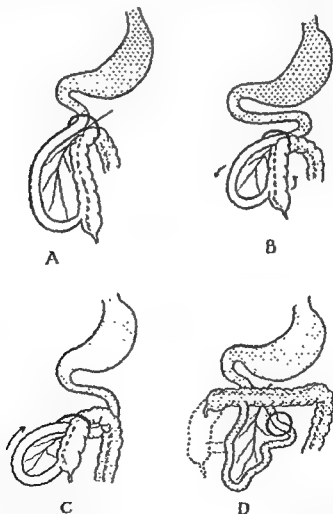


FIG 141. The normal rotation of the intestine in relation to the umbilical ring (as represented by black lined circle). A. The superior mesenteric vessels and the corresponding enterocolic loop outside the abdomen. B. A further stage—anticlockwise rotation through 180 degrees. C. At tenth week ; loop receding inside the abdomen. D. Showing descent of the caecum. (D. Levi, *Post. Grad. Med. Journ.*)

and incomplete descent of the caecum are common findings ; they are both examples of arrested development of the abdominal wall and intestine. Volvulus, although it may arise at any age, occurs most often in infancy. Symptoms and signs of high intestinal obstruction develop (i.e. gastro-duodenal distension and peristalsis with bile-stained vomitus and constipation), and occasionally blood may be passed per rectum. A radiograph of the abdomen may show gaseous distension of the stomach and duodenum and a few obstructed loops of bowel. Early operation is necessary. The bowel is untwisted and its viability is assessed. Strangulation of the mesenteric vessels with gangrene of the intestine demands resection of the devitalised bowel. In all these cases the duodenum must be examined and freed from any extrinsic adhesions, which are commonly present. Successful operation depends on relief of any coexisting duodenal obstruction as well as untwisting the volvulus.

Congenital failure of descent of the ileocaecal region from the upper abdomen to the right iliac fossa gives rise to upper abdominal pain and tenderness if appendicitis occurs. In such cases examination of the right iliac fossa may disclose that the caecum is not in its normal place.

**Atresia and Stenosis of the Jejunum and Ileum.** The entire small intestine is known to be a solid structure between the fifth and seventh week of foetal life, although there is some doubt as to whether such a stage exists in the case of the colon. Failure of the intestine to canalise produces lesions varying from minor degrees of stenosis to complete atresia of large tracts of intestine. A variety of these intestinal lesions is represented in Fig. 142. The above theory does not explain all the findings in cases of intestinal atresia. It may be that some defects are caused by strangulation of the bowel *in utero* by twisting, by bands or by intussusception. More recently, animal experiments support the hypothesis that intestinal atresia results from interference with the blood supply of a segment of foetal gut leading to disappearance of the de-vascularised zones (Louw, 1952 and 1955). This theory explains the gaps in the mesentery in multiple atresias. The bowel above the obstruction is greatly distended with bile-stained fluid and gas, and may become

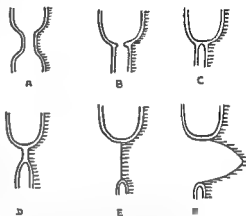


FIG. 142. Types of stenosis and atresia. A. Simple narrowing. B. Perforated diaphragm. C. Complete diaphragm. D. Short band connecting ends of the bowel. E. Thread-like band along the free edge of the mesentery ; the ends of the bowel are some distance apart. F. Gap in mesentery with no direct connection between the ends of the bowel. (Spriggs, *Guy's Hospital Reports*.)

twisted ; its wall is often hypertrophied. The distal bowel is collapsed and airless and contains plugs of inspissated pale meconium. Lesions are often multiple and consist of isolated cordlike lengths of intestine. The collapsed distal bowel has been termed "microcolon," although it is normal in structure and can attain normal dimensions.

*Symptoms.* With complete obstruction, at birth the abdomen appears normal but, within a few hours, increasing abdominal distension develops.



FIG. 143. Radiograph showing gaseous distension and fluid levels in obstruction of the ileum. This was a case of ileal atresia in a new-born infant, treated successfully by operation.

After about twenty-four hours vomiting of bile-stained fluid occurs and little or no meconium is passed. Later, dehydration and loss of weight are evident. In cases of stenosis the incomplete obstruction gives rise to similar symptoms, but they may be delayed for several days or weeks after birth and are usually intermittent. The diagnosis of complete obstruction can be made by radiography of the abdomen ; it shows distended loops of bowel and, in many instances, fluid levels (Fig. 143). Mild obstructive symptoms may require examination by a barium meal to demonstrate the site of the stenosis. Strangulated hernia, intussusception, Hirschsprung's disease, meconium ileus and rectal atresia simulate intestinal

atresia in the new-born ; the features of these conditions are given elsewhere.

*Treatment.* Atresia of the jejunum requires immediate operation. The junction of the distended and collapsed bowel is exposed ; the contrast between the grossly distended blind end and the small collapsed ribbon-like bowel below is most striking. The whole length of the distal bowel is examined for multiple lesions by injecting saline from a syringe through a fine needle introduced into the lumen of the bowel ; the distending fluid readily passes down the whole intestinal tract. This procedure not only helps to free the sticky meconium from the mucosa of the bowel, but also ensures its patency. The grossly distended loops of bowel above the obstruction and any isolated occluded segments below are best resected, with due regard to the length of intestine removed. It is most important

that healthy proximal bowel should be joined by end-to-end anastomosis to wholly patent intestine below. As this may not be practicable in every case, especially in ileal obstruction, a temporary ileostomy is made and an anastomosis carried out a few days later. The success of all these procedures depends largely on maintaining the normal fluid and electrolyte balance and the gradual return to oral feeding. Infants with atresia of the intestinal tract die if operation is not performed. Although the operative mortality is still high, increasing numbers of infants are surviving. In special centres, the recovery rate is between 60 and 70 per cent. Treatment is more successful in duodenal obstruction than in lesions of the small intestine. In incomplete obstruction (stenosis) the chances of success are better. Associated congenital abnormalities and delay in recognition of the obstructive symptoms contribute to the high mortality.

**Meconium Ileus.** This is a form of intestinal obstruction in the newborn produced by blockage of the intestine with inspissated meconium. It is commonly associated with cystic fibrosis of the pancreas (Andersen, 1938). There is diminished secretion of mucus, especially in the duodenum, and trypsin is absent from the pancreatic juice. The meconium becomes dry and tenacious. The lower part of the ileum and the colon are filled with firm, pale plugs of inspissated meconium adherent to the mucous membrane (Fig. 144). Occasionally these pellets can be felt like a string of beads lying in right lower quadrants of the abdomen. The proximal part of the small intestine, which is markedly distended with green viscous meconium, may become twisted or even perforated (Fig. 145).

Early operation is indicated. The plugs of inspissated meconium are freed from the adherent mucosa by injection of saline downwards into the lumen of the bowel, and are then squeezed out of an opening made in the bowel wall until the ileum is cleared. Removal of the tenacious meconium from the distended proximal bowel may be impossible; resection of this part of the bowel is therefore usually carried out, followed by end-to-end anastomosis or, preferably, a double-barrelled enterostomy; the latter is closed a few days later. Occasionally, when fibrocystic disease of the pancreas is diagnosed early by the absence of trypsin in the duodenal juice and stools, attacks of transient obstruction due to meconium ileus may be relieved by administration of trypsin.\* Children affected with this disease require a high protein and high calorie diet, and also antibiotics to guard against pulmonary infection which is so liable to develop. Although the cause of the disease is unknown, recent appreciation of its pathology has led to some improvement in results, but nevertheless operative treatment still carries a mortality of about 70 per cent.

**Meconium Peritonitis.** The morbid appearances of this condition, first described by Morgagni in 1751, have only recently become recognised. It is caused by leakage of the sterile meconium into the peritoneal cavity

\* 4 ml. of 10 per cent. solution of trypsin three-hourly by mouth.

from a hole in the bowel. In some cases a primary obstructive lesion in the foetus leads to rupture of the distended bowel, whereas in others perforation of the bowel occurs without any evident cause. The resulting peritoneal reaction varies with the amount of extravasated material, from marked ascites to a small localised mass of adhesions; the latter usually contain gritty, chalky deposits. The abdomen is distended with ascitic fluid at birth, and later the distension is increased by swallowed



FIG. 144. Above : Normal meconium. Below : Inspissated meconium which caused blockage of the lower end of the ileum.

air. There is also evidence of intestinal obstruction. A radiograph of the abdomen shows fluid levels within and outside the intestine, and also the characteristic calcification which occurs in the extravasated meconium (Fig. 146). Surgical treatment is only occasionally successful, for the anatomy is often obscured by the presence of widespread adhesions. Sometimes, however, a mass of adherent intestines can be removed and the continuity of the bowel restored.

**Peritonitis.** Peritonitis may arise in infancy from such conditions as perforated appendix, perforated Meckel's diverticulum, or rarely from extravasation of bile from a hole in the biliary passages. Peri-umbilical

infection in the new-born may involve the peritoneal cavity secondarily, causing peritonitis and even abscess formation.

**Meckel's Diverticulum.** This is a congenital defect caused by persistence and growth of the intestinal end of the vitelline duct (the primitive connection between the foetal intestine and the yolk sac). It occurs



FIG. 145. Meconium ileus. A large obstructed loop of bowel which was resected from an infant with meconium ileus. This proximal length of small intestine is grossly dilated, containing dark green sticky meconium, as seen in cross-section below on the right. The distal part of the intestine which is narrower, contained white inspissated plugs of meconium as seen in cross-section below on the left.

in about 2 per cent. of the population and affects males more often than females. It is found in the ileum about 1 metre from the ileocaecal junction, on the antimesenteric border of the intestine. The diverticulum is about 5 cm. long and has an arterial supply derived from the continuation of the superior mesenteric artery, which sometimes lies in a separate mesenteric fold as far as the apex of the diverticulum (Fig. 147). The structure of a Meckel's diverticulum is similar to that of the small intestine, except that occasionally heterotopic gastric mucosa is present, secreting gastric juice; very rarely biliary, pancreatic or colonic mucosa may be found. The apex of the diverticulum may lie free or it may



be attached to the umbilicus by a fibrous band, the remains of the rest of the vitello-enteric canal. Occasionally a part or the whole of this attachment is patent.

*Symptoms.* The majority of Meckel's diverticula do not cause symptoms and are discovered incidentally during abdominal operations. However,



FIG. 146. Radiographic appearances of meconium peritonitis in a new-born infant. The bowel was obstructed in the duum and there was a mass of adhesions in the right iliac fossa with extravasated meconium, some of which was calcified as seen in the right iliac fossa.

abdominal symptoms do sometimes arise from the presence of a diverticulum. They are caused by : (a) Intestinal obstruction ; (b) inflammation ; (c) ulceration ; or (d) an umbilical fistula.

*Intestinal obstruction* may be caused by intussusception ; this usually occurs as the result of invagination of the thickened fundus of the diverticulum. This condition cannot be distinguished from other forms of acute intussusception. After reduction of the intussusception the diverticulum is removed to prevent recurrence. Volvulus of a Meckel's

diverticulum may occur alone or in association with congenital malrotation of the intestine, or exomphalos. Adhesions of the mesentery of the diverticulum may occur and lead to thrombosis of its vessels and consequent gangrene of the pouch. The omphalo-intestinal cord may cause intestinal obstruction if loops of bowel become twisted around this axis.

*Inflammation.* Acute diverticulitis may arise and cause symptoms indistinguishable from those of acute appendicitis. Therefore at operation for the latter condition, failure to find inflammation of the appendix should always lead to a search for an inflamed Meckel's diverticulum. An apparent recurrence of "acute appendicitis" at any time after appendicectomy, may be due to inflammation of a Meckel's diverticulum. Rarely, a foreign body may perforate the wall of a diverticulum and lead to peritonitis.

*Ulceration* may arise in ectopic areas of gastric mucosa in the fundus of the diverticulum, or near its internal opening. The peptic ulceration may cause bleeding, perforation or stenosis. Hæmorrhage from an intestinal peptic ulcer should be considered if melæna and abdominal colic occur in a child, and intussusception, Henoch's purpura and intestinal polypi have been excluded. Not infrequently a correct preoperative diagnosis of bleeding from a Meckel's diverticulum can be made. The diverticulum is excised, or if the erosion is in the adjacent intestine, the affected segment of the bowel together with the diverticulum is resected. Perforation of a peptic ulcer in a Meckel's diverticulum is treated by resection of the diverticulum. Stenosis of the ileum caused by a peptic ulcer near the mouth of a diverticulum may have to be treated by resection of a small segment of bowel along with the diverticulum.

*Umbilical Fistula.* A patent omphalomesenteric duct causes a discharge from the umbilicus and is best treated by removal of the tract.

*Operation for Removal of Meckel's Diverticulum.* The mesentery of the diverticulum is first tied and the junction of the diverticulum and the ileum is then crushed with forceps and divided; the ileum is closed with a single layer of inverting stitches. It is advisable to place this line of sutures oblique to the length of the bowel, thereby preventing stenosis of the ileum. When a Meckel's diverticulum is found at operation performed for some other condition, it should be removed if the risk is not thereby increased.

*Hirschsprung's Disease.* In recent years elucidation of the pathology

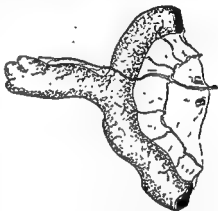


FIG. 147. A Meckel's diverticulum with its blood supply derived directly from the superior mesenteric artery.

of this disease has led to rational treatment (Swenson and Bill, 1948). It is now established that a narrow segment of bowel causes intestinal obstruction. Microscopically, the affected length of bowel shows absence of ganglion cells in the intramural plexus and the corresponding sympathetic nerve trunks are abnormal; they are thicker than normal. The pelvirectal part of the bowel is most often affected. Rarely, a long length of large bowel is involved and the aganglionic portion may even extend to the small intestine. The narrow segment causes gross dilatation and hypertrophy of the proximal large intestine. Hirschsprung's disease is present at birth; it affects boys more often than girls in the proportion of about 10 to 1. Sometimes it is familial. The disease is characterised by chronic or intermittent large-bowel obstruction of variable severity.

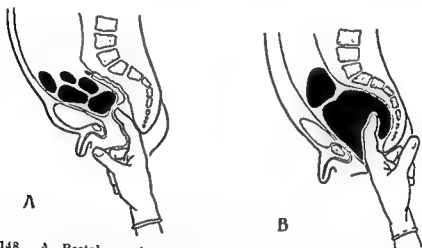


FIG 148. A. Rectal examination in a case of Hirschsprung's disease. The examining finger reaches the narrow segment, and impacted faeces can be felt through the bowel wall. B. Rectal examination in a case of idiopathic colonic inertia. The examining finger on entering the anus comes in contact with a mass of faecal material within the rectum. (After Bodian.)

Constipation is accompanied by increasing abdominal distension. Examination of the rectum reveals absence of faeces, and narrowing of the pelvirectal junction; an accumulation of hard faeces in the pelvic colon is often palpable *per rectum* as well as on abdominal examination (Fig. 148). Intermittent bouts of intestinal obstruction are liable to develop; vomiting, abdominal distension and visible peristalsis occur. Children with Hirschsprung's disease are usually underweight and lack vigour.

A plain radiograph shows distended loops of large intestine in which fluid levels are seen; shadows of a faecal accumulation in the pelvic colon are also usually present. A barium enema given without preliminary emptying of the bowel shows the narrow segment below and the outline of the faeces above. In the new-born the narrow segment, being underdeveloped, is difficult to demonstrate radiologically, but in later months it can usually be well shown. In doubtful cases resort to biopsy of the narrow segment of the rectum is necessary to confirm the diagnosis.

Despite the fact that Hirschsprung's disease is the commonest cause of neonatal obstruction, the diagnosis is often missed during life.

*Differential Diagnosis.* In *idiopathic colonic inertia* the rectum is distended with faeces which lie just above the anus (Fig. 148); this condition may arise secondarily to a painful anal fissure. In these cases the abdominal distension is less marked and is more easily treated by enemas and bowel training than is Hirschsprung's disease. *Dolichocolon*, which is probably a variant of colonic inertia, produces similar symptoms. It is demonstrated radiologically by the presence of long loops of large bowel of even calibre. The loops are liable to become twisted; if treatment by wash-outs and laxatives is unsuccessful, resection of the redundant loops is necessary. *Anal stenosis* likewise causes chronic constipation, but on rectal examination the narrowing of the anal opening is felt. Ascites, coliac disease and fibrocystic disease of pancreas are included in the differential diagnosis, for they also cause abdominal distension.

*Management of Hirschsprung's Disease.* The ultimate aim of treatment is the removal of the narrow segment of bowel by rectosigmoidectomy. Some surgeons operate when the child is only a few months old, whereas others think it wiser to wait until the child is one to two years old, for the operation is then easier and safer.

At first, laxatives, enemas and glycerine suppositories may succeed in preventing faecal accumulation above the narrow segment. Often the blockage by the impacted faeces can be relieved from below by passing a finger into the rectum. Sooner or later, despite constant care, intestinal obstruction usually develops, necessitating relief by transverse colostomy. This temporary opening is maintained until after the rectosigmoidectomy. When symptoms occur in early life colostomy can sometimes be avoided although the affected infants tend to remain underweight and lacking in vigour. Rectosigmoidectomy can be performed as a one-stage operation, although preliminary colostomy is desirable to relieve the chronic intestinal obstruction and to encourage healing of the anastomosis after the removal of the narrow segment. The amount of bowel removed is dictated by the extent of the narrow segment and the level at which normal neural ganglia appear, as determined by frozen section of the bowel examined during the operation.

*Rectosigmoidectomy.* The abdomen is opened with the infant in the Trendelenburg position. The distal part of the colon, including the narrow segment, is mobilised from the splenic flexure to the floor of the pelvis just above the levatores ani without damaging the blood supply of the bowel. This is accomplished by dividing the lateral peritoneal attachments of the descending and pelvic colon and then freeing the narrow segment from the pelvis. Next, the level of the bowel section is selected well above the aganglionic segment so that the divided proximal end of the colon has a good blood supply and can reach the anus without

tension. A sigmoidoscope is then passed into the rectum from below, and long traction sutures are introduced from above into the lumen of the instrument. By downward pull on these sutures the rectosigmoid region is intussuscepted through the anus. The ensheathing rectal wall is then incised obliquely within 2 to 3 cm. of the anal margin and the bowel within is withdrawn and divided at the selected level. After the cut margins of the anorectal wall have been carefully sutured to the divided



FIG. 149. A mesenteric cyst occurring in a child aged ten. Histological examination showed it to be a lymphatic cyst.

end of the pelvic colon the line of junction is allowed to recede through the anus. The abdomen is then closed. Two or three weeks later the colostomy is closed.

*Complications.* Peritonitis may follow breaking down of the anastomosis, especially in the absence of a colostomy. Obstruction may persist if the anastomosis is too narrow, or too great a length of transitional bowel between the dilated and narrow segments is retained.

*Results.* Formerly, the majority of infants with Hirschsprung's disease died in childhood, but now with earlier diagnosis and rational treatment, increasing numbers of children are surviving and most of them have normal bowel function after operation. In special centres the operative mortality is about 5 per cent. (Ehrenpreiss, 1955).

**Mesenteric Cysts.** Mesenteric cysts are seen more often in the pathology museum than in hospital practice. They occur as uni- or multilocular cysts in the mesentery (Fig. 149). They are thought to arise by malformation of the lymphatics (Willis, 1948). The cysts contain either clear lymph or milky chylous fluid. They tend to interfere with bowel function and may cause intestinal obstruction or volvulus. They may rupture, or hæmorrhage may occur in them. Histologically, the inner lining consists of a single layer of endothelial cells, compatible with lymphatic origin. Mesenteric cysts are thin walled and, in contrast with *enterogenous cysts*, are separate from the adjacent bowel and have an independent blood supply. The abdominal manifestations vary from vague discomfort to intestinal obstruction. A large mesenteric cyst presents as a smooth abdominal swelling which is more mobile at right angles to the line of attachment of the mesentery than in the direction of this line. An X-ray of the abdomen shows displacement of bowel shadows by a rounded mass.

**Treatment.** Enucleation of the cyst may be all that is necessary, but if it is of such a size as to interfere with the blood supply of the adjacent bowel the latter has to be resected. Simple puncture of the cyst and marsupialisation are also practised, but are not recommended because of the likelihood of recurrence. The prognosis after removal of a mesenteric cyst with few loculi is better than that after removal of a cyst with many loculi, for it may be impossible completely to eradicate the latter type of cyst.

**Duplication of the Intestine and Enterogenous Cysts.** These congenital conditions may occur anywhere along the intestine. Clinically they may simulate a mesenteric cyst by causing a palpable abdominal swelling and intermittent intestinal obstruction. One theory is that these congenital defects arise as a diverticulum of the bowel along the mesenteric border. They are composed of an epithelial lining surrounded by a muscle layer. Thus they have a thicker wall than mesenteric cysts. Their blood supply is shared with that of the adjacent intestine (cf. the independent blood supply of mesenteric cysts). Therefore resection must always include the corresponding segment of bowel. Duplication of the intestine can sometimes be demonstrated by X-ray examination after a barium meal.

**Other intra-abdominal cysts** (e.g. renal cysts, retroperitoneal cysts arising from aberrant renal tissue, pseudopancreatic cysts and hydatid cysts) are relatively fixed compared with those arising from the mobile mesentery or intestine.

## CHAPTER 20

### MALFORMATIONS OF THE RECTUM AND ANUS

THE anorectal end of the alimentary canal is developed above from the hind-gut, below from indentation of the proctodeal membrane at the anal pit, and in the middle from the dorsal part of the cloaca separated from the urogenital canal by downgrowth of the urorectal septum (Fig. 150). After completion of the subdivision of the cloaca, the urogenital and anal pits at first lie close to each other on the proctodeal membrane, but later they become separated by the development of the perineum. At the same time the end of the rectum migrates dorsally and caudally. When development of the perineum is impaired the anal membrane fails to



FIG. 150. The caudal end of human embryo. (After Keibel.) A. Showing development of the urorectal septum (embryo 11.5 mm. long). B. Showing separation of the cloaca completed in the female (embryo 25 mm. long).

migrate, so that the anus remains in an ectopic anterior position (Fig. 151). Furthermore, the inner genital folds, which overlie the perineum in the female, develop into the labia minora; in the male they fuse in the midline to form the median raphe of the perineum; excessive fusion of these folds will give rise to the condition known as "covered anus" (Denis Browne, 1951, and F. Douglas Stephens, 1953).

**Types of Malformation.** *Imperforate Rectum.*—(1) Ending as a blind pouch; (2) ending with a fistula into the urinary or genital tract. *Malformation of the Anus.*—(1) Imperforate anal membrane; (2) imperforate anus; (3) covered anus; (4) ectopic anus.

Failure of development of the cloacal portion of the rectum causes the bowel to end above the pelvic floor, either as a blind pouch (Fig. 152) or as a fistula; in the male such a fistula enters the base of the bladder or the urethra at the level of the verumontanum, whereas in the female the inter-position of the genital tract between the bladder and the rectum

transposes the fistula lower down, so that the rectum opens into the posterior wall of the vagina at various levels (Fig. 153).

The anal membrane may be imperforate or incompletely absorbed. The obstruction or narrowing occurs at the level of the anal valves more often than in the rectum. *Imperforate anus* is caused by failure of the distal end of the rectum to migrate dorsally and caudally to the anal pit

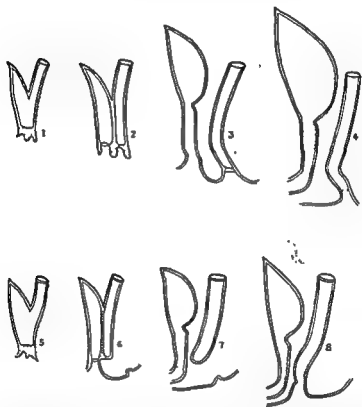


FIG. 151. 1-4. Show the normal formation and migration of the anal membrane and the development of the perineum. 5-7. Show deficient formation of the anal pit; the terminal end of the rectum has failed to migrate from its original site close to the urogenital membrane. 8. Shows an ectopic anus due to failure of development of the perineum and lack of migration of the otherwise normally developed anus. (F. Douglas Stephens, *Austral. N.Z. Journ. Surg.*)

(Fig. 151). The level of the resulting deformity corresponds in the new-born to the junction between the membranous and bulbous parts of the urethra.

*Covered anus* is caused by excessive fusion of the inner genital folds, which unite from behind forwards. The anal orifice may be completely closed over, or projected forwards forming an anocutaneous fistula, opening at any point in the median raphe. Sometimes the anal canal communicates with the bulbous part of the urethra, producing an anobulbar fistula. Similarly, in the female, an anovulvar fistula is formed



opening into the fourchette (Fig. 154). The ectopic anus results in failure of backward migration of the anal membrane, causing the anus to be placed anteriorly in the midline, close to the urethra in the male or the posterior margin of the vaginal orifice in the female (Fig. 155).

The anatomical levels of the above malformations can be correlated with the skeletal structure of the pelvis in the new-born (F. D. Stephens,



FIG. 152. The rectum is imperforate, ending as a blind pouch at the level of the base of the bladder.

1953). A line drawn from the upper surface of the pubis to the sacrococcygeal articulation—the pubococcygeal line—represents the level of the neck of the bladder, where the imperforate rectum and a recto-urinary fistula commonly end (Fig. 153). The lower border of the ossified ischium corresponds to level of the junction of the membranous and bulbous parts of the urethra, where the rectum ends in the case of imperforate anus, whereas 1 to 1.5 cm. below this marks the level of an imperforate anal membrane.

*Three-quarters* of the infants with malformations of the anorectal region have other associated congenital defects; thus cardiac or urological anomalies each affect a third, and oesophageal atresia a quarter, of the

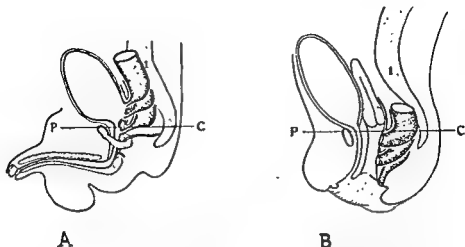


FIG. 153. A. The various levels of fistulous communication between the rectum and the urinary tract in the male. The line P-C represents the pubococcygeal line. B. The various levels of fistulae between the rectum and the genital tract in the female. (After F. D. Stephens.)

cases. Malformation of the sacrum is frequent and indicates poor development of the levator ani muscles. Also, the external sphincter is usually poorly developed.

**Clinical Appearances.** Inspection of the perianal skin of the new-born infant reveals most of the congenital abnormalities in this region, but, unless a rectal examination is carried out, rectal stenosis or atresia may be missed, for the anus usually appears normal in these deformities. External examination may show an ill-developed anal pit covered by a thickened median raphe with a thin membrane on either side, through which meconium may be seen (Fig. 156) or, rarely, a covered anus may be found

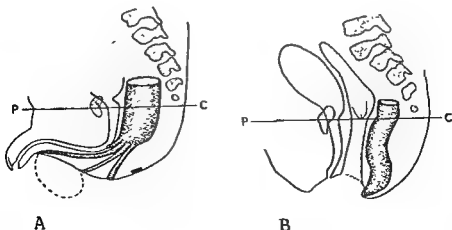


FIG. 154. A. Types of covered anus in the male, including ano-bulbar and ano-cutaneous fistulae. B. Covered anus in the female—ano-vulvar fistula. (F. Douglas Stevens, *Austral. N.Z. Journ. Surg.*)

and a trace of meconium may be seen exuding from a minute fistulous opening anteriorly in the midline. In the absence of a normally placed anus in the female exploration of the posterior vaginal wall with the tip of a urethral catheter may reveal a small aperture, and the degree of obliquity of the tract indicates whether it is an anocutaneous or recto-vaginal opening.

In low level obstruction straining or crying causes the anal region to bulge, and palpation enables the depth of the pent-up meconium to be appreciated. The appearance of a narrow string of meconium on straining,



FIG. 155. An ectopic anus. The opening lies posterior to the fourchette. In this case rectal function was normal.

indicates the presence of stenosis which can then be confirmed by rectal examination. Traces of meconium at the end of the male urethra or in the urine is evidence of a fistula within the urinary tract. In neglected cases of anorectal atresia, abdominal distension, visible peristalsis and vomiting occur *within a few hours after birth*; the intestines become filled with swallowed air. In all anorectal anomalies, before feeding is begun, a small rubber oesophageal tube should be passed to exclude any associated oesophageal atresia. Other congenital anomalies should also be sought.

**Differential Diagnosis.** Narrowing of the rectum can be shown by a barium enema. Occasionally the outline of a swelling such as a sacro-coccygeal tumour or dermoid cyst may be seen compressing the rectum. Relative narrowing also occurs in Hirschsprung's disease. Biopsy of the rectal wall in the suspected aganglionic area confirms the diagnosis in the latter disease (Chap. 19).

**Management.** In low-level obstruction the meconium-filled rectum causes bulging of the perineum on straining. Passage of a blunt instrument (e.g. a closed artery forceps) through the thinnest part of the obstructing tissue produces an easy flow of meconium. In the female probing of the lower part of the posterior vaginal wall may reveal an opening into the rectum. Once meconium has been obtained this tract is



FIG. 156. Usual appearance of imperforate anus. There is a thickened triangular part of the median raphe, in the posterior margin of the anal pit.

dilated and kept open by the regular passage of bougies of gradually increasing sizes. Good sphincter control usually results. If a covered anus is present the fistula is opened up towards the normal anal site. Skin tags are trimmed when necessary and the new opening is dilated regularly. In the case of an ectopic anus (Fig. 155) transposition of the anterior placed opening is not advisable, for provided the calibre of the ectopic anus is maintained, normal bowel control is ultimately achieved.

When the perineum does not bulge on straining the position of the blind end of the rectum is demonstrated by radiography. A radio-opaque marker is placed on the anal pit and lateral radiographs taken with the

infant inverted and after allowing time for gas to replace the meconium in the blind end of the bowel. This method demonstrates roughly the distance between the end of the bowel and the surface of the skin. If this is estimated to be less than 1.5 cm. a perineal approach is used to locate the blind pouch. In the case of an imperforate anus the lumen of the bowel is surrounded by the levator ani muscle and is usually close to the bulbocavernosus muscle. When meconium has been found with the aid of an exploring needle the tract is enlarged and kept widely patent by



FIG. 157. Radiological appearances in the case of imperforate rectum. The outline of the blind rectal pouch has been obtained with Myodil. The site of the anal pit is indicated by the marker—a square of sorbo rubber painted with barium emulsion.

regular dilatation. If the obstruction appears to be higher than 1.5 cm. from the skin a restorative abdominoperineal procedure is necessary. The tendency nowadays is to carry out this major operation in one stage as soon as possible after birth. If, however, the baby is weak or suffers from additional abnormalities an immediate colostomy is performed. An opening in the transverse colon is preferable to an iliac colostomy, as the former allows the pelvic colon to be mobilised later at the restorative operation. After establishment of the colostomy the level of the blind end in relation to the bony pelvis can be assessed radiologically by introducing radio-opaque fluid into the distal end of the colon (Fig. 157). Closure of a recto-urinary fistula should be carried out as soon as possible after birth to prevent urinary infection, but when there is no fistula the

restorative operation is postponed until the infant is thriving. If a preliminary radiograph of the sacrum shows its structure to be normal it can be assumed that the levator ani muscles are intact, although in high-level obstruction, at or above the pubococcygeal line, the levator muscles are drawn upwards and forward against the vagina or prostate (Stephens, 1953). The opening of a recto-urethral fistula is at the level of the verumontanum, i.e. above the puborectalis sling; a rectovaginal fistula may lie above this muscle, although it usually extends downwards between the levatores ani and the vagina before opening into the posterior vaginal wall.

**Restorative Operation.** This operation is used for high-level obstruction and is designed to preserve the function of the puborectalis (sphincteric part of the levator ani), for subsequent rectal control depends on this muscle. The blind rectal pouch is first freed from the pelvis and any fistula closed. An incision is made in the anal skin, and the mobilised bowel, which is further liberated proximally, is drawn through the anterior placed gap in the puborectalis muscle and thus comes to lie just posterior to the prostate (Fig. 158). The distal end of the freed rectum is finally stitched to the edges of the anal skin. After an interval of two or three weeks, when the margin of the newly formed anus is secure, the transverse colostomy, if present, is closed. The results of this operation vary. There may be control of the faeces but not of flatus. Sometimes bladder function is disturbed. The restorative operation is a major procedure which may not be practicable if the levator ani muscle is poorly developed or the infant's physical state is poor. A permanent colostomy is the only alternative and should be made in the left iliac region where a permanent colostomy is easier to manage than one in the transverse colon.

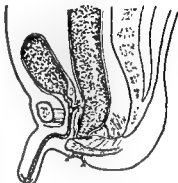


FIG. 158. The final appearances of the restorative operation in a case of recto-urethral fistula and imperforate rectum. The fistula has been closed and the rectal pouch transposed to the anal margin.

## CHAPTER 21

### TUMOURS AND SINUSES IN THE SACROCOCYGEAL REGION

#### SACROCOCYGEAL TUMOURS

TUMOURS in the sacrococcygeal region are uncommon, but they may present considerable difficulty in diagnosis.

Teratoma (literally a monster) is a misleading term, for, according to Willis, it is a true neoplasm composed of multiple tissues foreign to the



FIG. 159. : Appearance caused by a teratoma occupying the right posterolateral aspect of the rectum.

part of the body in which it arises. Thus it is distinct from a suppressed twin to which the term monster may be correctly applied. The growth usually arises in the presacral region where it may distort the rectum or bulge the perianal region externally (Fig. 159). Its rate of growth and size are variable ; it may be of hard or soft consistency, for some parts of the tumour are solid and others cystic. It thus has an irregular shape. Histologically it is seen to be composed of heterogenous embryonic tissue consisting of ectodermal, mesodermal and endodermal elements, giving rise to a corresponding variety of tissues. These teratomas are usually benign but they may become malignant. Their extent can be assessed by rectal examination or a barium enema. They should be completely removed

if possible. Removal of the coccyx through a posterior incision allows access to the tumour above the levatores ani and facilitates lateral and anterior dissection. The tumours usually envelop the rectum on one or both sides. Complete excision is curative, but residual tumour tissue may give rise to recurrence. However, total removal may be impossible owing to involvement of the rectum or even the bladder.

Meningoceles most commonly arise in the midline posteriorly in the lumbosacral region. If situated laterally (Fig. 160) or in the presacral region they may be confused with teratomas. Meningoceles are, however, cystic and smooth, and become tense with crying (see also Chap. 59).



FIG. 160. A meningocele lying lateral to the midline.

Chordomas arise from remains of the notochord. They are rare malignant tumours which may be discovered at any age. They form lobulated swellings in the sacrococcygeal region. On section they consist of cystic and solid parts. They are locally invasive but do not usually cause metastasis. X-ray examination shows local destruction of bone. Histologically they are seen to consist of masses of large clear cells distended with mucinous material giving a vacuolated appearance. Chordomas are usually considered to be inoperable and they are radio-resistant.

Duplication of the rectum is a very rare cause of a swelling in this region. It forms a smooth, cystic mass, containing clear mucoid fluid. It is ultimately connected with the rectum, and histologically consists of a smooth muscle wall on the outside and rectal mucosa on the inside. The duplicate portion of rectum should be excised.

Metastatic tumours in the sacral region should be borne in mind in the diagnosis of obscure swellings which show osteolytic changes in X-rays.



## SINUSES IN THE SACROCOCCYGEAL REGION

**Congenital Dermal Sinus.** This type of sinus may be found anywhere in the midline from the occiput to the coccyx, but usually appears as a dimple in the skin of the natal cleft. It may be a shallow depression or a deep tract in the midline (Fig. 161). Sometimes the skin around is raised, naevoid or pigmented. The sinus may be due to (a) sequestration of ectoderm, (b) failure of separation of the neural and dermal elements of the ectoderm, or (c) spina bifida deformity. Thus, some of these sinuses



FIG. 161. A congenital dermal sinus.

are superficial, whereas others are deep and may be connected to the spinal theca or to the coccyx by a fibrous cord. Moreover, there may be associated underlying lesions of the spinal cord or sacrum (see under Spina Bifida). Most of these sinuses do not cause trouble, but if they become infected meningitis may arise when there is a connection with the spinal meninges. Patients may first be seen with meningitis which leads to the discovery of an inflamed sinus as the primary source of infection. Treatment of these sinuses begins with keeping the region as clean as possible. If any infection occurs appropriate antibiotics are given and the tract is later excised. Deep connections are divided and if necessary sealed. Unnecessary interference with the tract by probing should be avoided lest infection be introduced. With ordinary standards of hygiene most of these dermal sinuses remain free from infection.

**Pilonidal Sinus.** This condition is considered to be acquired. It is discussed in another section.

## CHAPTER 22

### SOME ACUTE ABDOMINAL CONDITIONS IN CHILDREN

#### ACUTE APPENDICITIS IN INFANCY AND CHILDHOOD

ACUTE appendicitis is the commonest acute abdominal condition in childhood but, whereas the *total* death rate of the disease has steadily declined, there has been no corresponding improvement in the mortality figures in infants under the age of five years. This is because the clinical manifestations of the disease in children are protean and differ considerably from those occurring in adults ; therefore the diagnosis may be delayed or even missed. Furthermore, children often develop infections and gastrointestinal disturbances which subside without surgical intervention.

**Pathology.** The pathology of acute appendicitis is the same in children as in adults (see Chap. 1).

**Symptoms in Infants.** It is impossible to elicit an accurate history in the very young, but at the age of five years children give a fair description of symptoms. The classical history of central abdominal pain *followed by* pain localised to the right iliac fossa, is rarely obtained. The pain, which is often colicky, may appear to be widespread or localised to the right side of the abdomen. Vomiting usually occurs later, but may be absent or occasionally it may precede the onset of abdominal pain. Constipation or diarrhoea may occur. Occasionally, the drastic effect of a purgative adversely affects the clinical picture. Sometimes persistent vomiting may be associated with diarrhoea and lead to a diagnosis of gastro-enteritis. This grave error is most likely to be made when the inflamed appendix lies in the pelvis. Frequency of micturition or even retention of urine occurs if the appendix is lying in contact with the bladder. There may be anorexia, lassitude, or irritability. Tonsillitis or upper respiratory infection may precede or coincide with appendicitis in infants.

Sometimes in children the initial symptoms are so vague that the doctor is not called until complications have developed. It is estimated that over 50 per cent. of the children who undergo operation for acute appendicitis have an appendix abscess or peritonitis. Antibiotics tend to mask the symptoms. Acute appendicitis should always be suspected in children with pains anywhere in the abdomen.

**Physical Signs.** In young children suffering from acute appendicitis, the temperature tends to be somewhat higher than in adults—up to 103° F. Once the co-operation of the child is gained, gentle and careful examination can be performed. Abdominal tenderness, which is slight

at first, increases within a few hours. As in adults, the abdominal signs and site of maximum tenderness vary respectively with the stage of the disease and the location of the inflamed appendix (see under Appendicitis in Adults). Tests for hyperalgesia and rebound tenderness are unlikely to help in children owing to lack of necessary co-operation. *Rectal examination is of the utmost importance*, for an inflamed appendix in the pelvis causes tenderness and/or induration on the affected side; ballooning of the rectum is found in pelvic peritonitis and gastroenteritis. Fluid faeces may be found in either condition, but in the latter they are very offensive. Initial abdominal signs are often slight, if the appendix is deeply placed in the pelvis or behind the caecum. Following rupture of an inflamed appendix, abdominal pain and tenderness temporarily diminish; this deceptive presentation may delay the diagnosis. If, at first, the evidence of appendicitis is inconclusive frequent repeated examinations are carried out until a confident opinion can be given. *The most valuable and constant sign is local tenderness over the appendix wherever it may lie.* In some cases it will be "wiser to look and see than to wait and see."

**Differential Diagnosis.** *Acute non-specific mesenteric adenitis* is the disease most likely to be confused with acute appendicitis (*vide infra*). Abdominal pain and guarding and even vomiting are sometimes caused by *pneumonia*. Both clinical and radiological examination of the chest should always be carried out in children presenting with obscure abdominal pain. *Pneumococcal Peritonitis.*—This disease which is now rare occurs more often in children than adults, and more often in girls than in boys. The disease may occur in association with pneumonia but usually no other pneumococcal infection is found. *Blood culture is positive in about half the cases.* The onset occurs suddenly with a high temperature. There is persistent vomiting and diarrhoea and general tenderness and rigidity of the abdomen. It is not possible to diagnose the disease with sufficient certainty to justify conservative treatment. The majority of patients with this disease are therefore submitted to laparotomy. At operation typical green pus is found, if the disease is sufficiently advanced. Penicillin is given together with ■ sulphonamide. *Pyelitis* causes pain in the loin and vomiting and thus may be mistaken for inflammation of ■ high retrocaecal appendix or one arising from a maldescended caecum. *Hydro-nephrosis* is occasionally mistaken for appendicitis; the pain and tenderness affect mostly the upper abdomen and sometimes the smooth distended pelvis of the kidney can be felt. Vomiting is a prominent feature in such cases.

**Treatment.** Operation should be carried out as soon as possible, but if the child is dehydrated and toxic, preliminary restoration of fluid and electrolyte balance is essential. A gastric catheter should be passed and left in place if the child has been vomiting. Thus, the stomach can be kept empty by periodic suction through the tube, and thereby the risk of

inhalation of vomitus when regaining consciousness after the anæsthetic avoided. The operation is similar to that performed in adults. When the diagnosis is certain, a muscle-splitting (gridiron) incision is used. It is made high or low in the right iliac fossa depending on the site of the appendix. It is important to palpate the abdomen when the child is anæsthetised and before the incision is made, for the appendix or the omentum around it can usually be felt. This incision usually gives easy access and can be extended medially or laterally as desired. A paramedian incision is used if there is any doubt about the diagnosis of appendicitis. If the inflammation is confined to the appendix, appendicectomy is performed, and the wound is closed without drainage. If the appendix is perforated and there is gross soiling of the peritoneal cavity, the appendix is removed and the pus is gently sucked out and the appendix area is drained. The need for drainage is debatable; some surgeons advise closure of the abdomen in all cases. In children this practice is unwise. Drainage of the peritoneal cavity does little harm and the track of the drain acts as a line of least resistance for the release of any collection of pus in the vicinity. If peritonitis is present, appropriate antibiotics are given (Antibiotic Treatment, Chap. 110).

Post-operative management of general peritonitis aims at resting the alimentary tract until signs of recovery of intestinal movements have returned. Therefore in the immediate post-operative period, the stomach is kept empty by intermittent aspiration until only small amounts of fluid are withdrawn, and intravenous fluid therapy is given to restore electrolyte, water and protein loss. Oral feeding begins with fluids and is introduced slowly when the bowel activity returns.

Conservative treatment has no place in the management of the appendix abscess in children, for with modern methods of control of infection and fluid balance, operation is safe at any stage of acute appendicitis. Removal of the appendix and drainage of an abscess, when present, should be carried out, except that the appendix is left in place in the rare event of its being firmly adherent to surrounding structures. Removal is then postponed for two to three months when dissection is likely to be easier. In the majority of cases, however, simultaneous removal of the appendix and drainage are possible. The appendix is followed distally into the mass of omentum with the finger, and if an abscess is present it is opened with minimum disturbance to surrounding structures. After aspiration of the pus, the remains of the appendix, with any fecolith are removed. The site of an abscess is drained for forty-eight hours. Occasionally, late cases of appendicitis, possibly suppressed previously by antibiotics, come to operation. In these cases, firm adhesions in the appendix region, with or without abscess formation, render appendicectomy difficult or impossible, and considerably prolong convalescence.

Complications following operation for appendicitis in children are similar to those occurring in adults.

## ACUTE MESENTERIC ADENITIS IN CHILDREN

This disease occurs commonly in children and is easily mistaken for acute appendicitis. It affects children from the age of three years onwards, and occurs more often in males than females. The enlargement of the mesenteric lymph glands is a local manifestation of a generalised non-specific infection. The temperature is usually  $100^{\circ}$  to  $102^{\circ}$  F., and the pulse rate is increased. There is usually a leucocytosis exceeding 20,000 polymorphs per cmm. The face is usually flushed and sometimes there is an associated upper respiratory tract infection, tonsillitis or cervical adenitis. Occasionally headache occurs. The abdominal pain is colicky. It is usually generalised but may be localised to the right lower abdomen or epigastrium. Vomiting may occur but bowel function is usually normal. A history of previous attacks of a similar nature is common. Tenderness and guarding in the right iliac fossa are found, but spontaneous rigidity does not occur. Sometimes tender glands can be felt in the ileocaecal region. The abdominal symptoms are probably caused by spasm of the intestine in relation to the enlarged glands. The characteristic features of the disease are the colic and the rise of temperature out of proportion to the mild abdominal signs. The severe symptoms subside in forty-eight hours or sooner. Persistence or worsening of symptoms means that the diagnosis is wrong. If at any stage of the illness there is the slightest doubt about the diagnosis, laparotomy should be carried out. At operation in acute mesenteric adenitis, there is an increase of the peritoneal fluid; the surface of the intestine is usually inflamed and the lymph nodes in the ileocaecal angle of the mesentery are enlarged and succulent. Usually the lymph glands are enlarged all along the superior mesenteric vessels. The appendix, although of normal appearance, is removed to eliminate the possibility of future appendicitis. Sometimes the lower end of the ileum feels thicker than normal. The nature of the mesenteric lymphadenopathy is unknown, for microscopy and bacteriological examination of the hyperplastic glands fail to reveal any specific disease. It is probably a virus infection. Recurrence of the disease is common, although it occurs less often after appendicectomy than following conservative management, despite the normal histology of the appendix. The ultimate prognosis is almost invariably good. Symptoms usually disappear in a few months. The tubercle bacillus is the cause of the adenitis in less than 2 per cent. of cases.

## ACUTE INTUSSUSCEPTION IN CHILDHOOD

This condition commonly occurs between the ages of three to nine months. Males are more often affected than females in the proportion of 5 to 4. The children, who develop the condition, are characteristically well nourished. The cause is unknown in most cases although it is thought that the change from liquid to solid food or unsuitable diet may

cause irregular peristalsis, which may initiate intussusception. Sometimes hypertrophy of the submucous lymphoid tissue, a polyp or a Meckel's diverticulum may be found at the apex of an intussusception. The intestine behaves as if it is attempting to expel the abnormality of its wall. An intussusception is formed by three layers of bowel, namely, the entering and returning layers constituting the intussusceptum and the outer ensheathing layer, called the *intussusciens* (Fig. 162). The apex, propelled distally by peristalsis of the ensheathing layer, progresses as far as the mesentery allows. At first, the intussusceptum advances and recedes with contraction and relaxation of the intestinal wall but later oedema prevents further advancement.

Various types of intussusception are described, namely, ileo-ileal, ileo-colic, ileocaecal and colocolic, according to the part of the intestine

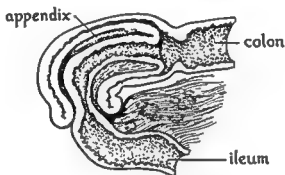


FIG. 162. The various layers of an ileocaecal intussusception. The position of the appendix is indicated.

involved. The ileocolic and ileocaecal varieties occur most often, together accounting for about 75 per cent. of acute intussusceptions. In these two types, the ileum enters the large bowel through the ileocaecal valve. In all types of intussusception, the attached mesentery is drawn through the neck of the ensheathing layer and becomes strangulated. The corresponding length of bowel becomes swollen, inflamed and later gangrenous. The lymphatic glands in the mesentery also become markedly enlarged. At the same time, various degrees of intestinal obstruction occur, depending on the diameter of the involved bowel and the degree of swelling of the intussusception. Sometimes, especially in older children, an intussusception may undergo spontaneous reduction, although usually the condition is progressive and, unless treated, complete intestinal obstruction develops and the affected bowel becomes gangrenous.

**Symptoms and Signs.** Attacks of abdominal colic occurring in an otherwise healthy infant suggest the onset of an intussusception. The attacks increase in duration and severity. During the spasm of pain, the child draws up its legs or gets up on its hands and knees on the bed. The

face becomes pale during an attack of pain. During the intervals, the child remains quiet and appears normal. Vomiting occurs at the onset in nearly all cases. At first the gastric contents are vomited, followed several hours later by bile or foul-smelling faecal-looking fluid. The passage of blood and mucus in the stool is characteristic. The amount of blood passed gives no indication of the severity or extent of the intussusception. Blood is seen in about half the cases in the first twenty-four hours,



FIG. 163. A barium enema in an infant showing the head of an intussusception as a rounded shadow in the proximal part of the transverse colon.

whereas in a quarter of the cases mucus is seen without blood. Occasionally neither blood nor mucus is present. The temperature varies from  $99^{\circ}$  to  $103^{\circ}$  F. In neglected cases, fatigue, listlessness and dehydration occur. Infants with this condition resent interference. Thus examination is difficult. At first the abdomen appears normal, but later there is abdominal distension. During an interval between attacks, the abdominal wall is relaxed. Usually the sausage-shaped intussusceptum is palpable along the line of the large intestine. It is most easily felt when lying in the transverse colon over the vertebral column, but it is difficult to feel in the regions of the hepatic and splenic flexures. The swelling may be

felt to contract with the spasm of pain, and may be induced to recede or advance. Sedation may help when examination is difficult. Absence of a palpable swelling does not exclude intussusception. Rectal examination is always done; blood and mucus are found on the examining finger; the rectum is sometimes ballooned and devoid of solid faeces. The head of the intussusception may be felt in the pelvic colon.

Radiography is unnecessary as a routine diagnostic aid, but occasionally in recurrent intussusception in older children having infrequent or mild symptoms, a barium enema is useful, for the outline of the head of the intussusception may be shown as a round cup-shaped filling defect (Fig. 163). The pressure of the barium may partially or completely reduce the intussusception. A free flow of barium into the ileum follows complete reduction.

**Differential Diagnosis.** Other conditions which may have to be considered are gastro-enteritis, purpura, Meckel's diverticulum and prolapse of the rectum (when the apex of an intussusception presents at the anus). In gastro-enteritis, there are mucoid watery and offensive stools. In purpura, subserous hæmorrhage in the bowel causes abdominal colic and blood in the stools (Henoch's purpura). Although a purpuric skin eruption is often observed, it appears two to three days after the abdominal symptoms and is therefore of no immediate value in diagnosis. Occasionally the length of hæmorrhagic bowel becomes intussuscepted, giving rise to a palpable swelling. This serious complication should be considered in all cases of Henoch's purpura. A Meckel's diverticulum, if ulcerated, may simulate an intussusception by causing bleeding and abdominal colic. In prolapsus ani, which is sometimes mistaken for an intussusception, the prolapsed rectal wall is continuous with the encircling anal margin, whereas in intussusception protruding from the anus, the finger can be insinuated between the presenting bowel and the anal margin.

**Treatment.** Acute intussusception is treated by immediate operation. When necessary parenteral fluids are given (see Chap. 23). The stomach is always emptied by means of an indwelling gastric tube. The operation is performed under general anaesthesia. A right lower paramedian incision is used. When the abdomen is opened, the cæcum is found to be displaced from its usual position. The intussusception is located by introducing two fingers into the abdomen, and by gently squeezing the bowel just distal to the intussusception, it is slowly reduced (Fig. 164A). Occasionally complete intra-abdominal reduction can be achieved, but more often the bowel is held up in the ileocæcal region and has to be delivered outside the abdomen and reduced under vision. Often that portion which protrudes through the ileocæcal valve is difficult to reduce and requires firm pressure on the ensheathing layer just distal to the head of the intussusception (Fig. 164B). Application of hot packs lessens the oedema and congestion of the intestine and assist the last part of the reduction. Usually the reduced bowel is healthy and intact. Occasion-



ally, however, the intussusception is irreducible, especially in small infants in whom treatment tends to be delayed. Resection of the bowel in one or two stages is necessary. Primary anastomosis then is the best method, but removal of the whole length of bruised bowel is necessary to ensure healing of the anastomosis. The strangulation of the mesenteric vessels caused by the intussusception jeopardises the blood supply of the

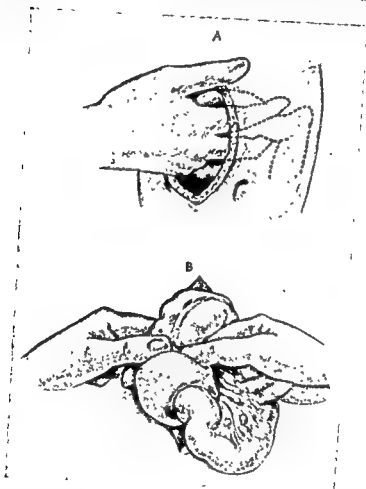


FIG. 164. A. Showing the method of reducing the intussusception by intra-abdominal manipulation. B. The last few inches of the intussusception, which are the most difficult to reduce, have to be brought under direct vision outside the abdomen.

ileocaecal region and most of the ascending colon; all of this part of the intestine must therefore be removed. If, however, the infant's condition is poor, the irreducible bowel is exteriorised after approximating the proximal and distal limbs of the affected segment which is then removed and a double-barrelled ileocolostomy established. This is closed four to five days later, either by end-to-end anastomosis after opening the abdomen, or by suturing the edges of the open bowel after crushing the tissue separating the two limbs of the ileocolostomy with an enterotome. Convalescence is usually uneventful when the intussusception is reducible.

After resection, maintenance of fluid and electrolyte balance is of great importance. Recurrence of intussusception occurs in up to 8 per cent. of cases; it may recur within a few days or months of the operation and is usually associated with a primitive mesentery (i.e. the ileocaecal region has not become fixed to the right posterolateral wall of the abdomen). The site of recurrence is not necessarily the same as that of the original lesion.

**Conservative Treatment.** Reduction of acute intussusception by means of a barium enema is not popular in Great Britain, but good results of this method have come from Scandinavia and Australia. The chances of reduction are best if the conservative method is restricted to infants over six months old who have had symptoms for less than twenty-four hours (Zachary, 1935). This treatment should be used only when adequate facilities are available. Warm barium is run into the rectum from a container held not more than 3 feet (1 metre) above the anus through a non-lubricated rectal tube. The advance of the barium is observed under the X-ray screen. In successful cases, the head of the intussusception recedes towards the caecum and finally the barium flows freely into the ileum. If reduction is uncertain or unsuccessful, immediate operation is carried out. This method should not be used for seriously ill children.

**Results.** Successful results depend on early diagnosis and immediate treatment. Delay beyond twenty-four hours carries the risk of irreducibility and complete intestinal obstruction. Resection of bowel in a severely ill child increases the mortality at least tenfold.

**Chronic Intussusception.** Symptoms are intermittent and last several days or even weeks. They are recurrent colic and occasionally the passing of blood per rectum. There may or may not be a palpable abdominal mass. The symptoms are caused by repeated intussusception and spontaneous reduction. Chronic intussusception occurs in older children and adults more often than the acute type. The head of the intussusception is usually formed by an innocent tumour in the young and a Meckel's diverticulum or a carcinoma in adults. This condition should be borne in mind in cases of recurrent abdominal pain; sometimes the symptoms subside without operation, but when operation is required the causal lesion in the bowel is discovered.

### FOREIGN BODIES IN THE ALIMENTARY TRACT

Young infants show little discrimination in the choice of objects which they put in their mouths and swallow. Occasionally, the loss of the swallowed article may not be traced until the child develops symptoms. Hair grips, pins, needles, coins, small toys, ornaments, dried fruit and strands of hair are often swallowed.

Foreign bodies may be held up at any level in the alimentary tract, but the common sites are the post-cricoid region of the oesophagus, the pylorus, the ileocaecal valve and the anal opening. When a child presents

with a history of having swallowed an article, X-ray examination is always necessary. Most dangerous foreign bodies such as pins are radio-opaque and, if seen lying in the œsophagus, should be removed through an œsophagoscope. In the case of blunt objects, sedatives may be given in the hope that relaxation of œsophageal spasm may enable them to pass downwards. Radiotranslucent foreign bodies such as fish bones, wood splinters and glass, usually cause the sensation of something sticking in the gullet. Sometimes the site of hold up can be seen radiologically after the patient has swallowed a piece of cotton wool impregnated with barium. However, œsophagoscopy is imperative if a sharp object is known to have been swallowed, for penetration of the œsophagus is likely, and would cause serious complications. These can be prevented by early removal through an œsophagoscope.

If a foreign body reaches the stomach it will probably be passed naturally. The choice between conservative or operative treatment depends on the nature of the foreign body. In the case of blunt objects conservative treatment is tried first. Their position and progress are ascertained by radiological examination. Conservative treatment consists in giving bulky food, such as porridge, to envelope the object. Bowel lubricants (*not* purgatives) such as liquid paraffin or agar preparations may also be given. All the stools are examined until the object is found. If progress is slow, or if the object is held up anywhere, exploration should not be delayed longer than two to three weeks from the time the object is swallowed. Sharp things are removed without delay.

**Operative Treatment.** A preoperative radiograph of the abdomen should be taken to indicate the site to be explored. If the object lies in the stomach it can be removed by making a small incision in the gastric wall. If it is held up in the duodenum—it is here that a hair grip is most likely to be found—the foreign body is manipulated back through the pylorus and removed through a small gastric incision. Blunt objects held up at the ileocaecal valve are best manipulated through this opening into the cæcum and allowed to pass naturally. If this is not possible removal is carried out through a small opening made in the ileum. Some irregular-shaped foreign bodies, having passed through most of the alimentary tract, are finally held up at the anus and have to be removed with the aid of a proctoscope. Rabbit bones are occasionally held up here and if neglected may give rise to perirectal abscesses and fistulae.

## CHAPTER 23

# FLUID REQUIREMENTS FOR INFANTS AND CHILDREN UNDERGOING SURGICAL TREATMENT

by

W. F. YOUNG

THE general principles of the management of intravenous fluid therapy in infants are similar to those applying to adults (see chapter on Reactions to Injury), but the metabolic needs of the young child differ in the following ways : (a) Water exchange in the infant is normally three to four times as rapid as in the adult (Gamble). (b) Severe states of dehydration occur early and frequently among young infants because the volumes of any abnormal losses from the bowel and of the normal excretions by the skin and lungs are both large relative to the body weight. Electrolyte deficits are also likely to be relatively great. (c) The new-born infant's kidneys are unable to secrete urine as efficiently as those of adults. (d) The young child does not seem to be affected by the immediate reaction to operation occurring in adults in whom there is water retention and consequent oliguria. A small urinary output persists only until the volume of the body fluids has been restored. Thus in infancy oliguria can usually be assumed to reflect unrelieved dehydration, calling for a high fluid intake.

**Principles of Treatment.** Fluid therapy aims to : (1) Restore the circulating blood volume ; (2) establish and maintain a free flow of urine ; (3) restore and maintain the volume and composition of the body fluids ; (4) replace abnormal losses. The history and clinical condition indicate the extent of a previous deficient intake and abnormal losses. In intestinal obstruction pooling in the proximal bowel causes a large loss of *sodium* from the body fluids. After relief of the obstruction rapid absorption of the fluid from the bowel may embarrass the circulation. At first the blood volume is restored rapidly with solutions of glucose and sodium salts without potassium (to avoid potassium intoxication). Infants and children with severe blood loss and those suffering from malnutrition or anaemia should be given plasma and/or blood at this stage.

As soon as a free flow of urine is established potassium salts are added to the infusion fluids. Restoration or avoidance of a severe deficit of intracellular electrolytes (potassium salts) prevents the distortion of body fluids arising from giving sodium without potassium and lessens the risks of *sodium* retention and formation of oedema. The administration

of potassium with sodium salts is safe provided that a dilute solution is used, the concentration of which should not exceed 24 m. equivalents per litre, and a total of not more than 3 m. equivalents per kilogram body weight per twenty-four hours is given. A free flow of urine must be maintained. These conditions are easy to fulfil in children because the daily volume of infusion fluid is proportionately large compared with that given to adults.

Laboratory investigations are usually unnecessary in straightforward cases when patients tolerate oral feeding within forty-eight hours of operation. The requirements are gauged by the clinical state, the history of fluid intake and assessment of any abnormal losses together with a check on the urine output. In more complicated cases, in which it is anticipated that oral feeding must be withheld for longer than forty-eight hours, initial estimations of the hæmoglobin percentage, packed cell volume, total plasma proteins, sodium, potassium, chlorides,  $\text{CO}_2$ , and blood urea are useful as a base line with which to compare future levels; subsequent estimations are advisable so that deviations from the normal may be corrected before the clinical state deteriorates. Changes in hæmoglobin, packed cell volume and total protein indicate either a change in volume of the circulating blood or in the amount of hæmoglobin or protein in the circulation (e.g. the amounts are diminished by infection or malnutrition). Blood urea estimations are a guide to renal function, and the plasma electrolyte levels are sometimes helpful in the choice and amount of solutions to be used. As in adults, the plasma sodium levels may be kept within normal limits by variation in water balance, and they are therefore of little help in management compared with records of intake and output of fluid. The plasma potassium should be checked regularly in patients receiving prolonged treatment with intravenous fluids, and the  $\text{CO}_2$  and chlorides in those with intestinal obstruction in order to detect incipient acidosis or alkalosis. A comparison of body weights from day to day is a useful check on fluid retention or loss.

**Regime.** A guide to the approximate requirements of infants and children is outlined below. Table 1 shows the composition of solutions commonly used for infusions, and includes that of certain potassium salts in concentrated solution for addition when necessary.

*Initial infusion for restoration of blood volume until urine flow is established.*—The solution to be used is commonly five per cent. dextrose in half isotonic saline giving a volume of 20 to 30 ml. per pound (40 to 60 ml. per kilo) body weight in three to six hours, or at this rate for shorter or longer periods until urine is passed freely. A slower rate of infusion should usually be given throughout a surgical operation. A different initial infusion is required as follows: (1) When conditions promoting shock are present, plasma diluted with an equal part of ten per cent. dextrose should be given. If shock is apparent, and in cases of severe operative trauma, undiluted plasma and/or blood is needed. (2) Where

TABLE 1

	Na	m. equivalent per litre K	Cl	HCO <sub>3</sub>
(a) <i>Saline solutions</i>				
Isotonic saline . . . . .	154	—	154	—
5 per cent. dextrose, $\frac{1}{2}$ isotonic saline . . . . .	77	—	77	—
5 per cent. dextrose, $\frac{1}{5}$ saline . . . . .	30	—	30	—
(b) <i>Multi-electrolyte Solution</i>				
5 per cent. dextrose, $\frac{2}{5}$ isotonic Darrow's . . . . .	48	14	42	21
(c) <i>Dilutions of Plasma</i>				
5 per cent. dextrose, $\frac{1}{2}$ strength plasma (10 per cent. dextrose and plasma in equal parts) . . . . .	70	2.5	53	14
(d) <i>Dilutions of Amigen</i>				
5 per cent. dextrose, $2\frac{1}{2}$ per cent. amigen (5 per cent. dextrose N/5 saline and 5 per cent. dextrose, 5 per cent. amigen in equal parts) . . . . .	31	6.4	23	—
(e) <i>Potassium supplements</i>	1 ampoule contains g. per 10 ml.	m. equiv. per 10 ml.	1 ampoule per $\frac{1}{2}$ litre = m. equiv. per litre	
Potassium chloride . . . . .	0.75	10	20	
Potassium acetate . . . . .	1.0	10	20	

gastro-intestinal losses have been large from vomiting, diarrhoea or "pooling" in dilated intestine, isotonic saline is given as part of the initial infusion to replace the losses of sodium chloride. (3) In cases of oesophageal atresia or in selective operations when the losses of sodium chloride have been slight, five per cent. dextrose in one-fifth isotonic saline is indicated.

*Subsequent infusions for the restoration and maintenance of the volume and composition of the body fluids, with sufficient excess to maintain a free flow of urine.*—Experience has shown that the volumes indicated in Table 2 usually meet the requirements of infants and children.

After dehydration has been relieved, new-born infants, during the first week of life, should be given less than the amount required by older babies. Each day after birth, one-seventh of the full requirement for older infants should be added by increments. Extra volumes to cover abnormal losses may be needed to prevent the recurrence of dehydration. The composition

of potassium with sodium salts is safe provided that a dilute solution is used, the concentration of which should not exceed 24 m. equivalents per litre, and a total of not more than 3 m. equivalents per kilogram body weight per twenty-four hours is given. A free flow of urine must be maintained. These conditions are easy to fulfil in children because the daily volume of infusion fluid is proportionately large compared with that given to adults.

Laboratory investigations are usually unnecessary in straightforward cases when patients tolerate oral feeding within forty-eight hours of operation. The requirements are gauged by the clinical state, the history of fluid intake and assessment of any abnormal losses together with a check on the urine output. In more complicated cases, in which it is anticipated that oral feeding must be withheld for longer than forty-eight hours, initial estimations of the haemoglobin percentage, packed cell volume, total plasma proteins, sodium, potassium, chlorides,  $\text{CO}_2$ , and blood urea are useful as a base line with which to compare future levels; subsequent estimations are advisable so that deviations from the normal may be corrected before the clinical state deteriorates. Changes in haemoglobin, packed cell volume and total protein indicate either a change in volume of the circulating blood or in the amount of haemoglobin or protein in the circulation (e.g. the amounts are diminished by infection or malnutrition). Blood urea estimations are a guide to renal function, and the plasma electrolyte levels are sometimes helpful in the choice and amount of solutions to be used. As in adults, the plasma sodium levels may be kept within normal limits by variation in water balance, and they are therefore of little help in management compared with records of intake and output of fluid. The plasma potassium should be checked regularly in patients receiving prolonged treatment with intravenous fluids, and the  $\text{CO}_2$  and chlorides in those with intestinal obstruction in order to detect incipient acidosis or alkalosis. A comparison of body weights from day to day is a useful check on fluid retention or loss.

**Regime.** A guide to the approximate requirements of infants and children is outlined below. Table 1 shows the composition of solutions commonly used for infusions, and includes that of certain potassium salts in concentrated solution for addition when necessary.

*Initial infusion for restoration of blood volume until urine flow is established.*—The solution to be used is commonly five per cent. dextrose in half isotonic saline giving a volume of 20 to 30 ml. per pound (40 to 60 ml. per kilo) body weight in three to six hours, or at this rate for shorter or longer periods until urine is passed freely. A slower rate of infusion should usually be given throughout a surgical operation. A different initial infusion is required as follows: (1) When conditions promoting shock are present, plasma diluted with an equal part of ten per cent. dextrose should be given. If shock is apparent, and in cases of severe operative trauma, undiluted plasma and/or blood is needed. (2) Where

The clinician is guided by the appearance of the patient, the assessment of abnormal losses, and urine output and concentration, and sometimes the plasma electrolyte levels.

*Plasma and Amigen solutions* used to supply protein should be added after forty-eight hours of intravenous therapy if oral feeding is delayed; the infant should have at least 1.0 gm. protein (or protein equivalent) per lb. (2.0 gm. per kilo) body weight in twenty-four hours and older children less. The composition of these solutions is shown in Table 1. Potassium salts should be added in smaller amounts than those used for supplementing saline solutions in order to raise the concentration of

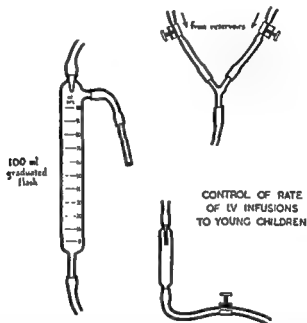


FIG. 165. Queen Elizabeth Hospital for Children pattern of graduated flask for use in intravenous therapy in infants. This enables accurate, measured amounts to be given. This apparatus is inserted between the reservoirs above, which contain the appropriate infusion fluid, and the drip bulb below.

potassium to about 20 m. equivalent per litre. Blood transfusions should be given if the patient becomes anaemic; 10 ml. per lb. (20 ml. per kilo) body weight can be expected to raise the haemoglobin level about 25 per cent. (Haldane).

*Antibiotics* are advisable to minimise the chances of infection which tends to be introduced by intravenous therapy.

*Vitamins* (ascorbic acid, the B complex and vitamin K) should be given intramuscularly on alternate days to patients receiving intravenous infusions for more than two days.

*Apparatus* There is a danger that excessive amounts of fluid may be given to infants if the usual  $\frac{1}{2}$ -litre flask is used. Therefore a small measuring flask, such as illustrated in Fig. 165, should be employed.



TABLE 2

Weight lb. (Kg)	Age year	Volume per 24 hours. ml.	Volume per hour. ml.
3 (1.4)		200- 240	10
4 (1.8)		240- 320	15
5 (2.3)		375- 500	20
6 (2.7)		400- 600	22
7 (3.2)		525- 700	25
8 (3.7)		600- 800	30
9 (4.1)		685- 900	35
10 (4.6)		750-1,000	40
11 (5)		825-1,100	45
12 (5.4)		900-1,200	47
13 (5.9)		975-1,300	50
14 (6.4)		1,050-1,400	55
15 (6.8)		1,125-1,500	60
	and up to 2 years		
	2- 5 "	about 1,750	70
	5- 7 "	" 2,000	80
	7-10 "	" 2,500	100

Table 2, showing volume of fluid per twenty-four hours and hourly to be given according to weight and age. *For an infant under seven days old divide the twenty-four hour volume by seven and multiply by his age in days.*

of the fluids depends on the clinical state of the patient and assessment of the composition and volume of the abnormal losses. Five per cent. dextrose in one-fifth isotonic saline with potassium chloride or acetate (one ampoule, containing 10 mEq. is added to each half litre) is suitable for patients if their dehydration has been relieved completely. One litre of this solution (the amount given to a 10- or 12-lb. infant) supplies the quantity of sodium chloride contained in 200 ml. isotonic saline, and this is often sufficient to cover abnormal losses by gastric suction as well as the small normal output through the skin. Until dehydration is relieved, or if the amounts of gastric suction or intestinal pooling are very large, the losses must be replaced by the addition of isotonic saline. Patients suffering from high intestinal obstruction, losing gastric contents including large amounts of chloride, require the addition of potassium chloride whereas in those with greater intestinal losses, the addition of potassium acetate is preferable.

A multi-electrolyte solution such as 5 per cent. dextrose in two-fifths isotonic Darrow's solution (Table 1) is useful in providing sodium and potassium with both chloride and bicarbonate equivalents in cases of paralytic ileus with variable losses. If obstruction is prolonged, addition of potassium salts to this solution is usually needed.

Sometimes on abdominal examination the line of fusion can be felt lying across the vertebral column in the midline. Crossed fusion (*crossed ectopia*), in which both kidneys are joined together on one side, is very rare ; in these cases the upper ends of the ureters and pelves lie adjacent on one side, but the distal end of the ureter of an ectopic kidney crosses the midline to enter the bladder normally.



FIG. 167. Pyelogram showing the pelvis of a left ectopic kidney lying over the ala of the sacrum.

**Ectopia of the Kidneys.** The kidney fails to "ascend" to the lumbar region during development, and thereby retains its primitive blood supply from the iliac vessels or the lower part of the abdominal aorta (Fig. 167) ; the corresponding ureter remains short, in contrast with a *ptosed kidney* in which the ureter is normal in length. A palpable swelling in this low position is liable to cause confusion in diagnosis ; thus a diseased ectopic kidney in the iliac or presacral position may be mistaken for an appendix abscess, mesenteric cyst, ovarian cyst, or a tumour lying in the pelvis.

**Cystic Changes in the Kidney.** The embryological explanation of renal

## CHAPTER 24

### CONGENITAL ABNORMALITIES OF THE URINARY TRACT

"Congenital abnormality is important rather by reason of its complications than on its own account, and where it is encountered care should be taken to exclude disease in all component parts of the upper urinary tract."—J. B. MACALPINE, 1940.

#### THE KIDNEY

Failure of development of the kidney—renal agenesis—may affect one or both sides; bilateral agenesis is seen only *post mortem*. The solitary kidney, which is often hypertrophied, may give rise to renal failure early in life. Failure of the renal (Wolffian) bud and/or the nephrogenic (metanephric) cap to develop is often associated with absence of the ipsolateral ureteric opening and lack of development of the corresponding half of the trigone of the bladder. The recognition of a solitary kidney is of paramount importance before considering nephrectomy for injury or disease, lest the one and only organ be removed.

Hypoplasia of the kidney results from underdevelopment of the nephrogenic cap (metanephros), and gives rise to a thin sac-like kidney surrounding an immature pelvis and immature calices. Excretion of urine is deficient. Inflammation or ischaemia of a hypoplastic kidney leads to atrophy with fibrous or cystic changes. In unilateral cases

arterial hypertension may occur, whereas in bilateral lesions renal failure and renal dwarfism develop.

**Fusion of the Kidneys.** The kidneys may be fused at their upper or lower poles, being joined together across the midline in front of the great abdominal vessels (*horse-shoe kidneys*). Very rarely a *discoid kidney* is formed by fusion at both upper and lower ends. These anomalies can cause vague abdominal pain, urinary infection, or stone formation. In the horse-shoe deformity the axes of the kidneys in a urogram form a V, and the outline of each pelvis shows a characteristic medial calix (Fig. 166).

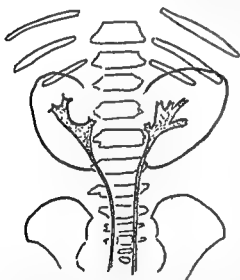


Fig. 166. Diagram of a pyelogram of a horse-shoe kidney. Note the medial calices on both sides and the V-shaped arrangement of the axes of the kidneys.

the first evidence of the tumour arises from pulmonary metastasis. Examination reveals a firm renal tumour with smooth lobulations on its surface. It extends anteriorly towards the midline. On the right the upper border of the tumour is separate from the liver. Fixity of the growth usually prevents its moving with respiration. Frequent abdominal palpation should be avoided for fear of causing metastasis. Blood may be seen in the urine on microscopic or naked-eye examination. A plain radiograph of the abdomen shows the outline of the tumour and displace-

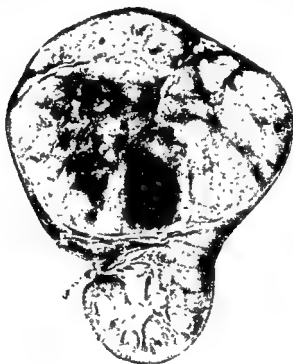


FIG. 168. Cross section of a Wilms' tumour.

ment of bowel shadows. Intravenous pyelography is, however, necessary to demonstrate a functioning contralateral kidney and the extent of involvement of the affected renal pelvis. Sometimes there is no excretion of contrast medium on the diseased side. In children below the age of two years the contrast medium (diodone) may be injected subcutaneously and hyaluronidase added to aid its dispersion. A solution containing 1 ml. of Hyalase (1,000 Benger units of hyaluronidase in 1 ml. of distilled water) and 10 ml. of 35 per cent. diodone is made up to 30 ml. with distilled water. Six to 10 ml. are injected subcutaneously into the gluteal region on each side and the areas of injection gently massaged to aid absorption. The diodone is excreted in good concentration twenty-five to forty-five minutes after injection. Neither retrograde pyelography nor

cyst formation is uncertain and complex. A disturbance of the potentialities of the formative tissue of the metanephric blastema and of the ureteral upgrowth is probable.

*Congenital cystic disease of the kidneys* may be unilateral or bilateral. Death occurs in early life, for the kidneys are hypoplastic. The diffuse multiple cysts give the kidneys a sponge-like appearance. *Polycystic kidneys* as seen in adults (see Chap. 26) are probably a different entity from that which affects the new-born, and are generally held to be due to failure of coalescence of the secreting and collecting systems of the kidney.

*Solitary and multilocular cysts* are very rare in children. They are probably due to distension of sequestered renal tubules. They are always solitary lesions within otherwise normal kidneys. Multilocular cyst formation differs from the polycystic kidney of adults, for in the latter there is normal renal tissue between the cysts, whereas in the former the loculi are adjacent. The solitary cyst, which presents as a renal swelling and occasionally with hæmaturia, is seen in the pyelogram as a smooth filling defect in the pelvis and/or calices. Sometimes this type of cyst is diagnosed as a renal neoplasm, and even at operation the distinction may not be obvious. Solitary cysts are best treated by local excision, but if the rest of the kidney is not worth saving nephrectomy is preferable.

*Cystic degeneration* which occurs secondary to chronic inflammation is distinct from all the cystic formations described above.

**Wilms' Tumour** (*Syn. Renal Embryoma or Nephroblastoma*). This renal tumour is one of the commonest malignant neoplasms occurring in children. It arises in the kidney in which it is surrounded by a false capsule of fibrous and compressed renal tissue. The tumour is smoothly lobulated. Its cut surface shows areas of hæmorrhage and necrosis (Fig. 168). The renal pelvis and surrounding structures soon become infiltrated, and invasion of the renal veins leads to metastasis in the lung; lymphatic spread also occurs. Wilms' tumour probably arises by aberrant differentiation of embryonic renal tissue. It consists of both epithelial and connective tissue. The former is composed of what appear to be primitive renal tubules and abortive glomeruli, and the latter is composed variously of undifferentiated mesenchyme, differentiated fibrous tissue, muscle, and sometimes cartilage, or even bone. There is a great variation in the histology, including the degree of cellular differentiation. Wilms' tumour arises most commonly in children before they reach the age of three years. It affects males twice as often as females. Rarely both kidneys are involved.

The majority of Wilms' tumours are symptomless, being noticed by the parents as a swelling of the abdomen. Often the tumours are very large before they attract attention. In about a quarter of the cases there is painless hæmaturia, indicating invasion of the kidney pelvis; sometimes

a quarter of the cases. When there is a complete double ureter the channel from the *lower* renal element enters the bladder *above* that of the other. Duplication *per se* does not cause symptoms, but urinary infection is liable to arise in the presence of any congenital abnormality of the urinary tract. Radiologically the associated double kidney is suggested by an elongated renal shadow, and the pyelogram may show duplication of the pelves and ureters, but more often one renal moiety does not fill. Coexistent infection usually affects a single element of the duplication; thus partial nephrectomy with the corresponding ureter is sufficient to cure the disease in most cases.

*Ectopia of the Ureter.* Embryologically the close relationship of the ureteric bud and the Wolffian duct derivatives explains the variety of ectopic openings of the ureter. In the male, the ectopic ureter, which is usually duplicated, opens into the urethra or the seminal vesicle, whereas in the female the abnormal ureter opens into the urethra or the vagina. This anomaly causes dribbling incontinence, but the normal desire to micturate is present, for the other ureter(s) opens into the bladder except in the very rare bilateral cases (see also under Enuresis). External examination may reveal the ectopic opening in females, but it is not always easily visible. An intravenous pyelogram may demonstrate the presence of double ureters. If not, duplication is suggested by a deficiency of visible calices on one side, outward displacement of the upper pole of the kidney, or shortening of the upper calix and displacement of the pelvis and upper ureter laterally. In cases of difficulty, the ureter is exposed by operation, and its ectopic opening demonstrated by injecting a radio-opaque medium into the lumen of the ureter or passing a ureteric catheter down the abdominal part of the ureter. The ectopic ureter should be removed with the corresponding part of renal tissue above. In the rare cases of double ectopia, bilateral partial nephro-ureterectomy is necessary.

**Ureteral Obstruction.** A *ureterocele* is caused by narrowing of the ureteric orifice. It causes ballooning of the ureter into the bladder. The cystic swelling can be seen as a filling defect in a cystogram, or directly by cystoscopy. This lesion may obstruct the internal *urethral* opening and cause retention of urine, but more commonly, coexistent infection causes frequency of micturition and dysuria. Treatment consists of drainage of the ureter by incising the ureterocele with diathermy by the transurethral route, or if the swelling is large it is best incised at open operation.

*Congenital stricture of the ureter* at the pelvi-ureteric junction gives rise to hydronephrosis (Fig. 169) and one at the ureterovesical opening to hydroureter. Less commonly, strictures arise between these sites. Consequent urinary infection and back pressure on the kidney cause renal damage. Sometimes vomiting and abdominal pain lead to unnecessary removal of the appendix. The diagnosis is made by excretory and retrograde pyelography, which show distension of the renal pelvis and/or

renal angiography are necessary. Radiography of the lungs is carried out to exclude pulmonary metastasis.

*Differential Diagnosis.* *Hydronephrosis* presents as a palpable swelling, but is smoother and softer than a neoplasm, and often varies in size. Pyelography elucidates the diagnosis. A renal cyst closely resembles a Wilms' tumour but occurs more rarely. Even at operation, a tense cyst may simulate a solid growth. In renal angiography a cyst is shown as a circular avascular area, whereas a Wilms' tumour is shown as a vascular pattern with pooling of the contrast medium within its boundaries. A neuroblastoma causes downward and lateral displacement of the kidney, demonstrable by pyelography; the liver is often enlarged by metastasis and is difficult to distinguish from the original abdominal neoplasm. In contrast, Wilms' tumours metastasise in the lung rather than in the liver. An embryoma is smooth whereas a neuroblastoma is usually nodular.

*Treatment.* In the absence of evident metastasis and provided the contralateral kidney is functioning well, urgent removal of the diseased kidney should be performed. An extensive abdominal or abdomino-thoracic approach is necessary. If possible ligation of the renal pedicle should be carried out before the kidney tumour is handled; thus the danger of intravascular spread of the tumour is minimised. Adherent perinephric tissue including fat and peritoneum are removed with the kidney. After operation, an immediate course of radiotherapy is advisable to destroy any residual malignant tissue. Sometimes it is also given preoperatively to shrink a very large tumour to allow subsequent removal. Although Wilms' tumours are very radiosensitive, irradiation is not considered to be adequate alone. Children vary greatly in their reaction to radiotherapy. Therefore careful regulation of the irradiation is required to prevent serious leucopenia and also to avoid damage to the vertebrae and consequent retardation of spinal growth. After one course of radiotherapy, these tumours become radio-resistant.

*Prognosis.* The total mortality is about 80 per cent. Infants seen with a tumour before the age of one year have a better chance of survival than older children. A combination of early surgical treatment followed by radiotherapy gives the best results. If there is no recurrence within eighteen months of completion of treatment the chances of survival are good.

### THE URETER

*Failure of development* of the ureteric outgrowth from the Wolffian duct results in ureteric agenesis and atrophy of the corresponding trigonal portion of the bladder. Similarly the ureter is often atrophic in unilateral renal agenesis. Unilateral or bilateral *duplication of the ureter* is a common deformity in which the renal pelvis may also be duplicated on one or both sides. The abnormality results from an accessory ureteric outgrowth from the Wolffian duct. It is liable to cause ureteric obstruction in about

a quarter of the cases. When there is a complete double ureter the channel from the *lower* renal element enters the bladder *above* that of the other. Duplication *per se* does not cause symptoms, but urinary infection is liable to arise in the presence of any congenital abnormality of the urinary tract. Radiologically the associated double kidney is suggested by an elongated renal shadow, and the pyelogram may show duplication of the pelvis and ureters, but more often one renal moiety does not fill. Coexistent infection usually affects a single element of the duplication; thus partial nephrectomy with the corresponding ureter is sufficient to cure the disease in most cases.

*Ectopia of the Ureter.* Embryologically the close relationship of the ureteric bud and the Wolffian duct derivatives explains the variety of ectopic openings of the ureter. In the male, the ectopic ureter, which is usually duplicated, opens into the urethra or the seminal vesicle, whereas in the female the abnormal ureter opens into the urethra or the vagina. This anomaly causes dribbling incontinence, but the normal desire to micturate is present, for the other ureter(s) opens into the bladder except in the very rare bilateral cases (see also under Enuresis). External examination may reveal the ectopic opening in females, but it is not always easily visible. An intravenous pyelogram may demonstrate the presence of double ureters. If not, duplication is suggested by a deficiency of visible calices on one side, outward displacement of the upper pole of the kidney, or shortening of the upper calix and displacement of the pelvis and upper ureter laterally. In cases of difficulty, the ureter is exposed by operation, and its ectopic opening demonstrated by injecting a radio-opaque medium into the lumen of the ureter or passing a ureteric catheter down the abdominal part of the ureter. The ectopic ureter should be removed with the corresponding part of renal tissue above. In the rare cases of double ectopia, bilateral partial nephro-ureterectomy is necessary.

**Ureteral Obstruction.** A *ureterocele* is caused by narrowing of the ureteric orifice. It causes ballooning of the ureter into the bladder. The cystic swelling can be seen as a filling defect in a cystogram, or directly by cystoscopy. This lesion may obstruct the internal *urethral* opening and cause retention of urine, but more commonly, coexistent infection causes frequency of micturition and dysuria. Treatment consists of drainage of the ureter by incising the ureterocele with diathermy by the transurethral route, or if the swelling is large it is best incised at open operation.

*Congenital stricture of the ureter* at the pelvi-ureteric junction gives rise to hydronephrosis (Fig. 169) and one at the ureterovesical opening to hydroureter. Less commonly, strictures arise between these sites. Consequent urinary infection and back pressure on the kidney cause renal damage. Sometimes vomiting and abdominal pain lead to unnecessary removal of the appendix. The diagnosis is made by excretory and retrograde pyelography, which show distension of the renal pelvis and/or



wide tortuosity of the ureter, depending on the site of narrowing. Hydronephrosis due to congenital stricture is best treated by a plastic procedure (see under Hydronephrosis in Adults), unless the kidney is destroyed by back pressure and infection, when nephrectomy is necessary (provided the contralateral kidney is normal). In hydroureter, instrumental dilatation of the stricture is worth trying, but, if unsuccessful, a plastic operation, such as re-implantation of the ureter into the bladder, should



FIG. 169. Intravenous pyelogram showing hydronephrosis on the left side. The pelvis and the calices were still distended with contrast medium three hours after the injection, whereas the right kidney had ceased to excrete it.

be considered, although usually persistent infection and marked renal damage demand nephro-ureterectomy in unilateral cases.

*Congenital Dilatation of the Ureter (Megalo-ureter).*—This condition is considered to be due to neuromuscular dysfunction of the ureter, causing dilatation of the ureter with or without hydronephrosis. The lesion is distinct from ureteric dilatation due to an organic obstruction. In some cases, there is reflux of urine from the bladder on micturition demonstrable by cystography (Fig. 170). The wide dilatation of the ureter, extending into its lumbar part, and delay of emptying of the contrast medium is shown by intravenous and retrograde pyelography. At cystoscopy, hypertrophy of the bladder wall and a widely patent ureteric orifice may be seen. The associated urinary infection should be treated by chemo-

therapy; in addition voiding of the bladder should be carried out by "triple micturition," for in cases of megalo-ureter with ureteric reflux, much of the urine in the bladder flows upward into the dilated ureters during urination and afterwards re-enters the bladder. Therefore by repeated micturition the bladder and ureter can be completely emptied. Much benefit is obtained by this simple routine. In cases without reflux, conservative treatment is advisable and comprises the administration of urinary antiseptics and dilatation of the ureteric orifice.

*Obstruction of the ureter by an aberrant renal blood vessel* (usually an artery) is fairly common and accompanies a large proportion of cases of hydronephrosis. Early division of the vascular obstruction with separation of adventitious adhesions around the pelvi-ureteric junction relieves the hydronephrosis in about half the cases in childhood. But division of the aberrant artery should be avoided if it is likely to result in extensive ischaemia of the lower pole of the kidney; this can be judged beforehand by observing the extent of any vascular change when the vessel is temporarily compressed. In such cases, plastic procedures or nephrectomy are necessary (see Hydronephrosis, Chap. 26).



FIG. 170. A cystogram (oblique view) showing reflux of diiodone from the bladder up the ureter which is seen to be markedly distended. The bladder shadow is on the right side of the picture. The infant had a severe urinary infection which improved after removal of the hydronephrotic kidney and ureter (the disease was unilateral).

### THE BLADDER

*Exstrophy (ectopia vesicae)* is the commonest anomaly of the bladder and is discussed under *Abnormalities of the Anterior Abdominal Wall* (Chap. 15). A *persistent urachus* is due to the failure of closure of the allantois. If it is completely patent, there is a urinary fistula opening to the exterior at the umbilicus. If only the vesical part of the tract is open there is a diverticulum at the apex of the bladder. Rarely a urachal cyst develops between the obliterated ends of the urachal tract; the cyst may be palpable in the midline, or if infected it may discharge pus through a sinus, at or just below the umbilicus. The introduction of a radio-opaque medium into the sinus demonstrates its deep origin. Excision of the tract and closure of the fundus of the bladder is indicated.

*Congenital bladder neck obstruction* usually causes symptoms from infancy, but may escape notice until childhood or even adult life. Urinary infection, retention or dribbling of urine with overflow draws attention

to the condition. There is contracture of the bladder neck produced by fibrosis and muscle thickening of unknown cause. Urinary investigations which should include a cystogram show the posterior urethra to appear narrower than normal, and there is a marked ridge at the level of the bladder neck which alters little on micturition. As the contour of the normal bladder and urethra is so variable it is difficult to decide when an abnormality is present. The only definite way of diagnosing this lesion is to feel *per rectum* the thickening of the posterior ridge of the bladder-neck hypertrophy against an indwelling bougie or cystoscope. Cystoscopically the lesion is difficult or impossible to visualise. Dilatation of the urethral orifice, or removal of the hypertrophic ridge by endoscopic resection through a small-size instrument is advisable, but the results are not uniformly successful.

### THE URETHRA

*Atresia, congenital diverticula, double urethra, congenital fistulae* are all rare anomalies in this region. Urethral stenosis may occur at the external urinary meatus, and should be treated by meatotomy, which is discussed under *meatitis*. The meatal obstruction causes a thin urinary stream and also produces back pressure in the upper urinary tract. *Epispadias*, which results from the absence of the dorsal wall of the urethra, is described under *Abnormalities of the Abdominal Wall*.

#### Valvular Obstruction in the Posterior Urethra.

The commonest type of urethral valve occurs in the region of the verumontanum (Fig. 171).

Its cusps prevent the bladder from emptying completely and cause ballooning of the posterior urethra above. Congenital obstruction of the urethra by a valve in the new-born has serious consequences. The occasional patient who survives to early childhood presents with dribbling incontinence due to retention with overflow. The back pressure of the urine, which is often infected, produces dilatation of the upper urinary tract and severe renal damage (Fig. 172). On examination the bladder is palpably distended and cannot be emptied completely at will. A catheter can be passed easily into the bladder for the valve only interferes with the outflow. A cystogram shows the posterior urethra to be dilated, lying at the base of the vesical shadow. Ureteric reflux may also occur. The treatment of valvular obstruction in the new-born is very difficult owing to the small size of the urethra, preventing adequate instrumentation. In these cases, decompression of the urinary tract is best carried out by nephrostomy. A suprapubic cystostomy is of little value, for the bladder wall, when decompressed,

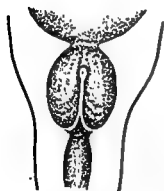


FIG. 171. The commonest type of valvular obstruction in the posterior urethra. The two cusps are centrally attached below the verumontanum.

shrinks markedly, causing obstruction to the ureteric outflow. In older children, who have survived infection and renal damage, fulguration of the cusps of the valve by means of an electrode relieves the local condition but does not restore the kidney damage.

**Hypospadias** is the commonest congenital urethral deformity, occurring in 1 in 500 males. It results from failure of closure of the urethral folds. The ventral part of the urethra may remain open anywhere along the



FIG. 172. Specimen of the urinary tract of a neonate showing gross changes due to valvular obstruction of the posterior urethra. Both kidneys are markedly hydronephrotic with very little renal tissue left. Both ureters are dilated and tortuous; the bladder is thin and distended. The left ureteric orifice is widely dilated. The posterior urethra is also dilated.

urethral groove. Thus there are three varieties, glandular, penile and perineal, determined by the site of the urethral orifice. The prepuce is incomplete, covering the glans penis like a hood (Fig. 173). Before correction of the deformity a preliminary meatotomy is usually necessary, for the abnormally placed orifice is often very small. Hypoplasia and fibrosis of the corpus spongiosum and shortening of the urethra give rise to chordee. This ventral curvature is corrected at about the age of eighteen months. A transverse incision is made proximal to the urethral opening and the ventral fibrous tissue is divided down to the corpora

cavernosa until the penis can be straightened. The meatus is then allowed to fall back towards the perineum and the wound is closed longitudinally. The next stage is to construct the urethral canal where it is deficient. This is usually undertaken at the age of four, although the age depends on the child's development. The bladder is first drained by an indwelling perineal catheter. The strip of the skin forming the



FIG. 173. Penile hypospadias. The prepuce is hooded and chordæ (deflection of the penis) is evident. (Mr. R. Battle's case.)

urethral groove is buried under a roof formed of adjacent lateral flaps of penile skin (Fig. 174). This operation produces ultimately a smooth, epithelialised urethra of even calibre and post-operative fistulae rarely occur.

**Recto-urethral and Ano-urethral Fistulae.** These occur in association with *imperforate rectum* or *covered anus*. In the recto-urethral fistula the rectum communicates with the posterior urethra, whereas in ano-urethral fistula the anus communicates with the bulbar part of the urethra (see under Malformations of the Rectum and Anus).

### THE PREPUCE

**Congenital Phimosis.** Normally, the prepuce in the new-born is closely adherent to the surface of the glans penis and gradually tapers towards

the preputial opening. This "congenital phimosis" is a natural anatomical finding in infancy. Gradual separation of the ensheathing foreskin from the underlying glans occurs with the increasing growth and activity of the child. At about the age of two years the prepuce can usually be fully retracted except at its base around the corona where there are a few adhesions. After retraction of the prepuce for washing, it should be pulled forward again without delay, for otherwise paraphimosis is likely to develop. As the penis grows, the foreskin becomes retractable without any undue tightness. The foreskin protects the end of the penis, preventing irritation of the glans and external urinary meatus. Occasionally separation of congenital adhesions by means of a probe is necessary. Once the foreskin is freed from the glans penis it remains separate, although its occasional retraction is advisable for cleansing. This conservative management aims at maintaining the normal protective covering of the penis and is becoming more and more accepted. Circumcision should be reserved for complications only. Nevertheless, circumcision, apart from ritual practice, is still routinely performed—

mostly by obstetricians—soon after the child's birth for "hygienic reasons."

**Acquired Phimosis.** Contraction of the preputial orifice, usually as the result of inflammation of the foreskin, may be so severe as to obstruct the flow of urine, causing ballooning of the prepuce on micturition. Rarely, urinary retention may occur if the minute preputial opening becomes blocked by smegma or a *postholith*. In acquired phimosis, retraction of the foreskin is usually impossible without splitting or stretching its

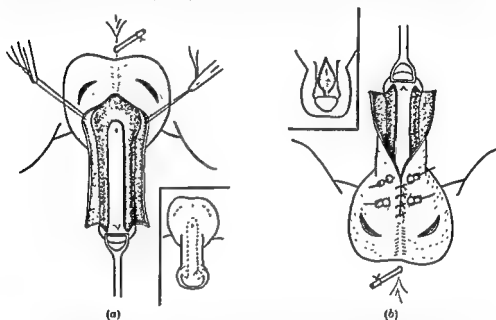


FIG. 174. Diagram showing plastic repair of hypospadias (Denis Browne's method). (a) An indwelling urethral catheter is secured in position in the perineum to drain the bladder. Next, the median strip of urethral skin is defined (see inset) and the surrounding skin flaps are widely raised. (b) A relaxation skin incision is made on the dorsum of the penis (see inset) so that the ventral flaps can be approximated without tension over the isolated urethral strip by double stop nylon sutures; the double stop, consisting of a glass bead and an aluminium sleeve threaded on the ends of the nylon stitch, is secured in position by crushing the metal cylinder upon the suture material; thus the skin edges which become adjacent can be stitched without tension by interrupted catgut sutures. The two scrotal incisions are for the relief of oedema after operation.

opening and thus causing further contraction. However, the phimosis can sometimes be overcome by slight daily retraction after freeing the adhesions, but circumcision or a dorsal slit is usually necessary.

**Paraphimosis.** This results from constriction of the glans penis by a retracted tight prepuce, and causes congestion and oedema of the strangulated ring of skin distal to the narrowing and ultimately ulceration of the skin at the constriction itself. This condition should be relieved without delay by pulling the foreskin forward over the glans by the manipulation depicted in Fig. 175. In severe cases, cold compresses or an injection of *hyaluronidase* will reduce the oedema (500 to 1,000 units of

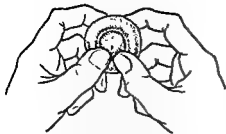


FIG. 175. Manipulation for reduction of paraphimosis.

hyaluronidase are dissolved in 2 ml. of 1 per cent. procaine and the solution is injected at several points near the constriction). Taxis is usually successful, but occasionally division of the constriction ring of the paraphimosis is necessary. Circumcision or a dorsal slit is required to prevent recurrence.

**Inflammation of the Prepuce (Posthitis).** This occurs in infancy and is

usually caused by contact of the foreskin with napkins wet with decomposing urine. The inflammatory changes are due to a diffuse ammoniacal dermatitis around the napkin area causing the prepuce to become sore and crusted. This condition responds to the treatment outlined below. *Ammoniacal dermatitis* affects the napkin area, and varies in severity from a superficial redness to desquamation or even ulceration (Fig. 176). It is due to irritation and burning of the skin by ammonia vapour, which is liberated by bacterial decomposition of urea, and can be readily smelt. This condition results from neglect to keep the area clean and infrequent changes of napkins, and is often aggravated by waterproof knickers. Cleanliness, including frequent changes of well-washed napkins, usually prevents this condition. Administration of an acid sodium phosphate mixture to acidify the urine clears up established dermatitis, for the bacterial decomposing action is inhibited by an acid medium. The condition rarely occurs in breast-fed babies.

**Balanitis.** This is defined as inflammation of the surface of the glans penis, and is caused by irritation and infection around the coronal margin. It is best treated by circumcision. It usually occurs in infants, but older children may also be affected. Balanitis in adults is discussed under *Genito-urinary Surgery*. The term "balanitis" is often misapplied to what is really inflammation of the prepuce (*posthitis*).

**Meatitis.** This is seen only in circumcised children. There is inflammation of the external urinary meatus. The margins of the opening are inflamed and ulcerated, and may be covered with crusts which occlude the meatus. The child holds his urine rather than suffer the discomfort



FIG. 176. Ammoniacal dermatitis.

of micturition. Separation of the crusts at the urethral orifice during micturition causes bleeding and is the commonest cause of hæmaturia in children. The avoidance of unnecessary circumcision in infancy is the best prophylaxis against this condition, for circumcision leaves the glans and its opening exposed to chaffing with wet napkins. Moreover, ammoniacal dermatitis further aggravates the condition. The affected area is bathed with a mild antiseptic lotion, or penicillin cream is applied. The urine should be acidified. The lips of the meatus are gummed together by inflammatory exudate. Therefore a dilator, such as a small eye-dropper glass rod, should be passed regularly to keep the opening patent. In neglected cases a mental ulcer leads to scarring and consequent reduction in the size of the orifice. In later years, if this obstruction is neglected, urinary infection and back pressure on the kidneys occur. *Meatotomy* should be carried out when the opening is so reduced that it cannot admit a normal-sized urethral bougie corresponding with the age of the child. The ventral lip of the meatus is cut with scissors downwards to one side of the midline—to avoid cutting the frænal artery—for a distance of about 1.5 cm. Either the cut edges are left gaping, and bleeding is controlled by pressure or, preferably, one or two hæmostatic catgut stitches are inserted along each edge of the incision (Fig. 177). After operation it is important to maintain the size of the external urethral opening by the regular passage of a glass rod or the tip of a urethral bougie, until the edges have healed and there is no sign of contraction.

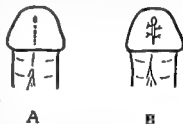


FIG. 177. Meatotomy. A. Dotted line indicates site of incision. B. After suturing the edges.

**Circumcision.** Ritual circumcision for racial and religious reasons has been practised at various ages for many centuries. Until recently circumcision was carried out frequently in infancy on hygienic and other grounds, but a better understanding of the protective function of the normal prepuce as described above has resulted in the operation being performed much less frequently. Apart from religious practices and such conditions as acquired phimosis, the operation is rarely necessary (in adults it should be carried out for balanitis, which may be secondary to an underlying ulcer, carcinoma, or warts of the penis). Circumcision is also advisable to prevent recurrence of paraphimosis. *The presence of ammoniacal dermatitis of the napkin area is a contraindication, for then the operation will certainly lead to balanitis and meatitis, perhaps with serious ulceration and cicatrization.* Carcinoma of the penis is a rare disease and does not occur at all in the circumcised (carcinoma of the cervix uteri is less common in circumcised races). Venereal disease is also comparatively infrequent in the circumcised. Nevertheless, these facts are not sufficient justification for routine circumcisions.



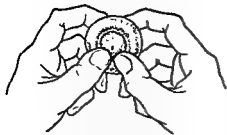


FIG. 176. Manipulation for reduction of paraphimosis.

hyaluronidase are dissolved in 2 ml. of 1 per cent. procaine and the solution is injected at several points near the constriction). Taxis is usually successful, but occasionally division of the constriction ring of the paraphimosis is necessary. Circumcision or a dorsal slit is required to prevent recurrence.

**Inflammation of the Prepuce (Posthitis).** This occurs in infancy and is

usually caused by contact of the foreskin with napkins wet with decomposing urine. The inflammatory changes are due to a diffuse ammoniacal dermatitis around the napkin area causing the prepuce to become sore and crusted. This condition responds to the treatment outlined below. *Ammoniacal dermatitis* affects the napkin area, and varies in severity from a superficial redness to desquamation or even ulceration (Fig. 176). It is due to irritation and burning of the skin by ammonia vapour, which is liberated by bacterial decomposition of urea, and can be readily smelt. This condition results from neglect to keep the area clean and infrequent changes of napkins, and is often aggravated by waterproof knickers. Cleanliness, including frequent changes of well-washed napkins, usually prevents this condition. Administration of an acid sodium phosphate mixture to acidify the urine clears up established dermatitis, for the bacterial decomposing action is inhibited by an acid medium. The condition rarely occurs in breast-fed babies.

**Balanitis.** This is defined as inflammation of the surface of the glans penis, and is caused by irritation and infection around the coronal margin. It is best treated by circumcision. It usually occurs in infants, but older children may also be affected. Balanitis in adults is discussed under *Genito-urinary Surgery*. The term "balanitis" is often misapplied to what is really inflammation of the prepuce (*posthitis*).

**Meatitis.** This is seen only in circumcised children. There is inflammation of the external urinary meatus. The margins of the opening are inflamed and ulcerated, and may be covered with crusts which occlude the meatus. The child holds his urine rather than suffer the discomfort



FIG. 176. Ammoniacal dermatitis.

micturition or ureteric reflux (Fig. 179). *Cystometry* (measurement of the intravesical pressure of water necessary to initiate the desire to micturate) is carried out by attaching a manometer to the end of an indwelling catheter, through which increasing volumes of water are introduced. In enuresis there may be differences in the urinary output and concentration by day and by night, but these findings are inconstant. *Cystoscopy* and *urethroscopy* may reveal such pathological states as trabeculation of the bladder wall, dilated or ectopic ureters, bladder-neck obstruction or urethral valves. *Electro-encephalography* may help to establish the epileptic origin of some cases of involuntary micturition.

There is however, usually no need to submit the child to this gamut of investigation, for in most cases a careful history followed by a full clinical

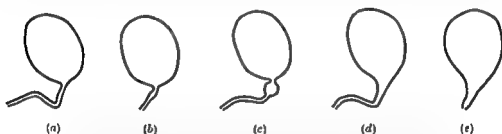


FIG. 179. Tracings of cystograms. (a) Normal outline in the male. (b) Normal outline in the female. (c) Urethral obstruction due to congenital valve causing dilatation of the posterior urethra. (d) "Neurogenic" bladder neck in the male. (e) In the female. In both (d) and (e) the bladder neck is widely dilated, giving a funnel-shaped appearance to the bladder outlet.

examination, including observation of the urinary stream and urinalysis, sort out the organic from the functional disorders. In other cases, a period of observation, the effect of training and/or drug therapy suffice to differentiate the two groups. The child is trained to hold urine for gradually increasing periods timed by the clock. Fluids before bedtime are restricted and the child is taken out of bed at night to urinate. Various mechanical alarm gadgets, which awaken the child when the urine leaks, can be usefully employed. Such regimes which require the co-operation of the parents and the child sometimes succeed. Quite often when the child is admitted to hospital for observation the bed wetting does not occur.

The variety of drugs used in enuresis reflect the lack of understanding of this disorder; moreover, a control series of "dummy" tablets often produce comparable beneficial effects. Preparations acting on the autonomic nervous system or the higher nervous centres may be tried. The following preparations are in common use: Tincture of belladonna, 0.5 ml. three times a day, increased slowly till the signs of intolerance develop; ephedrine, 15 to 30 mg. twice a day; propantheline (Probanthine), 15 to 30 mg. at night; or amphetamine sulphate, 5 to 15 mg. at night. The treatment of such associated conditions as constipation, threadworms, or vaginitis is necessary.

*The Operation.*—The technique of this operation in infants and adults differs only slightly. In children, after the area is cleaned with 1 per cent. cetrimide, the prepuce is drawn forward by two forceps applied to the margin of its opening (Fig. 178); only *slight* tension is required lest too much skin is removed. Another clamp is then placed obliquely across

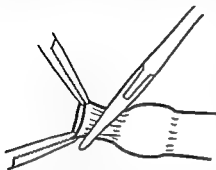


FIG. 178. The prepuce is drawn just taut by two forceps whilst another is placed obliquely just beyond the tip of the glans. N.B.—Too much tension will result in too much skin being removed.

the foreskin just distal to the glans (Fig. 178), so that by cutting *against* the distal surface of this clamp all the prepuce will be removed except for the sleeve of the inner layer, which lies exposed over the glans. This so-called "mucosal" layer is divided mesially as far as the coronal sulcus, and after freeing this layer of the skin off the glans both sides of the sleeve are trimmed off, leaving a margin of about 2 mm. or so of skin around the base of the glans. After bleeding points have been secured with fine catgut ligatures, the adjacent edges of the penile and "mucosal" skin are stitched together with interrupted

catgut sutures. A dressing of sterile vaseline gauze is then firmly applied. *Circumcision in adults* is described under *Genito-urinary Surgery*.

### ENURESIS

Children normally attain bladder control by their third birthday, achieving first diurnal and later nocturnal continence. But there is a wide variation of age at which children achieve full vesical control. Enuresis (lit. the passing of urine in) implies *involuntary* micturition, either during the day or night, and should not be confused with increased urgency or frequency of micturition. It is largely a functional disorder which is aggravated by emotional stress and anxiety. Threadworms and constipation make the condition worse. Inflammation and narrowing of the external and internal urinary meatus, preputial adhesions, vulvovaginitis and trigonitis have been held responsible in some cases. Sometimes a small capacity bladder which is sensitive to slight changes of pressure is found. In some cases control by the higher centres is apparently poorly developed, and is abolished by deep sleep. In others, neurological disorders, such as those occurring in spina bifida, or injury or disease of the spinal cord, cause incontinence.

*Investigations.* Exhaustive studies of the function and structure of the bladder have so far revealed no specific cause for this disorder, although in some cases radiography may show a bony deformity of the sacrum; *intravenous pyelography* may reveal pathological lesions of the upper or lower urinary tract; a *cysto-urethrogram* carried out during micturition may reveal bladder-neck or urethral obstruction, residual urine after

## SECTION III

### GENITO-URINARY SURGERY

by  
BASIL H. PAGE

#### CHAPTER 25

#### INVESTIGATION OF A GENITO-URINARY CASE

IN the diagnosis of urological disease special investigations are of decisive importance; nevertheless the clinical aspects are fundamental as in other branches of surgery. The interpretation of symptoms is of particular importance, for in so many cases the clinical signs are insignificant.

**Pain.** Pain of renal origin is distinguished mainly by two features: its site and its quality. Its most characteristic reference is to the loin, and the patient may indicate its position by turning slightly and placing the tip of the thumb precisely in the renal angle. This has been called "*thumb pain*." Another characteristic demonstration is for the patient to grip his side with the thumb behind and the fingers in front to show where the pain seems to be. Sometimes renal pain is felt only anteriorly, in the hypochondrium, and may then cause difficulty in diagnosis. Renal pain often radiates downwards and forwards towards the groin or further on to the testis or labium. This is also found in ureteric disease; the exact site of the cause cannot reliably be inferred from the location of the pain, for visceral sensations are too imprecise to be used in this way. Renal pain may radiate more widely. Thus it is quite common for it to spread well below the level of the iliac crest but it hardly ever ascends above the level of the costal margin. It usually remains strictly confined to one side of the body but occasionally it is felt right across the abdomen. The evidence sometimes suggests that disease of the kidney causes pain not on the side of the disease but on the other side, the so-called *reno-renal reflex*, but it is never possible to prove conclusively in these cases that the pain is not caused by the apparently normal kidney, and it is a good rule always to assume that the cause of renal pain lies on the same side as the pain no matter what the state of the other kidney may be.

The quality of visceral pain can never be adequately described in words, even by a very intelligent patient. Such terms as gripping,

**Organic Causes of Enuresis.** A favourable response to treatment as outlined above is often obtained in enuresis, and then suggests a functional basis for the disorder, although about 10 per cent. of children with enuresis have an organic lesion. It is important to diagnose causal lesions *early* so that active treatment can be carried out before irreversible complications develop. Children who have a structural defect in the urinary tract are usually wet day *and* night and never have a dry night. Constant dribbling is a feature of ectopic ureter. Urinary obstruction due to a urethral valve, bladder-neck obstruction or neurological disorder may cause overflow incontinence. Normally the bladder empties with a good even flow which ceases abruptly. In the presence of urethral or bladder-neck obstruction the flow is poor and dribbling occurs towards the end of micturition. Residual urine can be assessed clinically by bimanual abdominal and rectal palpation of the bladder region after micturition. In ectopic ureter the bladder itself empties normally but constant dribbling occurs from the abnormal external opening of the ureter. A complete investigation (*vide supra*) reveals such conditions as valvular obstruction of the posterior urethra, bladder-neck obstruction and neurogenic bladder.

tinence, both usually requiring mechanical correction; one type occurs mainly in women and the other in men. *Stress incontinence* is the voiding of urine, usually in small amounts, on straining, coughing or sneezing, and is commonly due to acquired defects in the supports of the bladder neck in women. In men a minor form of incontinence, known as "*after-dribbling*," is the loss of a little urine after the act of micturition seems to be complete. It is due to defective closure of the bladder neck or to the collection of urine in an enlarged posterior urethra in prostatic enlargement.

Hæmaturia is one of the commonest urinary symptoms, and, because it often signifies neoplastic disease, it is one of the most serious symptoms.

### HAEMATURIA

#### TIME RELATION OF BLOOD IN URINARY STREAM

##### INITIAL



SOURCE OF BLEEDING:- URETHRA

##### TERMINAL



SOURCE OF BLEEDING:- BLADDER AND PROSTATE  
(SLIGHT BLEEDING)

##### WHOLE STREAM



SOURCE OF BLEEDING:- BLADDER AND PROSTATE (PROFUSE  
BLEEDING) KIDNEY AND URETER

FIG. 180. *Hæmaturia*. Diagrams representing the likely sources of bleeding in the various types of hæmaturia.

If a patient thinks he has blood in his urine he is usually right, and he must always be given the benefit of the doubt. There are, however, certain conditions which may simulate hæmaturia. The brick-red sediment of urates in cooled urine and the high colour of very concentrated urine may both mislead patients. Rarely the urine may be turned red by the excretion of ingested substances and drugs. The only unequivocal test for blood is the finding of red cells on microscopy, but in most cases in which hæmaturia is stated to have occurred such confirmation is unnecessary.

Blood in the urine sometimes appears throughout the stream (total or whole-stream hæmaturia), sometimes only as the stream is finishing (terminal hæmaturia), and sometimes only at the onset of the stream (initial hæmaturia). This classification provides a rough clinical screening but it is not very reliable (Fig. 180). Initial hæmaturia is the least

cramping, colicky are suggestive and helpful but must not be accepted uncritically, for they may be misleading. The milder degrees of pain may be described as nagging, like toothache, or (more suggestive) "like wind." Severe renal pain, even when the patient cannot describe it in words, may often be recognised by three features: its severity, its duration, and the effect it has on the patient's behaviour. Although the degree of severity is very difficult to assess, the words agonising or unbearable usually leave little room for doubt; a woman may compare it with the pains of labour. The duration of an attack of renal pain is seldom less than thirty minutes and commonly lasts several hours. During this time the pain is usually continuous and often constant: it seldom comes and goes like intestinal colic and in this respect resembles gall-bladder pain. By enquiring what the patient did when the pain was at its worst, decisive information may be obtained. The patient is usually unable to keep still and may have paced the room or even rolled about on the bed. Vomiting and sweating may occur. These details of behaviour are worth more in diagnosis than an attempt by the patient to describe in words the quality of the pain.

One of the commonest simulators of renal pain is musculo-skeletal disease including rheumatism and fibrositis. The distribution of the pain in these conditions may strongly suggest a renal origin: on the other hand the aggravation of the pain by movement and straining and its dependence on certain postures are very unlike renal pain.

**Frequency of Micturition** is a common symptom of urinary disease. It is usual to record frequency by day and by night separately but there is no virtue in the traditional usage of setting this out as a fraction. The patient usually finds it easier to give the number of times urine is passed by night and the length of time urine can be held by day. The record might then for example read:—N = 3 times: D = hourly. Sometimes frequency is a manifestation of polyuria (the secretion of an abnormally large quantity of urine) particularly at night, and this important point can usually be settled by inquiry.

**Incontinence of Urine** may take several forms which can be distinguished clinically. **Enuresis** (also Chap. 24) is the emptying of the bladder without the ability of the patient to control it. As it is so much more common during sleep than waking, the term enuresis is often used synonymously for nocturnal enuresis. Except in imbeciles, young children and the aged, diurnal enuresis is likely to be due to organic disease of the nervous system (for example disseminated sclerosis). **Urgency incontinence** must be distinguished from enuresis. It is the uncontrollable voiding of urine due to the violent and often painful contraction of the bladder which may occur in cystitis. Long-continued obstruction of the bladder neck quite commonly results in incontinence due to passive overflow from a distended bladder, the so-called *retention (or distension) with overflow*. There are two other varieties of incon-

left. In examining the kidney, its normal descent on inspiration is exploited and it must be realised that as the kidney slides downwards, it remains tethered by its vascular pedicle so that it tends to swing medially, and as it does so it rides over the psoas with the lower pole directed forwards. This change of axis makes a low kidney feel large unless the examiner realises that he is feeling its length and not its thickness between his two hands. A renal tumour (the term is here used in the sense of any renal enlargement) usually has the general characteristics of a kidney in regard to position and movement. It is usually resonant on percussion due to the presence of the colon in front of it, unless the tumour is large enough to displace the colon to one side. In the male, genital and urinary systems are intimately related, and in the diagnosis of urinary disease much help may be got by examination of the vesicles, prostate and epididymes which are all readily accessible.

Accessory investigations play a decisive role in genito-urinary diagnosis, and the three most important are the examination of the urine, radiography and cystoscopy. The standard examination of the urine includes testing for albumen, microscopy of the centrifuged deposit, and culture. The important findings in the centrifuged deposit are red cells, an abnormal number of white cells, casts and crystals. Bacteria may also be found by direct examination of the deposit, but usually much fuller and more precise information about them can be obtained by culture. The urine for this routine examination is obtained in the female by catheter and in the male usually by collecting the urine in a sterile container after a simple cleansing of the meatus, rejecting the first part of the stream which may be contaminated by organisms washed out of the distal urethra.

**Radiography.** The plain film, control film or straight X-ray as it is variously called is an essential preliminary and a pyelogram can never be interpreted without it. This film must include the whole of the urinary tract from the top of the higher kidney to the bottom of the prostate and often also the whole urethra. Its most obvious use is the revealing of calculi or calcification, but a good film also shows the outline of the kidneys, psoas muscles and bladder and other soft-tissue shadows.

A *pyelogram* delineates the collecting system of the kidney and particularly the calices and pelvis. The radiopaque medium may be excreted by the kidney itself (intravenous pyelogram (I.V.P.) or excretion pyelogram) or injected into the pelvis through a ureteric catheter (retrograde, ascending or instrumental pyelogram).<sup>\*</sup> The excretion pyelogram is one of the most valuable of all the investigations of the urinary tract. It not only shows the shape and size of the pelvis and calices but also gives an indication of the function of each kidney. *Its great weakness is that the density of the shadow cast by the medium is usually less than that*

<sup>\*</sup> It is a common practice to speak of the contrast medium as dye, using the word in a figurative sense.



common and almost always arises in the urethra, usually the prostatic urethra. Terminal hæmaturia is the usual kind in cystitis and is very common. It almost always indicates a source in the bladder (including the vesical surface of the prostate) which is bleeding slowly (so that the blood trickles into the base of the bladder and is voided last) or which is made to bleed by contraction of the bladder. Whole-stream hæmaturia suggests a source of brisk bleeding in the bladder (frequently a tumour) or bleeding from the kidney. These observations will probably suggest a tolerably correct clinical diagnosis, but it is always essential to investigate a case of hæmaturia until a certain diagnosis is established, and this requires cystoscopy and pyelography in almost every case.

In the investigation of hæmaturia, time can be saved by knowledge of the relative incidence of the main causes (Fig. 181). In general, the two

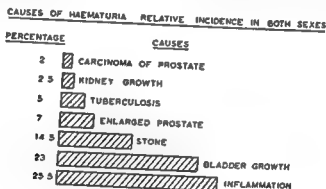


FIG. 181. *Hæmaturia*. Scheme showing the relative frequency of the main causes of hæmaturia in adults.

commonest causes are bladder tumours and inflammatory diseases of the urinary tract: between them they account for half the total cases. In women, inflammatory diseases are a specially frequent cause, but after the fifth decade bladder tumours are the commonest cause in both sexes. No other single cause comes near these two except in elderly men in whom the enlarged prostate is a common cause of hæmaturia. Calculi are a fairly common cause in the middle age groups. Important but relatively infrequent causes are tuberculosis and renal tumours. In children under ten years, hæmaturia is most often due to nephritis or urinary infection. In a certain proportion of cases (authorities vary in their estimate between 1 and 9 per cent.), no cause for the hæmaturia is found even after full investigation. To use the term essential hæmaturia for such cases is unhelpful and may even suggest that a diagnosis has been made. Patients with unexplained hæmaturia should be carefully followed up.

In urological cases, it is noteworthy that abnormal physical signs relating to the genito-urinary system are usually absent. In the majority of patients, with or without urinary disease, neither kidney can be felt; quite often the normal right kidney can be felt, and much more rarely the

left. In examining the kidney, its normal descent on inspiration is exploited and it must be realised that as the kidney slides downwards, it remains tethered by its vascular pedicle so that it tends to swing medially, and as it does so it rides over the psoas with the lower pole directed forwards. This change of axis makes a low kidney feel large unless the examiner realises that he is feeling its length and not its thickness between his two hands. A renal tumour (the term is here used in the sense of any renal enlargement) usually has the general characteristics of a kidney in regard to position and movement. It is usually resonant on percussion due to the presence of the colon in front of it, unless the tumour is large enough to displace the colon to one side. In the male, genital and urinary systems are intimately related, and in the diagnosis of urinary disease much help may be got by examination of the vesicles, prostate and epididymes which are all readily accessible.

Accessory investigations play a decisive role in genito-urinary diagnosis, and the three most important are the examination of the urine, radiography and cystoscopy. The standard examination of the urine includes testing for albumen, microscopy of the centrifuged deposit, and culture. The important findings in the centrifuged deposit are red cells, an abnormal number of white cells, casts and crystals. Bacteria may also be found by direct examination of the deposit, but usually much fuller and more precise information about them can be obtained by culture. The urine for this routine examination is obtained in the female by catheter and in the male usually by collecting the urine in a sterile container after a simple cleansing of the meatus, rejecting the first part of the stream which may be contaminated by organisms washed out of the distal urethra.

**Radiography.** The plain film, control film or straight X-ray as it is variously called is an essential preliminary and a pyelogram can never be interpreted without it. This film must include the whole of the urinary tract from the top of the higher kidney to the bottom of the prostate and often also the whole urethra. Its most obvious use is the revealing of calculi or calcification, but a good film also shows the outline of the kidneys, psoas muscles and bladder and other soft-tissue shadows.

A *pyelogram* delineates the collecting system of the kidney and particularly the calices and pelvis. The radiopaque medium may be excreted by the kidney itself (intravenous pyelogram (I.V.P.) or excretion pyelogram) or injected into the pelvis through a ureteric catheter (retrograde, ascending or instrumental pyelogram).<sup>\*</sup> The excretion pyelogram is one of the most valuable of all the investigations of the urinary tract. It not only shows the shape and size of the pelvis and calices but also gives an indication of the function of each kidney. Its great weakness is that the density of the shadow cast by the medium is usually less than that

<sup>\*</sup> It is a common practice to speak of the contrast medium as dye, using the word in a figurative sense.

common and almost always arises in the urethra, usually the prostatic urethra. Terminal hæmaturia is the usual kind in cystitis and is very common. It almost always indicates a source in the bladder (including the vesical surface of the prostate) which is bleeding slowly (so that the blood trickles into the base of the bladder and is voided last) or which is made to bleed by contraction of the bladder. Whole-stream hæmaturia suggests a source of brisk bleeding in the bladder (frequently a tumour) or bleeding from the kidney. These observations will probably suggest a tolerably correct clinical diagnosis, but it is always essential to investigate a case of hæmaturia until a certain diagnosis is established, and this requires cystoscopy and pyelography in almost every case.

In the investigation of hæmaturia, time can be saved by knowledge of the relative incidence of the main causes (Fig. 181). In general, the two

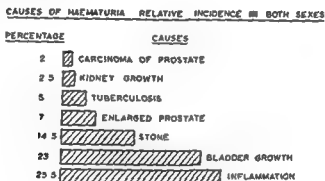


FIG. 181. *Hæmaturia.* Scheme showing the relative frequency of the main causes of hæmaturia in adults.

commonest causes are bladder tumours and inflammatory diseases of the urinary tract: between them they account for half the total cases. In women, inflammatory diseases are a specially frequent cause, but after the fifth decade bladder tumours are the commonest cause in both sexes. No other single cause comes near these two except in elderly men in whom the enlarged prostate is a common cause of hæmaturia. Calculi are a fairly common cause in the middle age groups. Important but relatively infrequent causes are tuberculosis and renal tumours. In children under ten years, hæmaturia is most often due to nephritis or urinary infection. In a certain proportion of cases (authorities vary in their estimate between 1 and 9 per cent.), no cause for the hæmaturia is found even after full investigation. To use the term essential hæmaturia for such cases is unhelpful and may even suggest that a diagnosis has been made. Patients with unexplained hæmaturia should be carefully followed up.

In urological cases, it is noteworthy that abnormal physical signs relating to the genito-urinary system are usually absent. In the majority of patients, with or without urinary disease, neither kidney can be felt; quite often the normal right kidney can be felt, and much more rarely the

interesting and sometimes useful information may be obtained by *perinephric air insufflation* and by *renal angiography* (aortography).

Cystoscopy allows not only direct inspection of almost the whole of the interior of the bladder but also indirectly it gives valuable information about the kidneys through a study of the ureteric orifices and their efflux, and also by ureteric catheterisation. The two essential components of a cystoscope are a light and an optical system, and they are assembled in a tubular sheath which is passed along the urethra into the bladder

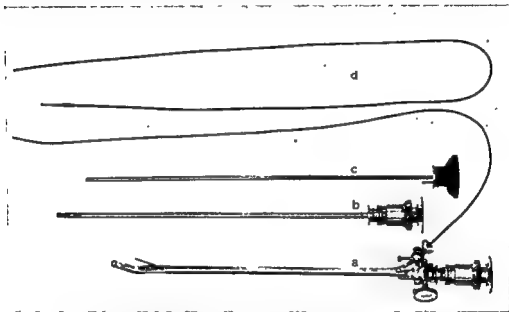


FIG. 183. *The Ringeb Type of Cystoscope. Ureteric Catheters.* (a) Sheath for ureteric catheterisation with one catheter in position for entering a ureteric orifice. (b) Viewing sheath. In these sheaths the lamp is not immediately adjacent to the fenestra in which the objective lens of the telescope lies. (c) Telescope. This make of cystoscope is boatable. Vision is not possible during the flow of irrigating fluid. (d) Chevasse type of ureteric catheter for ureterography.

(Fig. 183). Cystoscopy requires a medium of clear fluid in the bladder, and some cystoscopes are designed so that the bladder can be inspected while the irrigating fluid (usually water) is actually flowing in; this ensures visibility in spite of some bleeding and in other conditions which tend to cloud the medium. In the male, some form of anæsthesia (usually local) is desirable on account of the discomfort caused by straightening out the curves of the urethra and in passing the instrument through the sensitive and rather rigid prostatic urethra.

In the female an anæsthetic is usually not needed. Cystoscopy is particularly valuable in the investigation of hæmaturia, but there are both advantages and disadvantages in making the examination while bleeding is still in progress. The great advantage of doing the cystoscopy

of an instrumental pyelogram and details are therefore sometimes indistinct; also precisely because it is such an excellent index of renal function, it gives correspondingly poor pictures when the function of a kidney is failing. Although it is usually a very safe investigation, fatalities occasionally occur from sensitivity to the iodine in the compound, resulting in anaphylactic shock. It is important therefore to give a



FIG. 182. *Normal Instrumental (Retrograde) Pyelogram (right).*

small test dose (0.2 ml.) and wait before injecting the full amount (usually 20 ml.). The bowel should contain the minimum of gas and solid faeces, and, in order to ensure the best concentration of the medium, fluid should be withheld from the patient for twelve hours before the examination. The great advantage of retrograde or instrumental pyelography is the relative density of the shadow and the fact that this density is independent of the functional state of the kidney (Fig. 182). Its disadvantages are that it necessitates cystoscopy and creates artificial conditions by passively dilating the kidney. The practical position is that excretion pyelography is the first method to be used and in most cases it suffices. An instrumental pyelogram is done next if any points require clarification. Further

interesting and sometimes useful information may be obtained by *perinephric air insufflation* and by *renal angiography* (aortography).

Cystoscopy allows not only direct inspection of almost the whole of the interior of the bladder but also indirectly it gives valuable information about the kidneys through a study of the ureteric orifices and their efflux, and also by ureteric catheterisation. The two essential components of a cystoscope are a light and an optical system, and they are assembled in a tubular sheath which is passed along the urethra into the bladder

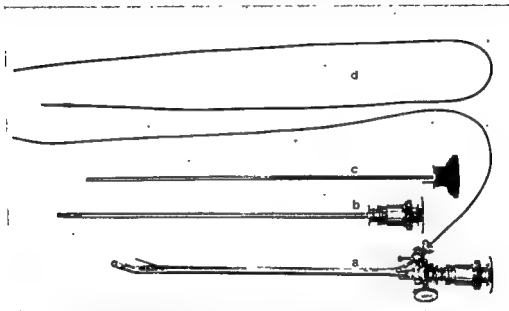


FIG. 183. *The Ringleb Type of Cystoscope. Ureteric Catheters.* (a) Sheath for ureteric catheterisation with one catheter in position for entering a ureteric orifice. (b) Viewing sheath. In these sheaths the lamp is not immediately adjacent to the fenestra in which the objective lens of the telescope lies. (c) Telescope. This make of cystoscope is boilable. Vision is not possible during the flow of irrigating fluid. (d) Chevaasu type of ureteric catheter for ureterography.

(Fig. 183). Cystoscopy requires a medium of clear fluid in the bladder, and some cystoscopes are designed so that the bladder can be inspected while the irrigating fluid (usually water) is actually flowing in; this ensures visibility in spite of some bleeding and in other conditions which tend to cloud the medium. In the male, some form of anaesthesia (usually local) is desirable on account of the discomfort caused by straightening out the curves of the urethra and in passing the instrument through the sensitive and rather rigid prostatic urethra.

In the female an anaesthetic is usually not needed. Cystoscopy is particularly valuable in the investigation of haematuria, but there are both advantages and disadvantages in making the examination while bleeding is still in progress. The great advantage of doing the cystoscopy

during an attack is that if the bleeding is of renal origin, the site of the lesion is clearly indicated by the issue of blood from the corresponding ureteric orifice. The disadvantage is that if bleeding is profuse, a clear view may not be obtained and the examination has to be repeated at some other time. Moreover, if an excretion pyelogram is to be done (as it is in most cases of hæmaturia), it is better done before cystoscopy, for it may indicate the need for more detailed information about one or other kidney and this would be supplied by an instrumental pyelogram, a by-product of the cystoscopy. However, in all cases of hæmaturia in which no cause has been found after full investigation, it is important for cystoscopy to be done as an emergency during the next attack of bleeding, should one occur.

**Assessment of Renal Function.** Although renal function is a complex process in which glomerular filtration, tubular reabsorption and tubular excretion all play different parts, in general it is true that surgical diseases which damage the kidney, and in particular those in which there is obstruction and suppuration, affect all components of the nephron so that in practice it is seldom necessary to apply specific tests for the separate processes. Clinical examination of the patient gives some indication of renal function although it is somewhat unreliable and difficult to interpret. Chronic renal impairment often shows itself by loss of weight (unless there is œdema which is unusual in "surgical" renal failure), anorexia, nausea, vomiting, lassitude, and sometimes thirst, polyuria and diarrhoea. The patient may be drowsy or even comatose, and the acidosis may cause hyperpnoea, a sign of advanced renal damage.

**Tests of Renal Function.** The blood urea estimation is by far the most widely used test, and if its limitations are recognised, it is a good indication of renal function. The normal range is 20 to 40 mg. per 100 ml. Basically it is a test of glomerular filtration. It is a simple and quick laboratory procedure and requires 2 ml. of oxalated blood. There are two main limitations of the blood urea estimation as a test of renal function. One is that the level may be raised in spite of normal renal function, by extra-renal causes such as increased tissue breakdown, dehydration or arterial hypotension. Common conditions in which some or all of these extra-renal factors operate to raise the blood urea are gastroduodenal hæmorrhage, intestinal obstruction and heart failure. The other limitation is that the blood urea does not begin to rise until there has been a considerable deterioration in renal function. A more accurate and earlier indication of impaired renal function based on the blood urea is the *urea clearance test* (Van Slyke) which measures the rate at which the kidneys clear the blood of urea. The result is expressed as a percentage of the normal and is an accurate measure of the glomerular filtration rate. The blood urea may be normal when the clearance rate is only 30 per cent. of normal. However, in spite of the relative inaccuracy of the simple blood urea estimation, it is used in surgery far more than the urea clearance test ;

there are probably two reasons for this. Firstly, the rather time-consuming and complicated urea clearance test is often unsuitable for acutely ill patients. Secondly, the issues of surgery are usually decided by the more extreme indications of functional failure. Any kidney with 30 per cent. renal function is worth saving: any patient with 30 per cent. renal function is a reasonable operative risk and it helps relatively little to know whether the function is for example 95 per cent. or 45 per cent.

The density of the shadow in an excretion pyelogram may be used as a rough indication of renal function. Some of the iodide compounds are excreted by the tubules and some almost exclusively by glomerular filtration, but in either case concentration can only be effected by the tubules. This radiological test is chiefly useful in comparing the function of one kidney with that of the other: as an absolute indication of renal function it is less reliable because concentration and therefore the density of the shadow depend so much on the patient's state of hydration and the radiographic technique.

The excretion of *phenol-sulphone-phthalein* is another standard test and measures chiefly tubular function. It is a simple but not an accurate test and is not very commonly used. A dye in much more common use is *indigo-carmin*, which is given intravenously in a dose of 5 ml. of a 0.4 per cent. solution. It is mainly used during cystoscopy to compare the function of the two kidneys. In a conscious patient who is not dehydrated, the dye should begin to appear in the ureteric efflux about three minutes after injection. With poor renal function the interval is longer, and the dye when it appears is more dilute. The test gives a quick and convenient qualitative estimation of renal function but it is vitiated by ureteric obstruction or general anaesthesia.

The estimation of the ability of the kidney to concentrate the urine is a sound physiological test of tubular function. A normal patient from whom water is withheld for twelve hours overnight excretes urine with a specific gravity of over 1025 and a urea concentration of over 2 per cent. In surgical practice, the total quantity of urine excreted in twenty-four hours is often used as a rough and ready guide to renal function. This is permissible if it is realised what very limited information is thereby obtained. A free flow of urine merely excludes some such condition as severe acute renal inflammation (in particular suppurative pyelonephritis) or some serious prerenal cause of oliguria such as arterial hypotension or heart failure. On the other hand, severely damaged kidneys (particularly those in which the tubules are mainly affected) are capable of excreting a large volume of dilute urine: in fact they are incapable of concentration. As this type of renal damage is not common in surgical diseases of the urinary system a free flow of urine is generally a favourable feature.



## CHAPTER 26

### DISEASES OF THE KIDNEY

Developmental anomalies of the kidney are common. They are important for two main reasons ; firstly because they may be a source of confusion or error in interpreting pyelograms, and secondly because they are quite commonly associated with obstruction, and may therefore be complicated by infection or stone formation. One of the commonest anomalies is a tendency for the kidney and particularly the calices and pelvis to be partly subdivided into two, a smaller upper and a larger lower part. Rather less commonly this duplication also involves the



FIG. 184. *Double Renal Pelvis and Ureter (Pylon Duplex) (right).* Instrumental (retrograde) uretero-pyelogram (by means of Chevasu catheter). The lower renal component and its ureter are greatly dilated. The two ureters on the right side join each other just outside the bladder.

ureter, which is double for a part or the whole of its course (Fig. 184). Malrotation is a rather less common anomaly, and causes the calices to lie directly behind the pelvis instead of posterolateral. The fusion of the lower poles of the two kidneys in front of the aorta produces a "horseshoe" kidney which gives suggestive signs in the pyelogram, but



FIG. 185. *Renal Fusion ("Horseshoe" Type).* Instrumental (retrograde) pyelogram. The lower calices are directed medially from the renal pelvis, especially on the left.

these signs are a striking understatement of the actual condition (Fig. 185). There are many other rarer anomalies such as ectopia, aplasia and the absence of one kidney. Anomalies of vascular supply are very common and are important as they may be associated with hydronephrosis and may cause surprises at operation. Developmental errors within the nephrons are thought to be the cause of polycystic disease and other types of renal cysts. (This subject is further discussed later in this chapter and also in Chap. 24.)

**Renal Trauma.** The kidney is a fairly tough organ and is well protected by ribs, spine and muscle so that it is not often injured. Penetrating injuries (such as gunshot wounds) are very rare in peacetime. Laceration of the kidney is usually due to crushing or a direct blow on the loin. The symptoms are hæmaturia and pain. The signs are tenderness, rigidity and often external bruising. Later, if hæmorrhage continues into the perinephric tissues, a mass may form. The diagnosis is usually straightforward, but the extent of the injury may be unsuspected unless an intravenous pyelogram is done, and even then it may be under-estimated. The injury varies from a slight split in the cortex to the complete severance of a major part of the kidney. Hæmorrhage is always the most serious result, but extravasation of urine is an added danger, particularly as it may lead to infection and possibly to consequent secondary hæmorrhage. The initial diagnosis and management may be complicated by injuries of other organs and structures. In the majority of cases of laceration of the kidney, the bleeding stops and the breach in the calix or pelvis heals without surgical intervention. If the primary hæmorrhage is severe or long-continued, operation is necessary and often necessitates nephrectomy. Partial nephrectomy or repair is sometimes feasible, but it must be remembered that these conservative operations may be followed by continued or recurrent (secondary) hæmorrhage which might impose an intolerable strain on an already weakened patient, and furthermore, urgent operation demanded by infection and secondary hæmorrhage may sometimes be difficult and dangerous.

### INFECTIONS OF THE KIDNEY

Infection may reach the kidney by various routes including the blood stream, the lumen of the ureter, the lymphatics or even by direct extension from an adjacent focus of infection. A very important and common group of infections of the kidney results from infection of the lower urinary tract; the route of invasion in these is probably the lumen of the ureter (lymph flow around the ureter is not in a continuous vertical stream, but successive segments of the ureter drain horizontally into the corresponding iliac or para-aortic lymph glands). The ascent of infection in the lumen of the ureter against the stream of urine is exactly analogous to the well-established process by which epididymitis arises from an infection in the prostate. Ascending renal infection obviously involves the pelvis first. If it goes on to involve the renal parenchyma it tends to follow the line of the vessels until it reaches the capsule. Hæmatogenous infections are less common but include such important examples as tuberculosis and renal carbuncle; the renal lesions incidental to septicæmia and pyæmia are in the same group but have less surgical importance.

Infection is often secondary to some underlying condition of the

kidney which weakens its resistance. The most important of these is obstruction to the urinary flow involving either the whole or only a part of the kidney ; other conditions predisposing to infection are stone (even when it is not causing obstruction) and neoplasm. The detection, and whenever possible the treatment, of these underlying causes form an important part of the management of urinary infections.

Renal infections are most commonly due to the *Bact. coli* (*Esch. coli*) which accounts for about two-thirds of all cases. Other Gram-negative organisms including *Proteus* and *Pseudomonas* account for another 10 to 15 per cent., a group which is important because of its resistance to chemotherapy. Staphylococci (aureus and albus) and streptococci ( $\alpha$  and  $\beta$  hæmolytic) are also found. The *Bact. coli* flourishes in acid urine. *Staphylococcus albus*, *Pseudomonas* and *Proteus* may all turn the urine alkaline by splitting urea and forming ammonium carbonate.

Pyelitis is the mildest form of acute renal infection and is usually due to invasion of organisms up the ureter. The inflammation is probably mainly confined to the pelvis, but little is known of the pathology of pyelitis, for it does not cause death nor is it an indication for operation ; thus direct study of the kidney is hardly ever possible. Pyelitis is far more common in females than males. It occurs at all ages but particularly in early childhood, following marriage, during pregnancy and at the menopause. The short urethra is probably the cause of the female predisposition to ascending infection. In pregnancy an additional factor operates, the mild obstruction of the ureters, particularly the right, brought about by the relaxing effect of progesterone on plain muscle, and perhaps aggravated by pressure of the enlarging uterus. Congenital anomalies resulting in minor degrees of obstruction at any level predispose to pyelitis.

Pyelitis usually begins fairly suddenly and may be preceded by the symptoms of cystitis. There are fever (fairly high, particularly in children) and sometimes rigors and vomiting. Sooner or later there is usually frequency of micturition, which is often painful ; sometimes there is hæmaturia. The patient does not appear more ill than the degree of fever would warrant. The urine is acid, faintly opalescent, and on microscopy is seen to contain pus cells and many organisms (usually coliform). In children the picture is often far less typical, and there may be no obvious urinary symptoms but simply fever, vomiting and abdominal pain. In differential diagnosis there are two main problems. The evidence may point clearly to disease of the urinary system, and then the question to be answered is whether there is pyelitis or some other urinary disease causing similar symptoms. For example, renal or ureteric calculi may simulate pyelitis but the pain they produce is usually disproportionate to the other symptoms. On the other hand, a calculus or some other disease of the urinary tract may be the underlying cause of what appears to be a simple attack of pyelitis, and in practice the differential diagnosis can

often only be made by further investigation of any case of pyelitis which is atypical, resistant to treatment or recurrent. The other type of problem in differential diagnosis arises when pyelitis is difficult to distinguish from non-urinary diseases, particularly those of the abdomen. Appendicitis is mentioned first because it is so common and delay in diagnosis may be very serious. In most cases, the symptoms and signs clearly distinguish between the two diseases, but atypical appendicitis is common and pyelitis may be slow to cause unequivocal urinary symptoms. A high retrocaecal appendix causes pain and tenderness in the flank or even in the loin, and pyelitis, especially in children, may cause diffuse abdominal pain. In adults, the height of the temperature is helpful evidence, for in appendicitis it is seldom over  $102^{\circ}\text{F}$ . There is no touchstone in this differential diagnosis but two general points may be made. If the evidence warrants a diagnosis of appendicitis operation must be done even though some features suggest pyelitis. The second guiding principle is that the finding of pus and bacteria on microscopy of the urine is very strong confirmation of the diagnosis of pyelitis.

The treatment of acute pyelitis is usually simple. Even twenty years ago the method of rendering the urine alkaline with potassium citrate was usually quickly effective, for the *Bact. coli* cannot tolerate an alkaline medium. Later the advent of the sulphonamides made the cure still more rapid and reduced the number of resistant cases. The standard treatment nowadays is the giving of a "sulpha" drug (for example, sulphadimidine 2 gm. followed by 1 gm. six-hourly for three or four days) combined with alkali (potassium citrate 30 gr. or 2 gm.) and a free fluid intake. The general measures for the treatment of a febrile illness are also often needed. If this treatment does not bring relief in two or three days, the appropriate antibiotic should be used as determined by sensitivity tests of the organism cultured from the urine. If this specific measure is not effective in two or three days, further investigations including cystoscopy and pyelography will be necessary to discover any underlying pathology.

**Pyelonephritis** is a more severe form of renal infection than pyelitis and is an important cause of fatal renal failure. The kidneys are enlarged and show on their surface small scattered yellow areas produced by subcapsular abscesses. There is some oedema of the perinephric fat and there may be perinephric suppuration. The opened kidney usually shows some inflammation of the pelvis, and this is severe in the common type due to ascending infection. The cut surface of the kidney shows radiating yellow streaks, the suppurating tracks running with the vessels towards the capsule. In places, these yellow streaks may have widened, coalesced and formed abscess cavities.

The onset of acute suppurative pyelonephritis is often accompanied by rigors and high fever. The patient looks ill; there are pain and tenderness in the loins; and the kidneys may be palpably enlarged. Renal function

is usually gravely affected, and in the early stages there is oliguria (i.e. the secretion of an abnormally small quantity of urine). The urine contains pus cells and red cells. The blood urea level is raised, and if the disease progresses, renal acidosis develops giving rise to the typical deep hissing respiration; death is usually preceded by uræmic coma. The diagnosis of pyelonephritis may be difficult when the patient is so gravely ill that a coherent history cannot be obtained and physical signs cannot be reliably elicited due to lack of co-operation. When pyelonephritis follows operations on the urinary tract (for example prostatectomy) the diagnosis may be complicated by other effects of the operation or the disease for which it was done.

In the treatment of pyelonephritis, any underlying predisposing factor, particularly obstruction, must be corrected. If there is retention of urine in the bladder a suprapubic cystostomy is often necessary. The causal organism must be attacked promptly and effectively. Because of the oliguria, sulpha drugs are best avoided, but while the urine is being cultured and sensitivity tests are carried out, treatment is started with an antibiotic considered likely to be effective (e.g. streptomycin or one of the broad spectrum agents). Fluid balance must be maintained and this may require parenteral administration.

**Chronic pyelonephritis** is an insidious type of renal infection which may not come to light until it has already greatly undermined renal function or perhaps caused hypertension. It does not cause suppuration but scattered chronic inflammatory foci particularly in the interstitial tissue. When the condition is widespread and of long standing, the kidney may be much scarred and contracted. Even in the active stages the clinical manifestations may be obscure, especially in children. Recurrent fever, gastro-intestinal disturbances, abdominal pain, undernourishment and anaemia are some of the general features, and the urinary symptoms may be overshadowed by them. The urine usually shows no macroscopic abnormality and even microscopically it may be normal at times. The main abnormalities are an excess of pus cells, red cells and a positive culture for organisms. The treatment is the ruthless eradication of infection from the urine and the systematic search for associated lesions of the urinary tract, especially congenital anomalies. Pyelography plays an extremely important part in the difficult problem of diagnosis. It is likely to show dilatation of the calices and irregularity in their outline of varying degrees (Fig. 186).

**Pyonephrosis** is a condition of generalised infection of the renal substance associated with obstruction of urinary outflow from the kidney. The common cause is calculous disease (Fig. 187). The kidney is enlarged; the pelvis and calices are dilated and contain purulent urine which may be very inspissated. The perinephric fat is oedematous, indurated and adherent to the renal capsule; the renal capsule itself on the other hand usually strips easily from the kidney. Suppuration may break out of

the kidney and form a perinephic abscess. The function of the kidney is usually lost without hope of recovery (Fig. 188).

In *acute* pyonephrosis the patient is feverish and toxic and suffers pain in the loin. *Chronic* pyonephrosis is sometimes symptomless. In either condition the kidney may be palpably enlarged and often feels hard and fixed, but there may be no detectable physical signs. The plain X-ray



FIG. 189. *Chronic Pyelonephritis*. Excretion Pyelogram (I.V.P.). Some of the calices (chiefly the upper) are dilated and over them the renal substance is very thin.

often shows stones, and excretion pyelography reveals no function on the affected side. Cystoscopy may show the very characteristic picture of thick purulent urine oozing from the ureteric orifice like toothpaste from a tube. The treatment is usually nephrectomy, but sometimes *nephrostomy* may be necessary as a preliminary measure so as to relieve the obstruction and allow some decline of the inflammation. Removal of a large and adherent pyonephrosis may be a difficult operation.

**Renal carbuncle** is the term given to an acute necrotising suppuration occurring in one part of the renal parenchyma. It is often due to the

*Staphylococcus aureus* and is then usually blood-borne, or it may be due to the coalescence of several areas of ascending suppurative pyelonephritis. The general and local symptoms are severe. The urine may show obvious signs of infection or in the hæmatogenous type it may be normal. Pycelography reveals a local lesion in the kidney. Treatment



FIG. 187. *Calculus Pyonephrosis* (right). Plain X-ray showing irregular triangular stone filling renal pelvis and smaller stones in calices. Same case as in Figs. 188 and 189.

consists first in the administration of the appropriate antibiotic (penicillin is usually effective) and when this has taken effect, the kidney is explored : nephrectomy is usually necessary.

**Perinephric Abscess.** Perinephritis occurs to some extent in most forms of renal suppuration but especially in pyonephrosis. Sometimes it is associated with an overgrowth of the perinephric fat, particularly with renal calculi. Perinephric abscess may be hæmatogenous or a complication of advanced inflammatory disease of the adjacent kidney (e.g. calculous pyonephrosis). In the hæmatogenous form, it may be that the



the kidney and form a perinephric abscess. The function of the kidney is usually lost without hope of recovery (Fig. 188).

In *acute pyonephrosis* the patient is feverish and toxic and suffers pain in the loin. *Chronic pyonephrosis* is sometimes symptomless. In either condition the kidney may be palpably enlarged and often feels hard and fixed, but there may be no detectable physical signs. The plain X-ray



FIG. 188. *Chronic Pyelonephritis. Excretion Pyelogram (I.V.P.).* Some of the calices (chiefly the upper) are dilated and over them the renal substance is very thin.

often shows stones, and excretion pyelography reveals no function on the affected side. Cystoscopy may show the very characteristic picture of thick purulent urine oozing from the ureteric orifice like toothpaste from a tube. The treatment is usually nephrectomy, but sometimes *nephrostomy* may be necessary as a preliminary measure so as to relieve the obstruction and allow some decline of the inflammation. Removal of a large and adherent pyonephrosis may be a difficult operation.

Renal carbuncle is the term given to an acute necrotising suppuration occurring in one part of the renal parenchyma. It is often due to the

*Staphylococcus aureus* and is then usually blood-borne, or it may be due to the coalescence of several areas of ascending suppurative pyelonephritis. The general and local symptoms are severe. The urine may show obvious signs of infection or in the haematogenous type it may be normal. Pyelography reveals a local lesion in the kidney. Treatment



FIG. 187. *Calculous Pyonephrosis (right)*. Plain X-ray showing irregular triangular stone filling renal pelvis and smaller stones in calyces. Same case as in Figs. 188 and 189.

consists first in the administration of the appropriate antibiotic (penicillin is usually effective) and when this has taken effect, the kidney is explored : nephrectomy is usually necessary.

**Perinephric Abscess.** Perinephritis occurs to some extent in most forms of renal suppuration but especially in pyonephrosis. Sometimes it is associated with an overgrowth of the perinephric fat, particularly with renal calculi. Perinephric abscess may be haematogenous or a complication of advanced inflammatory disease of the adjacent kidney (e.g. calculous pyonephrosis). In the haematogenous form, it may be that the

infection first lodges in the renal cortex, but if so it soon breaks out into the perinephric fat, and the renal lesion is inconspicuous, giving no signs in the urine or the pyclogram. Particularly in the hæmatogenous type, the onset is sudden with rigors, high fever and the constitutional evidence



FIG. 188. *Calculus Pyonephrosis.* Section through the right kidney of the case illustrated in Figs. 187 and 189. The renal pelvis (on the right) contains an irregular triangular stone and its walls are greatly thickened by fat and fibrosis. The calices (on the left) are dilated and contain pus and debris. Suppuration and fibrosis have destroyed the renal perenchyma. Perinephritis (in this case there was a perinephric abscess) had caused dense adherence of the perinephric fat.

of suppuration. There is deep pain, but quite commonly several days elapse before local physical signs develop; they consist of tenderness, rigidity and later a brawny swelling in the loin which may become clearly visible (Fig. 189). There is usually a lumbar scoliosis concave towards the affected side. Sometimes, particularly when there is primary disease of the kidney, the symptoms are very mild and out of all proportion to the physical signs. Treatment consists of incision and drainage.



FIG. 189. *Pyonephrosis Abscess* (right). Clinical photograph showing typical bulge in lumb. Same case as in Figs. 187 and 188.

Antibiotics play a helpful but secondary role. The kidney must be fully investigated, for it may later require definitive treatment (e.g. nephrectomy for pyonephrosis).

**Tuberculosis of the Kidney.** (Chap. 32.)

**Hydronephrosis** is the dilatation of the drainage system of the kidney. It usually involves both the pelvis and the calices, but sometimes the one and sometimes the other is predominantly affected (Figs. 190 and 191). The dilatation may occasionally be confined merely to one calix, a condition called *hydrocalicosis*. Hydronephrosis is usually due to obstruction which may lie anywhere between the preputial orifice and the uretero-pelvic junction. Any cause at the bladder neck or below it will of course produce bilateral hydronephrosis. The obstruction may be in the lumen of the urinary tract (e.g. a stone or a pedunculated tumour), outside it (e.g. pressure by an adjacent malignant tumour, kinking by an aberrant

renal artery) or in the wall (e.g. stenosis, achalasia, stricture, tumour). One of the commonest causes of hydronephrosis is obstruction at the pelviureteric junction, and this type is sometimes designated primary or idiopathic, for in most cases the cause cannot be fully explained. In this type, there is quite often an aberrant vascular leash running to the lower pole of the kidney and crossing in front of the pelviureteric junction. Sometimes the upper end of the ureter can be seen to loop over this leash of vessels with the bulging pelvis hanging forwards and downwards. In



FIG. 190. *Hydronephrosis. Pelvic Type.* Excretion pyelogram (I.V.P.). Good renal function. Pyeloplasty was done. The left kidney had previously been removed for advanced hydronephrosis.

such cases, there can be no doubt that the vessel itself is the main or even the only cause of the hydronephrosis, although similar aberrant vessels are often found without hydronephrosis; when there is associated hydronephrosis there may also be narrowing of the lumen (stenosis or stricture) at the pelvi-ureteric junction or a fixed kink due to periureteric adhesions. Disagreement about the relative importance of these factors in this type of hydronephrosis has led to the devising of a variety of operations.

The type of obstruction which causes hydronephrosis is usually incomplete and has to act for a long time, often for months or years. Sudden complete obstruction, as by ligature of the ureter, causes atrophy of the

kidney with relatively little dilatation of the pelvis and calices. Experimental work on animals, however, shows that this last rule does not always hold (Hinman). The effect of obstruction on the renal parenchyma is vitally important. The back pressure which dilates the pelvis and calices also dilates the renal tubules and causes atrophy of the cells lining them. Macroscopically this is reflected in the progressive thinning of



FIG. 191. *Hydronephrosis, Colicost Type. Instrumental (retrograde) pyelogram (left). (Courtesy of Dr. J. M. Stewart.)*

the renal parenchyma (Fig. 192). The glomeruli, which are more resistant than the tubules and are farther away from the obstruction, are affected later. This order of deterioration may lead to the production by the hydronephrotic kidney of a large volume of dilute urine due to failure of tubular reabsorption and secretion while glomerular filtration continues.

Hydronephrosis is not always due to obvious mechanical obstruction. Atony or incoordination of peristalsis may also be responsible. Atonic hydronephrosis commonly occurs during pregnancy; it affects the right kidney more than the left. The enlarging uterus may cause some mechanical obstruction in addition but it is relatively unimportant. This mild hydronephrosis is reversible and usually harmless but it weakens

renal artery) or in the wall (e.g. stenosis, achalasia, stricture, tumour). One of the commonest causes of hydronephrosis is obstruction at the pelviureteric junction, and this type is sometimes designated primary or idiopathic, for in most cases the cause cannot be fully explained. In this type, there is quite often an aberrant vascular leash running to the lower pole of the kidney and crossing in front of the pelviureteric junction. Sometimes the upper end of the ureter can be seen to loop over this leash of vessels with the bulging pelvis hanging forwards and downwards. In



FIG. 190. *Hydronephrosis. Pelvic Type.* Excretion pyelogram (I.V.P.). Good renal function. Pyeloplasty was done. The left kidney had previously been removed for advanced hydronephrosis.

such cases, there can be no doubt that the vessel itself is the main or even the only cause of the hydronephrosis, although similar aberrant vessels are often found without hydronephrosis; when there is associated hydronephrosis there may also be narrowing of the lumen (stenosis or stricture) at the pelvi-ureteric junction or a fixed kink due to periureteric adhesions. Disagreement about the relative importance of these factors in this type of hydronephrosis has led to the devising of a variety of operations.

The type of obstruction which causes hydronephrosis is usually incomplete and has to act for a long time, often for months or years. Sudden complete obstruction, as by ligature of the ureter, causes atrophy of the

calices, displacing the urine, and only later diffuses widely. On the other hand, in instrumental pyelography the urine can often be aspirated through the ureteric catheter before the medium is injected. Once the fact of hydronephrosis has been established, it is necessary to determine the site and nature of the cause and the state of function of the affected and of the opposite kidney.

In the treatment of hydronephrosis two primary considerations arise. The first is to decide whether the state of function of the kidney justifies conservative measures or whether nephrectomy should be done. This decision is made mostly on the result of the excretion pyelogram, but also partly on the appearance and the feel of the kidney at operation. In some cases of slight hydronephrosis with good renal function and no complications, it may be advisable to give no treatment but to keep the patient under periodic observation. The second main consideration is to



FIG. 193 *Hydronephrosis*. Diagram illustrating the principle of the Foley "Y-V" pyeloplasty.

remove the cause. In cases with an obvious local cause such as a ureteric calculus, the problem is relatively simple, but in the common type of "idiopathic" hydronephrosis with obstruction at the pelviureteric junction, the problem is less simple because the nature of the obstruction is not fully understood and treatment is largely empirical. Whenever any probable contributory factor is found such as an aberrant vessel or a kinking adhesion, it must of course be dealt with, but this alone is seldom sufficient, and some form of plastic operation designed to enlarge the lumen at the pelviureteric junction must also be done. The "Y-V" procedure of Foley (Fig. 193) allows this to be done without interrupting the continuity of the urinary tract, but good results are obtained by resection of the pelviureteric junction together with as much of the dilated pelvis as is considered necessary and reunion of the pelvis with the ureter by a wide anastomosis (Fig. 194). This method, described by Anderson and Hynes in 1949, has three main advantages over the simple widening procedure: (1) It reduces the size of the pelvis; (2) it allows the ureter to be transposed in relation to an aberrant vessel, thus eliminating this obstructive factor; (3) it actually removes the pelviureteric junction in which the pathological process causing the main obstruction



the kidneys' resistance to infection. In other types of non-obstructive hydronephrosis, neuromuscular disorder is sometimes postulated and, because of the analogy with Hirschsprung's disease, a search has been made for abnormalities of the autonomic ganglia in the muscle wall of the ureter but with no success. In fact, we understand little about the

working of the motor innervation of the urinary tract but enough to know that there is no simple reciprocation between sympathetic and parasympathetic nerve supply. These obscure cases provide a difficult problem in treatment. Inflammatory conditions of the kidney and pelvis (e.g. tuberculosis) are often associated with some degree of hydronephrosis, probably of the atonic variety.

The symptoms of hydronephrosis are not specific. One of the commonest manifestations is renal pain, which may be a dull ache or violent colic. Sometimes there are dyspeptic symptoms. Hydronephrosis is often brought to light by some complication, particularly urinary infection, and very occasionally it is found in the search for a cause of hypertension.

The physical signs are usually negative even when the radiological evidence shows well-developed hydronephrosis, but sometimes a renal swelling may be felt and occasionally it is very



FIG. 192. *Hydronephrosis.* Photograph of section of left kidney showing dilatation of calices and thinning of renal substance. Obstruction at the pelviureteric junction had been relieved by operation (Anderson-Hynes pyeloplasty) four years previously, but the renal changes had been too advanced; persistent urinary infection and pain necessitated nephrectomy.

large. In some cases the swelling varies in size from time to time.

The diagnosis is made by pyelography. In the interpretation of the excretion pyelogram, it must be remembered that the pelvis and calices are distended with urine through which the opaque medium must diffuse before it can produce a complete outline of the hydronephrosis. The result is that the calices are shown first, and shown best because they receive the medium in the strongest concentration the kidney can produce, whereas the pelvis must wait until the medium has become diffused and diluted. Even the instrumental pyelogram favours the calices at the expense of the pelvis because the relatively heavy medium flows back into the

occur in alkaline urine, whereas oxalate stones are found in acid urine. The colloid content of the urine is important for crystalloids are held in suspension by colloids. Another component of the urine preventing precipitation is citric acid, the concentration of which is increased in one phase of the menstrual cycle; this may explain why women are less liable to stone than men. A stone may form round a nucleus such as a foreign body, inflammatory debris, or a collection of bacteria. Once a stone has begun to form it is itself a nucleus on which fresh deposition occurs so that stones always tend to grow. Lack of vitamin A in the diet has been proved by animal experiments to be a cause of stone formation, and it is probable that deficiency of this vitamin was the chief factor concerned in the common occurrence of urinary stones in England up to 100 years ago, and it may still be a factor in countries where renal calculi are endemic. There is also evidence that the mineral content of the drinking water may also help to cause stones to form.

Any satisfactory theory of stone formation must not only account for the precipitation from the urine of solid particles, but it must also explain why these particles are not washed away in the urinary stream when they are still small. Obstruction in the renal tract might explain this, but in very many cases there is no obstruction. One obvious explanation of this failure of the nascent stones to be washed away is that they may be moored to the mucosa of the pelvis or calices, or even develop outside the lumen of the calices and only come in contact with the current of urine by destruction of the tissue overlying them. There is good evidence to support these hypotheses which link up with the concept of local pathology within the kidney as a factor in stone formation. There is reason to believe that low grade infection not only in the kidney but in other parts of the urinary tract (e.g. prostate, urethra) may play an important part in the aetiology. The great majority of renal calculi are composed of calcium oxalate or mixed phosphates or both. Phosphatic calculi are usually found in association with infection, particularly in alkaline urine. Rare stones are uric acid (not opaque to X-rays), urates, calcium carbonate, cystine (Fig. 195) and xanthine.

*Nephrocalcinosis* is a term used to describe diffuse calcification in the renal parenchyma, usually bilateral, with or without calculi in the pelvis or calices. It may be found in hyperparathyroidism (Fig. 196), hypervitaminosis D, renal acidosis, or without any discoverable metabolic abnormality.



FIG. 195. *Renal Calculi*. Photograph of renal calculi (cystine) removed by pyelo- and nephro-lithotomy.

may be assumed to lie. The aberrant vessel must be divided only if it is very small, otherwise a considerable part of the kidney will be deprived of its blood supply. Hamilton Stewart has devised an ingenious operation in which the kidney is rolled up on itself so that the upper and lower poles are held together and the aberrant vessel is thus lifted away from the pelviureteric junction.

Hydronephrosis in which there is no obvious local obstruction provides the most difficult therapeutic problem of all. Often the best treatment

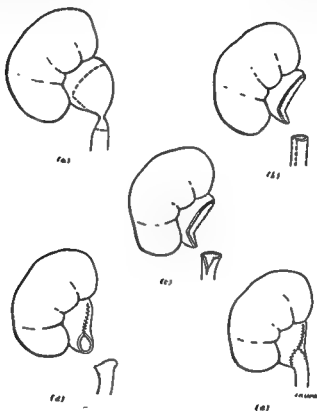


FIG. 194. *Hydronephrosis. The Anderson-Hynes pyeloplasty.*

is no treatment, but renal denervation has been advocated and success has also been reported following the use of pituitrin injections.

**Renal Calculi.** Renal calculi are common. A good deal has been discovered about many of the factors in the aetiology, but in spite of this the cause of most renal calculi is unknown. In theory, one of the most obvious factors should be the excessive concentration in the urine of the constituents of the various calculi. This factor does operate in some conditions, e.g. *hyperparathyroidism* (hypercalcaemia) and *cystinuria*. It is probably also a factor in recumbency calculi and also in dehydration. The state of the solvent from which the crystals precipitate is another obvious factor in theory, and it is known that the reaction is important, for certain types of stone such as those composed of "triple" phosphate

quite common for a stone to be discovered during the investigation of urinary infection.

The diagnosis of renal calculus is made by radiography (Figs. 107 and 108). Non-opaque stones are very rare, but difficulty is often caused by stones of rather poor density, particularly when they are small or obscured by an excess of gas or faeces in the colon. A shadow in the renal area is of course not necessarily a renal stone. It may be a gall stone, a calcified mesenteric gland or a concretion in the gut. Calcification in the costal cartilages or in the adrenals may also cause difficulty. A lateral X-ray or the comparison of the X-rays taken in inspiration and expiration may



FIG. 107. *Renal Calculus.* Plain X ray showing a calculus in the pelvis and lower calix of right kidney. At operation, the lower pole of the kidney and the stone were removed.

immediately clear up some of these doubts by showing whether or not the shadow in question is in the plane of the kidneys and moves with them. Any remaining doubts are usually dispelled by excretion pyelography which should always be done, not only to prove the presence of a calculus (by showing its constant relation to pelvis or calix) but even more important to assess renal function to demonstrate the existence and degree of any hydronephrosis, and to show up any underlying structural abnormality. In some cases instrumental (retrograde) pyelograms are necessary, particularly when renal function is poor. In all cases the urine is examined under the microscope (the deposit may contain important evidence, e.g. crystals of oxalate or cystine) and cultured. When both kidneys are affected, tests of total renal function are done. The routine investigations may have indicated some local aetiological factor such as infection or obstruction, or may have suggested the possibility of hyper-

The effects of renal calculi may be serious. The two commonest and most important ones are infection and obstruction. Either of these singly or still more so in combination may destroy the kidney by causing hydro-nephrosis, pyelitis, pyelonephritis or pyonephrosis. The infection may spread outside the kidney; thus there is quite commonly perinephritis



FIG. 196. *Nephrocalcinosis*. Widespread calcification in both kidneys due to hyperparathyroidism (adenoma). The oval dense shadows in the lower part of the film are enteric-coated capsules of sodium chloride in the gut.

and occasionally a perinephric abscess. The stones may pass on into the ureter and cause complications there. A rare complication in the kidney is squamous carcinoma of the renal pelvis. Although stones may cause all these serious effects, they may also remain tucked away in a calix and cause only the most trivial and local trouble over the course of years.

The characteristic *symptom* of uncomplicated renal calculus is pain which may be either colicky or dull and localised. Haematuria is quite a common symptom and the passage of a stone a very obvious one. The infective complications produce their characteristic symptoms, and it is

quite common for a stone to be discovered during the investigation of urinary infection.

The *diagnosis* of renal calculus is made by radiography (Figs. 107 and 108). Non-opaque stones are very rare, but difficulty is often caused by stones of rather poor density, particularly when they are small or obscured by an excess of gas or faeces in the colon. A shadow in the renal area is of course not necessarily a renal stone. It may be a gall stone, a calcified mesenteric gland or a concretion in the gut. Calcification in the costal cartilages or in the adrenals may also cause difficulty. A lateral X-ray or the comparison of the X-rays taken in inspiration and expiration may



FIG. 107. *Renal Calculus.* Plain X-ray showing a calculus in the pelvis and lower calix of right kidney. At operation, the lower pole of the kidney and the stone were removed.

immediately clear up some of these doubts by showing whether or not the shadow in question is in the plane of the kidneys and moves with them. Any remaining doubts are usually dispelled by excretion pyelography which should always be done, not only to prove the presence of a calculus (by showing its constant relation to pelvis or calix) but even more important to assess renal function to demonstrate the existence and degree of any hydronephrosis, and to show up any underlying structural abnormality. In some cases instrumental (retrograde) pyelograms are necessary, particularly when renal function is poor. In all cases the urine is examined under the microscope (the deposit may contain important evidence, e.g. crystals of oxalate or cystine) and cultured. When both kidneys are affected, tests of total renal function are done. The routine investigations may have indicated some local aetiological factor such as infection or obstruction, or may have suggested the possibility of hyper-

parathyroidism which, although accounting for only about 5 per cent. of urinary calculi, is one of the few curable metabolic causes.

The treatment of a renal calculus is to remove it and also, whenever possible, to remove its cause. If the stone is judged to be small enough to pass spontaneously, this is encouraged by an increased fluid intake and postural exercises designed to ensure that gravity acts to draw the stone towards the renal pelvic outlet at least once a day. Stones in calices



FIG. 198. *Renal Calculi*. Plain X-ray. The left renal pelvis is filled with a large stone and numerous smaller stones occupy the calices. These are metabolic stones due to cystinuria. Renal function was good and they were removed by pyelo- and nephro-lithotomy. See Fig. 195.

causing neither symptoms nor infection may be kept under periodic radiographic review until some indication to intervene arises. In some cases of large bilateral "staghorn" calculi which ramify throughout the calices without causing obstruction, operation may be more harmful to the kidney than the continued presence of the stone, and the recurrence rate after operation in such cases is high.

The operations which may be done for renal calculi are *pyelolithotomy*, *nephrolithotomy*, *partial nephrectomy* and *nephrectomy*. The operation of choice is pyelolithotomy, as it causes the least damage. Nephrolithotomy involves an incision through the renal substance, but may have to be used for calculi in the calices when they cannot be extracted through an incision in the pelvis of the kidney or for very large calculi, especially

when the pelvis is enclosed within the renal substance. It is a more severe operation than pyelolithotomy, and the incision and suture of the renal substance results in permanent damage to the kidney; also serious post-operative complications, particularly secondary haemorrhage, are more common and may even necessitate emergency nephrectomy. Partial nephrectomy is a very useful operation for stones in the calices, particularly when they are at one pole (they are commonly found in the lowest group of calices), because it removes not only the stone, but also the damaged and diseased part of the kidney which made the stone, thus reducing the risk of recurrence. Although the possible complications of this operation are greater than those of pyelolithotomy, they can be greatly reduced by meticulous technique and a judicious use of antibiotics. Nephrectomy is reserved for cases in which the complications of stone have destroyed the function of the kidney beyond hope of useful recovery.

Renal stones may recur after removal (30 per cent. recurrence has been recorded by urologists of great experience). The avoidance of recurrence demands a knowledge of the factors concerned. The first requirement is the removal of every fragment of stone however small, for any remaining fragment acts as a nucleus for the formation of a fresh stone. X-ray control during operation provides a valuable check. The second requirement is the elimination of infection after operation, especially infection with urea-splitting organisms. In the third place, any underlying cause, either local or general, must be treated whenever possible.

### RENAL TUMOURS

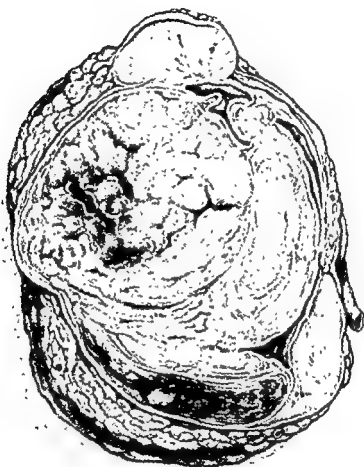
Renal tumours are important because they are almost always malignant. They are however not very common particularly when compared with bladder tumours. Small benign adenomata are quite a common incidental finding *post-mortem*, but an adenoma large enough to cause symptoms and constitute a surgical problem is extremely rare. Although transitional-cell papilloma of the renal pelvis is histologically benign, its frequent association with tumours of the ureter or bladder necessitates radical treatment so that in practice it is hardly benign.

Most renal tumours fall into two classes: (a) *Adenocarcinoma* of the renal parenchyma and (b) *transitional-cell tumour* of the mucosa lining the pelvis or calices. In addition, there are two types of tumour which are composed of cells not normally found in the kidney. These are the *nephroblastoma* or *Wilms' tumour* which occurs in childhood (see also Chap. 24) and *squamous carcinoma* of the renal pelvis which is usually associated with and is perhaps caused by calculi and chronic infection.

Malignant growths of the parenchyma are the commonest type of renal tumour. They frequently contain cells filled with lipid and in general appearance resemble the cells of the adrenal cortex. For this reason they have been thought to be derived from adrenal rests in the kidney and were therefore given the name *hypernephroma*. Although this theory



is largely discredited, the term remains and is applied sometimes to all parenchymatous tumours, or sometimes only to those showing the peculiar "adrenal" features in a marked degree when the remainder are called adenocarcinomas. The term Grawitz tumour is roughly synonymous with



ENGLAND.

FIG. 199. *Carcinoma of Renal Parenchyma.* Section through nephrectomy specimen. The tumour replaces the mid-portion of the kidney and bulges into the pelvis which is stretched over it. It has a deceptive appearance of encapsulation. The cut surface shows the three characteristic types of tissue: (1) Haemorrhagic (bottom). (2) Yellow due to lipoid (right). (3) Necrotic with tendency to liquefaction and pseudocyst formation (top left). Note that the kidney has been removed in its covering of fat. Same case as Fig. 201.

hypernephroma. Macroscopically, parenchymatous tumours are often rounded, apparently encapsulated masses usually at one or other pole of the kidney, distorting and compressing the renal substance (Fig. 199). The cut surface shows yellow areas (lipoid), red areas (haemorrhage, for these tumours are very vascular), and sometimes cystic areas (due to

colliquative necrosis). Their natural history is strange and unpredictable. They behave like malignant, often very malignant tumours, but with a difference. A renal carcinoma may remain apparently unchanged for years; but on the other hand it is common for extensive recurrence or metastasis to develop within a year of operation. Tumour cells frequently

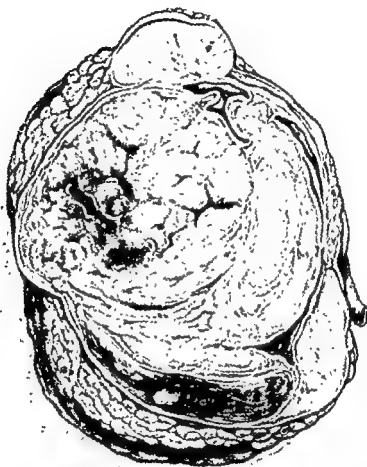


FIG. 200. *Renal Carcinoma.* X-ray of chest showing "cannon ball" secondary deposit in left lung.

spread by the blood stream, and secondary deposits are found in the lungs and bones (Fig. 200). The growth also spreads directly into the perinephric tissues and to the para-aortic lymph glands; the liver also may be involved.

**Symptoms and Signs.** The commonest single symptom is hæmaturia, but it occurs in only about half the cases. The bleeding may be profuse, and clots in the ureter may cause colic, and in the bladder retention of urine. Other symptoms due to the primary growth are renal pain and swelling, but only too often the first symptoms are due to widespread

is largely discredited, the term remains and is applied sometimes to all parenchymatous tumours, or sometimes only to those showing the peculiar "adrenal" features in a marked degree when the remainder are called adenocarcinomas. The term Grawitz tumour is roughly synonymous with



C. PLAMÉ

FIG. 199. *Carcinoma of Renal Parenchyma.* Section through nephrectomy specimen. The tumour replaces the mid-portion of the kidney and bulges into the pelvis which is stretched over it. It has a deceptive appearance of encapsulation. The cut surface shows the three characteristic types of tissue: (1) Hemorrhagic (bottom). (2) Yellow due to lipoid (right). (3) Necrotic with tendency to liquefaction and pseudocyst formation (top left). Note that the kidney has been removed in its covering of fat. Same case as Fig. 201.

hypernephroma. Macroscopically, parenchymatous tumours are often rounded, apparently encapsulated masses usually at one or other pole of the kidney, distorting and compressing the renal substance (Fig. 199). The cut surface shows yellow areas (lipoid), red areas (hemorrhage, for these tumours are very vascular), and sometimes cystic areas (due to

polycystic disease is almost always bilateral and a family history may be obtainable. A solitary cyst may be even more difficult to distinguish, especially if it is situated well within the kidney. Renal angiography should be the ideal means of settling this point by showing up the increased vascularity of the tumour or even the pooling of the contrast medium in extravasated blood, in contrast with the bloodless space of a cyst. However, the evidence of the angiogram often falls short of finality and nothing less than finality suffices when there is the possibility of a renal tumour. Perinephric air insufflation (followed by radiography), and tomography are other investigations which may help in difficult cases.

**Treatment.** The treatment of renal carcinoma is nephrectomy. If possible, the kidney should be removed with its covering of fat intact, and the vascular pedicle should be secured as early in the operation as is feasible in order to prevent the dislodgement of tumour emboli into the general circulation. The tumours are always vascular and may be large, therefore a free exposure is needed. Radiotherapy alone has not yet proved an adequate alternative but it may be used in addition to surgery either before or after operation; it is often used alone as a palliative in inoperable cases. In this rather strange disease the presence of a solitary metastatic deposit is not necessarily a contraindication to nephrectomy if the patient's general condition is satisfactory, for cases have been recorded of the successful resection of pulmonary deposits; on the other hand pulmonary metastasis may remain symptomless for a long time.

---

**Tumours of the Renal Pelvis.** These tumours are usually composed of transitional cells, and histologically may appear either benign or malignant, but even the apparently benign ones may be associated with other similar tumours in the ureter, bladder or even urethra (Fig. 202). Whether the lower placed tumours are due to implantations of "seedlings" from the higher tumours, whether they are independent tumours arising from multicentric foci of neoplastic activity, or whether all the tumours are caused by a carcinogenic agent in the urine, are still disputed questions, but they have little effect on the practical management, for in any case it would be necessary to remove the whole ureter with the kidney and afterwards to inspect the bladder and urethra periodically. Malignant tumours of the renal pelvis spread to the regional lymph glands and also to the lungs and bones.

Hæmaturia is by far the commonest symptom. Pyelography usually shows a filling defect in pelvis or calix (Fig. 203). Abnormal physical signs are uncommon. The treatment is nephroureterectomy; the whole tract is removed in one piece with care to avoid any spilling of the urine, for this may lead to implantation of tumour cells in the wound. (*Wilms' Tumour*.—See under Pædiatric Surgery, Chap. 24.) The prognosis of

secondaries, e.g. a pathological fracture. Sometimes there is prolonged irregular fever. The physical signs of the primary tumour may be a palpable enlargement of the kidney, but it is quite common for the tumour to be impalpable.

The diagnosis depends mainly on pyelography which shows distortion, a filling defect or lack of renal function (Fig. 201). Usually both excretion



FIG. 201. *Renal Carcinoma.* Instrumental (retrograde) pyelogram of right kidney showing obliteration of middle calices and distortion of upper and lower groups of calices by a tumour occupying the mid portion of the kidney. Same case as Fig. 199.

and instrumental pyelograms are needed to confirm the diagnosis. It is often striking how little abnormality of the pyelogram may be caused by an extensive tumour. Conditions which may simulate a tumour, particularly in the pyelogram, are cysts and the results of infection. Chronic pyelonephritis may produce great distortion of the calices, and a chronic abscess may cause a filling defect. The other features in these cases may decide the issue quite clearly but sometimes the difficulty is great. The pyelogram in polycystic disease is often very like that of tumour, but

kidneys, and it is quite common for polycystic disease to give a pyelogram which it is difficult to distinguish from the extremes of normal.

Surgery has little to offer in the treatment of polycystic disease. If the other kidney is functioning fairly well, nephrectomy may rarely be justifiable for severe complications such as persisting hematuria which is threatening life. There has been a tendency to revive Rovsing's operation



FIG. 203. *Papilloma of Renal Pelvis.* Instrumental (retrograde) pyelogram showing filling defect of pelvis and middle calix. Same case as Fig. 202.

in which the kidney is exposed and as many cysts as possible punctured. By this operation it is hoped to relieve the pressure on the functioning nephrons at least for a time, but its effectiveness cannot be proved.

**Solitary Cyst.** In this condition the kidney as a whole is healthy but contains usually one cyst or occasionally several. They are commonly situated at one pole of the kidney but may be embedded in its substance. Their size varies greatly but is often comparable with that of the kidney and seldom less than a quarter its size (Fig. 204). They are lined with flattened or cuboidal epithelium. Their pathogenesis is not known, but

renal tumours is poor. Deming's figure is 10 per cent. ten-year survival for all types.

**Polycystic Disease.** This is a developmental abnormality, almost always affecting both kidneys, in which the renal substance is to a varying extent replaced by multiple cysts. It is thought to arise from the imperfect union between the excretory and the collecting elements of the

nephron. The disease is familial (Mendelian recessive), but patients are often unaware that any of their relations have been affected. Two main types are recognised. In the commoner types the disease does not manifest itself until the patient is adult or even middle-aged, whereas in the other type the condition is evident at birth. In the infantile variety, the baby is still-born or dies very shortly after birth. In adults, the disease is very slowly progressive.

The cysts slowly crush the life out of the normal nephrons by obstructing the drainage channels and causing pressure atrophy of the tubular cells. Renal function gradually fails and uræmia leads on to death. This insidious process is often punctuated by incidental complications. These kidneys are very liable to infection which may be a mild pyelitis or severe sup-puration involving the cysts as

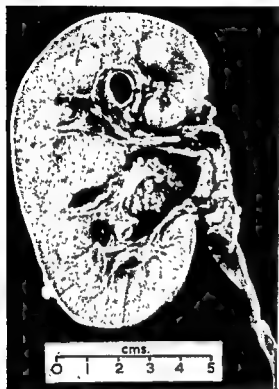


FIG. 202. *Papilloma of Renal Pelvis.* A section through the right kidney showing a papilloma growing from a lower pyramid and occupying the pelvis and one of the middle calices. Same case as Fig. 203.

well as the functioning tissue. Hæmaturia is quite common. Hypertension often develops.

The diagnosis of polycystic disease is usually fairly straightforward. The patient may present with renal pain, hæmaturia or urinary infection. He may even have felt an abdominal tumour. Usually there is palpable enlargement of both kidneys. The excretion pyelogram shows elongation, distortion and often enlargement of the calices, but in many cases the concentration of dye is rather poor so that details need to be filled in by instrumental pyelography. In general, the pyelographic findings somewhat resemble those due to tumour, but this causes difficulty only when the cystic changes predominantly affect one part of one kidney. The absence of a family history may cause difficulty. In any case the pyelographic findings always lag far behind the actual condition of the

kidneys, and it is quite common for polycystic disease to give a pyelogram which it is difficult to distinguish from the extremes of normal.

Surgery has little to offer in the treatment of polycystic disease. If the other kidney is functioning fairly well, nephrectomy may rarely be justifiable for severe complications such as persisting hæmaturia which is threatening life. There has been a tendency to revive Rovsing's operation

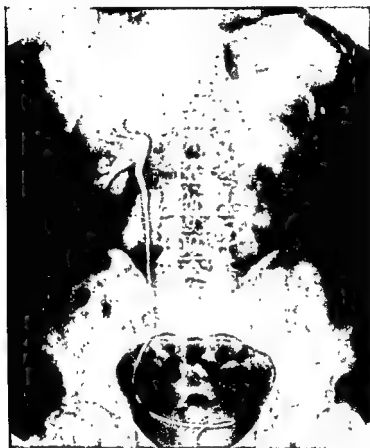


FIG. 203. *Papilloma of Renal Pelvis.* Instrumental (retrograde) pyelogram showing filling defect of pelvis and middle calix. Same case as Fig. 202.

in which the kidney is exposed and as many cysts as possible punctured. By this operation it is hoped to relieve the pressure on the functioning nephrons at least for a time, but its effectiveness cannot be proved.

**Solitary Cyst.** In this condition the kidney as a whole is healthy but contains usually one cyst or occasionally several. They are commonly situated at one pole of the kidney but may be embedded in its substance. Their size varies greatly but is often comparable with that of the kidney and seldom less than a quarter its size (Fig. 204). They are lined with flattened or cuboidal epithelium. Their pathogenesis is not known, but



renal tumours is poor. Deming's figure is 10 per cent. ten-year survival for all types.

**Polycystic Disease.** This is a developmental abnormality, almost always affecting both kidneys, in which the renal substance is to a varying extent replaced by multiple cysts. It is thought to arise from the imperfect union between the excretory and the collecting elements of the

nephron. The disease is familial (Mendelian recessive), but patients are often unaware that any of their relations have been affected. Two main types are recognised. In the commoner types the disease does not manifest itself until the patient is adult or even middle-aged, whereas in the other type the condition is evident at birth. In the infantile variety, the baby is still-born or dies very shortly after birth. In adults, the disease is very slowly progressive.

The cysts slowly crush the life out of the normal nephrons by obstructing the drainage channels and causing pressure atrophy of the tubular cells. Renal function gradually fails and uræmia leads on to death. This insidious process is often punctuated by incidental complications. These kidneys are very liable to infection which may be a mild pyelitis or severe sup-puration involving the cysts as

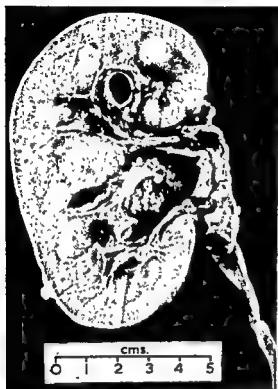


FIG. 202. *Papilloma of Renal Pelvis.* A section through the right kidney showing a papilloma growing from a lower pyramid and occupying the pelvis and one of the middle calices. Same case as Fig. 203.

well as the functioning tissue. Hæmaturia is quite common. Hypertension often develops.

The diagnosis of polycystic disease is usually fairly straightforward. The patient may present with renal pain, hæmaturia or urinary infection. He may even have felt an abdominal tumour. Usually there is palpable enlargement of both kidneys. The excretion pyelogram shows elongation, distortion and often enlargement of the calices, but in many cases the concentration of dye is rather poor so that details need to be filled in by instrumental pyelography. In general, the pyelographic findings somewhat resemble those due to tumour, but this causes difficulty only when the cystic changes predominantly affect one part of one kidney. The absence of a family history may cause difficulty. In any case the pyelographic findings always lag far behind the actual condition of the

kidneys, and it is quite common for polycystic disease to give a pyelogram which it is difficult to distinguish from the extremes of normal.

Surgery has little to offer in the treatment of polycystic disease. If the other kidney is functioning fairly well, nephrectomy may rarely be justifiable for severe complications such as persisting hamaturia which is threatening life. There has been a tendency to revive Rovsing's operation



FIG. 203. *Papilloma of Renal Pelvis.* Instrumental (retrograde) pyelogram showing filling defect of pelvis and middle calix. Same case as Fig. 202.

in which the kidney is exposed and as many cysts as possible punctured. By this operation it is hoped to relieve the pressure on the functioning nephrons at least for a time, but its effectiveness cannot be proved.

**Solitary Cyst.** In this condition the kidney as a whole is healthy but contains usually one cyst or occasionally several. They are commonly situated at one pole of the kidney but may be embedded in its substance. Their size varies greatly but is often comparable with that of the kidney and seldom less than a quarter its size (Fig. 204). They are lined with flattened or cuboidal epithelium. Their pathogenesis is not known, but

it is thought that they are retention cysts resulting from a developmental or inflammatory obstructive process. These cysts are harmless and often symptomless. If very large they may cause trouble by their weight and bulk. They may cause hæmaturia or at least may be found on investigation of a patient with hæmaturia. Their chief importance lies in the difficulty of distinguishing them from neoplasms both clinically and on pyelography. If a cyst causes symptoms, or if the possibility of tumour

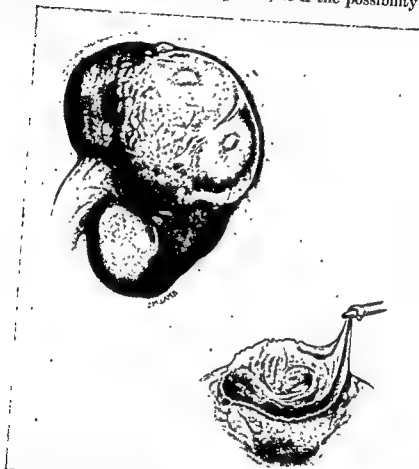


FIG. 204. *Renal Cyst.* A solitary cyst of the left kidney which had caused a filling defect in the pyelogram suggestive of tumour. The lower picture shows the appearance after removal of the projecting part of the cyst.

cannot be excluded, operation is necessary. Treatment consists simply in removing the part of the cyst which projects from the kidney surface (Fig. 204). Cysts embedded in the renal substance present a more difficult and dangerous problem and may require nephrectomy.

**Hypertension and Renal Disease.** The experiments of Wilson and Byrom showed that ischaemia of one kidney in the rat resulted in sustained hypertension. They also showed that in some cases the removal of the ischaemic kidney restored the blood pressure to normal, but that in the majority of rats with severe chronic hypertension the removal of the

ischemic kidney did not lower the blood pressure, or at least did not restore it to a normal level. These experiments are matched to some extent by clinical experience both in the hope they raise and in the warning they give. In the great majority of patients with unilateral renal disease and hypertension, removal of the diseased kidney has no permanent effect on the hypertension. The reason in most of these cases is simply that the renal disease is not the cause of the hypertension: in others it is possible that although the diseased kidney has caused the hypertension, the hypertension has produced changes in the other kidney which are capable of perpetuating the hypertension. There are, however, cases in which severe hypertension is permanently cured by the removal of a diseased kidney. The most hopeful type of disease is *chronic pyelonephritis*, but successful cases have also been reported with a number of other conditions including hydronephrosis, tuberculosis and tumour.

### OPERATIONS ON THE KIDNEY

Before operation the diagnosis must be soundly established in every detail. Diagnostic exploration is rarely justified, and then only if full and, if necessary, repeated investigations have failed to make the issue clear. The other kidney must be shown to be normal or at least in a condition to play the part required of it following operation. Any urinary infection must be under control as far as possible. For most renal operations, blood should be available in case transfusion becomes necessary. The kidney is relatively inaccessible for it lies mostly within the thoracic cage. Its convex border is fairly near the posterior plane of the body, but the vascular pedicle is almost as near the front of the body as the back and runs upwards as well as medially from the kidney. Pathological conditions which often cause difficulty in exposure and mobilisation are induration of the perinephric tissues of the kidney itself (commonly inflammatory) or increased vascularity (as with tumours). The usual approach is extraperitoneal through the loin, and many experienced surgeons use it for all types of case. Its main limitations are that it is low (caudal) and that the kidney lies between the surgeon and the vascular pedicle. When especially free exposure is needed with direct access to the pedicle (as for large tumours), there are important advantages in an anterior transperitoneal, or even a posterosuperior transpleural exposure.

**The Lumbar Approach.** The position of the patient is important. The patient lies on the side with the lumbar spine strongly flexed laterally (by a bridge, sandbag or "breaking" of the table), so as to open up the space between the costal margin and iliac crest. The beam of the light should be deflected somewhat upwards (towards the patient's head). The twelfth rib is identified by touch after the X-rays have been inspected, and the incision begins  $\frac{1}{2}$  inch below it at the outer border of the erector spinæ muscle and runs obliquely downwards and forwards for 6 inches

it is thought that they are retention cysts resulting from a developmental or inflammatory obstructive process. These cysts are harmless and often symptomless. If very large they may cause trouble by their weight and bulk. They may cause hæmaturia or at least may be found on investigation of a patient with hæmaturia. Their chief importance lies in the difficulty of distinguishing them from neoplasms both clinically and on pyelography. If a cyst causes symptoms, or if the possibility of tumour

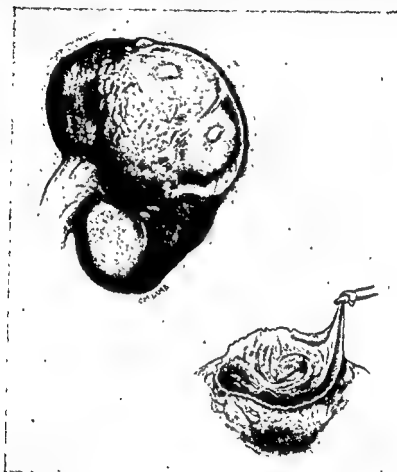


FIG. 204. *Renal Cyst.* A solitary cyst of the left kidney which had caused a filling defect in the pyelogram suggestive of tumour. The lower picture shows the appearance after removal of the projecting part of the cyst.

cannot be excluded, operation is necessary. Treatment consists simply in removing the part of the cyst which projects from the kidney surface (Fig. 204). Cysts embedded in the renal substance present a more difficult and dangerous problem and may require nephrectomy.

**Hypertension and Renal Disease.** The experiments of Wilson and Byrom showed that ischaemia of one kidney in the rat resulted in sustained hypertension. They also showed that in some cases the removal of the ischaemic kidney restored the blood pressure to normal, but that in the majority of rats with severe chronic hypertension the removal of the

ischaemic kidney did not lower the blood pressure, or at least did not restore it to a normal level. These experiments are matched to some extent by clinical experience both in the hope they raise and in the warning they give. In the great majority of patients with unilateral renal disease and hypertension, removal of the diseased kidney has no permanent effect on the hypertension. The reason in most of these cases is simply that the renal disease is not the cause of the hypertension: in others it is possible that although the diseased kidney has caused the hypertension, the hypertension has produced changes in the other kidney which are capable of perpetuating the hypertension. There are, however, cases in which severe hypertension is permanently cured by the removal of a diseased kidney. The most hopeful type of disease is *chronic pyelonephritis*, but successful cases have also been reported with a number of other conditions including hydronephrosis, tuberculosis and tumour.

### OPERATIONS ON THE KIDNEY

Before operation the diagnosis must be soundly established in every detail. Diagnostic exploration is rarely justified, and then only if full and, if necessary, repeated investigations have failed to make the issue clear. The other kidney must be shown to be normal or at least in a condition to play the part required of it following operation. Any urinary infection must be under control as far as possible. For most renal operations, blood should be available in case transfusion becomes necessary. The kidney is relatively inaccessible for it lies mostly within the thoracic cage. Its convex border is fairly near the posterior plane of the body, but the vascular pedicle is almost as near the front of the body as the back and runs upwards as well as medially from the kidney. Pathological conditions which often cause difficulty in exposure and mobilisation are induration of the perinephric tissues of the kidney itself (commonly inflammatory) or increased vascularity (as with tumours). The usual approach is extraperitoneal through the loin, and many experienced surgeons use it for all types of case. Its main limitations are that it is low (caudal) and that the kidney lies between the surgeon and the vascular pedicle. When especially free exposure is needed with direct access to the pedicle (as for large tumours), there are important advantages in an anterior transperitoneal, or even a posterosuperior transpleural exposure.

**The Lumbar Approach.** The position of the patient is important. The patient lies on the side with the lumbar spine strongly flexed laterally (by a bridge, sandbag or "breaking" of the table), so as to open up the space between the costal margin and iliac crest. The beam of the light should be deflected somewhat upwards (towards the patient's head). The twelfth rib is identified by touch after the X-rays have been inspected, and the incision begins  $\frac{1}{2}$  inch below it at the outer border of the erector spinæ muscle and runs obliquely downwards and forwards for 11 inches

it is thought that they are retention cysts resulting from a developmental or inflammatory obstructive process. These cysts are harmless and often symptomless. If very large they may cause trouble by their weight and bulk. They may cause haematuria or at least may be found on investigation of a patient with haematuria. Their chief importance lies in the difficulty of distinguishing them from neoplasms both clinically and on pyelography. If a cyst causes symptoms, or if the possibility of tumour

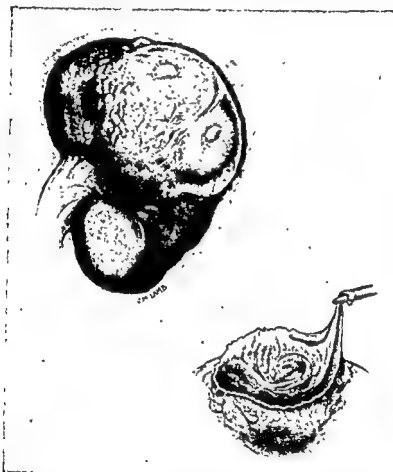


FIG. 204. *Renal Cyst.* A solitary cyst of the left kidney which had caused a filling defect in the pyelogram suggestive of tumour. The lower picture shows the appearance after removal of the projecting part of the cyst.

cannot be excluded, operation is necessary. Treatment consists simply in removing the part of the cyst which projects from the kidney surface (Fig. 204). Cysts embedded in the renal substance present a more difficult and dangerous problem and may require nephrectomy.

**Hypertension and Renal Disease.** The experiments of Wilson and Byrom showed that ischaemia of one kidney in the rat resulted in sustained hypertension. They also showed that in some cases the removal of the ischaemic kidney restored the blood pressure to normal, but that in the majority of rats with severe chronic hypertension the removal of the

ischemic kidney did not lower the blood pressure, or at least did not restore it to a normal level. These experiments are matched to some extent by clinical experience both in the hope they raise and in the warning they give. In the great majority of patients with unilateral renal disease and hypertension, removal of the diseased kidney has no permanent effect on the hypertension. The reason in most of these cases is simply that the renal disease is not the cause of the hypertension: in others it is possible that although the diseased kidney has caused the hypertension, the hypertension has produced changes in the other kidney which are capable of perpetuating the hypertension. There are, however, cases in which severe hypertension is permanently cured by the removal of a diseased kidney. The most hopeful type of disease is *chronic pyelonephritis*, but successful cases have also been reported with a number of other conditions including hydronephrosis, tuberculosis and tumour.

### OPERATIONS ON THE KIDNEY

Before operation the diagnosis must be soundly established in every detail. Diagnostic exploration is rarely justified, and then only if full and, if necessary, repeated investigations have failed to make the issue clear. The other kidney must be shown to be normal or at least in a condition to play the part required of it following operation. Any urinary infection must be under control as far as possible. For most renal operations, blood should be available in case transfusion becomes necessary. The kidney is relatively inaccessible for it lies mostly within the thoracic cage. Its convex border is fairly near the posterior plane of the body, but the vascular pedicle is almost as near the front of the body as the back and runs upwards as well as medially from the kidney. Pathological conditions which often cause difficulty in exposure and mobilisation are induration of the perinephric tissues of the kidney itself (commonly inflammatory) or increased vascularity (as with tumours). The usual approach is extraperitoneal through the loin, and many experienced surgeons use it for all types of case. Its main limitations are that it is low (caudal) and that the kidney lies between the surgeon and the vascular pedicle. When especially free exposure is needed with direct access to the pedicle (as for large tumours), there are important advantages in an anterior transperitoneal, or even a posterosuperior transpleural exposure.

**The Lumbar Approach.** The position of the patient is important. The patient lies on the side with the lumbar spine strongly flexed laterally (by a bridge, sandbag or "breaking" of the table), so as to open up the space between the costal margin and iliac crest. The beam of the light should be deflected somewhat upwards (towards the patient's head). The twelfth rib is identified by touch after the X-rays have been inspected, and the incision begins  $\frac{1}{2}$  inch below it at the outer border of the erector spinæ muscle and runs obliquely downwards and forwards for 6 inches



it is thought that they are retention cysts resulting from a developmental or inflammatory obstructive process. These cysts are harmless and often symptomless. If very large they may cause trouble by their weight and bulk. They may cause hæmaturia or at least may be found on investigation of a patient with hæmaturia. Their chief importance lies in the difficulty of distinguishing them from neoplasms both clinically and on pyelography. If a cyst causes symptoms, or if the possibility of tumour

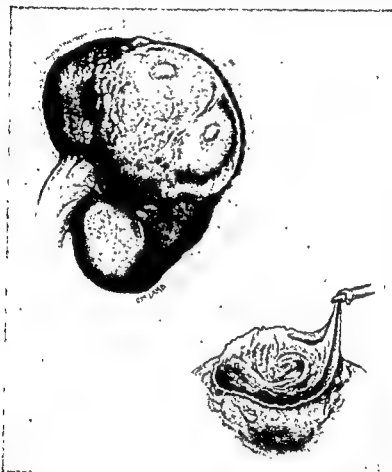


FIG. 204. *Renal Cyst.* A solitary cyst of the left kidney which had caused a filling defect in the pyelogram suggestive of tumour. The lower picture shows the appearance after removal of the projecting part of the cyst.

cannot be excluded, operation is necessary. Treatment consists simply in removing the part of the cyst which projects from the kidney surface (Fig. 204). Cysts embedded in the renal substance present a more difficult and dangerous problem and may require nephrectomy.

**Hypertension and Renal Disease.** The experiments of Wilson and Byrom showed that ischæmia of one kidney in the rat resulted in sustained hypertension. They also showed that in some cases the removal of the ischæmic kidney restored the blood pressure to normal, but that in the majority of rats with severe chronic hypertension the removal of the

ischaemic kidney did not lower the blood pressure, or at least did not restore it to a normal level. These experiments are matched to some extent by clinical experience both in the hope they raise and in the warning they give. In the great majority of patients with unilateral renal disease and hypertension, removal of the diseased kidney has no permanent effect on the hypertension. The reason in most of these cases is simply that the renal disease is not the cause of the hypertension: in others it is possible that although the diseased kidney has caused the hypertension, the hypertension has produced changes in the other kidney which are capable of perpetuating the hypertension. There are, however, cases in which severe hypertension is permanently cured by the removal of a diseased kidney. The most hopeful type of disease is *chronic pyelonephritis*, but successful cases have also been reported with a number of other conditions including hydronephrosis, tuberculosis and tumour.

### OPERATIONS ON THE KIDNEY

Before operation the diagnosis must be soundly established in every detail. Diagnostic exploration is rarely justified, and then only if full and, if necessary, repeated investigations have failed to make the issue clear. The other kidney must be shown to be normal or at least in a condition to play the part required of it following operation. Any urinary infection must be under control as far as possible. For most renal operations, blood should be available in case transfusion becomes necessary. The kidney is relatively inaccessible for it lies mostly within the thoracic cage. Its convex border is fairly near the posterior plane of the body, but the vascular pedicle is almost as near the front of the body as the back and runs upwards as well as medially from the kidney. Pathological conditions which often cause difficulty in exposure and mobilisation are induration of the perinephric tissues of the kidney itself (commonly inflammatory) or increased vascularity (as with tumours). The usual approach is extraperitoneal through the loin, and many experienced surgeons use it for all types of case. Its main limitations are that it is low (caudal) and that the kidney lies between the surgeon and the vascular pedicle. When especially free exposure is needed with direct access to the pedicle (as for large tumours), there are important advantages in an anterior transperitoneal, or even a posterosuperior transpleural exposure.

**The Lumbar Approach.** The position of the patient is important. The patient lies on the side with the lumbar spine strongly flexed laterally (by a bridge, sandbag or "breaking" of the table), so as to open up the space between the costal margin and iliac crest. The beam of the light should be deflected somewhat upwards (towards the patient's head). The twelfth rib is identified by touch after the X-rays have been inspected, and the incision begins  $\frac{1}{2}$  inch below it at the outer border of the erector spinae muscle and runs obliquely downwards and forwards for 6 inches

it is thought that they are retention cysts resulting from a developmental or inflammatory obstructive process. These cysts are harmless and often symptomless. If very large they may cause trouble by their weight and bulk. They may cause hæmaturia or at least may be found on investigation of a patient with hæmaturia. Their chief importance lies in the difficulty of distinguishing them from neoplasms both clinically and on pyelography. If a cyst causes symptoms, or if the possibility of tumour

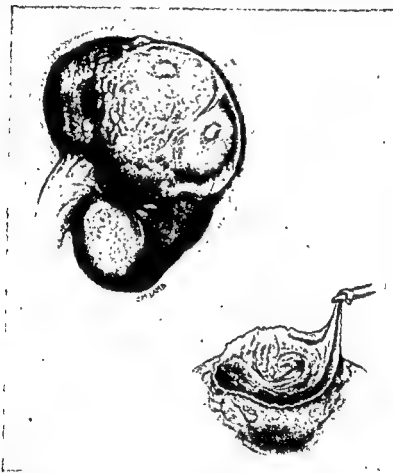


FIG. 204. *Renal Cyst.* A solitary cyst of the left kidney which had caused a filling defect in the pyelogram suggestive of tumour. The lower picture shows the appearance after removal of the projecting part of the cyst.

cannot be excluded, operation is necessary. Treatment consists simply in removing the part of the cyst which projects from the kidney surface (Fig. 204). Cysts embedded in the renal substance present a more difficult and dangerous problem and may require nephrectomy.

**Hypertension and Renal Disease.** The experiments of Wilson and Byrom showed that ischaemia of one kidney in the rat resulted in sustained hypertension. They also showed that in some cases the removal of the ischaemic kidney restored the blood pressure to normal, but that in the majority of rats with severe chronic hypertension the removal of the

## CHAPTER 27

### DISEASES OF THE URETER

The ureter is investigated chiefly by cystoscopy and radiography. At cystoscopy the ureteric orifice can be directly inspected and slightly less direct information obtained about the intramural ureter (i.e. the part which passes through the bladder wall). The movements of the orifice and the type of efflux provide evidence of the functional state of the ureter. In the excretion pyelogram the ureter is frequently outlined, but usually only in part of its course depending on the position reached by the peristaltic wave when the exposure is made. A more complete outline of the ureter may be obtained by passing a ureteric catheter and injecting opaque fluid directly into it, but even with this method it may not be possible to fill the whole ureter as some dye may flow back round the catheter into the bladder: also it is sometimes impossible to pass the catheter far enough. The method of bulb injection may solve some of these problems: a ureteric catheter with a swelling just short of its tip is plugged into the ureteric orifice and retrograde injection carried out (Fig. 183D).

**Congenital Anomalies.** Duplication of the ureter in the upper part of its course is fairly common, and duplication throughout its course is not rare (Fig. 184). Such anomalies are sometimes associated with imperfect drainage and may therefore lead to infection or hydronephrosis in one or both parts of the kidney. These conditions are usually brought to light during the investigation of urinary infection or renal pain. The uncomplicated anatomical abnormality is only important when the ureter is to be operated on (e.g. transplantation into the colon) when it complicates the technique of operation; if its presence is not recognised, more serious consequences may arise (e.g. extravasation of urine if the unnoticed ureter is divided, or at least the need for a subsequent operation to transplant the remaining ureter when its presence is revealed by the continued secretion of urine into the bladder). In many cases the diagnosis is made or at least suggested by excretion pyelography. If the duplication is complete the two orifices can be seen on cystoscopy, the lower one (i.e. the orifice nearer the internal urinary meatus) corresponding with the upper component of the kidney. Sometimes one of the orifices is ectopic, opening into the urethra or vagina and so causing incontinence of urine.

*Stenosis of the ureteric orifice* may occur with or without duplication of the ureter. The obstruction leads to a gradual dilatation of the ureter and renal pelvis and progressive destruction of the kidney. It may be

or more, dividing the whole thickness of the flank muscles. The subcostal nerve is carefully watched for, and if it is encountered it is displaced downwards. The twelfth rib is dislocated upwards after dividing the lateral lumbocostal ligament. Bleeding from the posterior end of the wound under the rib may need to be controlled by a stitch. The wound edges are retracted, the peritoneum is pushed forward and the *fascia of Gerota* (renal fascia) cleanly incised. The perinephric fat is then stripped off until the kidney can be grasped. The next steps depend on the nature of the operation to be done.

**Nephrectomy.** The kidney is systematically mobilised by stripping the fat off it and dividing any vessel running direct to its outer surface (there is often one at the upper pole). After the lower pole is cleared, the ureter is identified and divided between ligatures as low as can be reached. Mobilisation is now completed by drawing the kidney with its attached ureter out of the wound and stripping the hilar surface until only the vascular pedicle remains. This is carefully defined as a whole; individual vessels need not be identified. The whole pedicle is secured by two separate encircling ligatures of any strong material and then divided between the distal ligature and the hilum. It is usual but not always necessary to drain the wound. The muscles are sutured with catgut, preferably with interrupted stitches, care being taken not to include any nerve branches. This standard nephrectomy procedure must often be varied for reasons of pathology or sometimes anatomy.

**Nephrostomy.** The kidney is turned forward and mobilised just enough to expose the posterior surface of the pelvis. Two stay sutures are inserted into this, and it is incised between them. A sucker is used to keep the wound dry. A long curved forceps is inserted into the renal pelvis and passed carefully into one of the lower calices and then thrust through the renal substance at a point where it is judged a tube will lie fitly in relation to the wound. A rubber tube of appropriate size (15 Charrière suffices if clear urine is to be drained) is grasped in the forceps and drawn into the pelvis. It is secured with a catgut stitch where it passes through the renal capsule, and the incision in the pelvis is then sutured. The wound is closed round the tube after introducing a drain down to the incision in the renal pelvis. Finally the tube is proved to drain freely by syringing it with saline.

in the wall of the ureter and the other side washed by the stream of urine. A stone may remain in such a situation for weeks or months without causing symptoms or complications. Sometimes a stone which has been lodged in the ureter for months suddenly floats free and passes out into the bladder without obvious cause. This type of prolonged and often harmless lodgement is most likely to occur in the lower quarter of the ureter, often just outside the bladder wall; it is much less common in the upper half of the ureter where lodgement often means unyielding obstruction.

By far the most serious effect of a ureteric calculus is obstruction to the flow of urine which, if it continues unrelieved, causes progressive deterioration of renal function. The dilatation of the urinary tract above the stone is seldom great and usually affects the calices more than the pelvis and ureter. Infection is another important complication of ureteric calculus, but it often does not develop even when obstruction is severe.

The diagnosis of ureteric calculus is usually straightforward, but it is sometimes very difficult. The main and often the only symptom is pain, usually a bout of severe renal or ureteric colic. Sometimes (usually when the stone is in the lower end of the ureter) there are also symptoms referable to the bladder such as frequency, pain on micturition, or strangury. There may be hæmaturia. Sometimes the symptoms suggest unilateral pyelitis, but the pain is disproportionately severe compared with the other features. There are usually no physical signs unless the kidney is infected. Rarely the stone can be felt in the lower part of the ureter *per rectum* or (more likely) *per vaginam*. The diagnosis is sometimes settled by the patient's passing the stone and finding it in his urine, but often the passing of the stone is hardly noticed by the patient.

In most cases the diagnosis is decided by radiography. The control film shows the characteristic small irregular oval shadow in the course of the ureter with its long axis in the line of the ureter (Fig. 206). The shadow may be very small, for a calculus no bigger than a grape stone may produce severe symptoms. Other calcified objects may cause difficulty in diagnosis, particularly phleboliths near the lower end of the ureter. These are usually rounder and smoother and more definite than a ureteric calculus, but their intrinsic appearance may be insufficient to prove their identity. When a calculus lies in front of the lateral mass of the sacrum, quite a common occurrence, it may be difficult or impossible to detect in the X-ray. The excretion pyelogram often shows some degree of dilatation of the urinary tract above the stone or lack of concentration of the urine, but it may show no abnormality at all if the stone is causing no obstruction at the time. The easiest cases are those in which the ureter is overfilled down to the level of the suspected shadow and empty below it. Sometimes the ureter may not show up even when the pelvis and calices are dilated. Acute ureteric obstruction may turn an excretion pyelogram into a "nephrogram," i.e. a uniform density of the whole renal shadow due to damming back in the collecting tubules of

complicated by infection of the stagnating urine or by stone formation. Sometimes the stenosis produces an interesting condition at the terminal (intravesical) part of the ureter known as *ureterocele* in which the contractions of the ureter inflate the lower end, blowing it up into a small balloon protruding within the bladder, and then it slowly deflates as the urine spurts out of the narrow orifice, blowing up again at the next ureteric contraction (Fig. 205). The cystoscopic picture is very characteristic. The treatment is enlargement of the orifice usually by endoscopic diathermy incision. Congenital anomalies of the ureter are also discussed in Chap. 24.

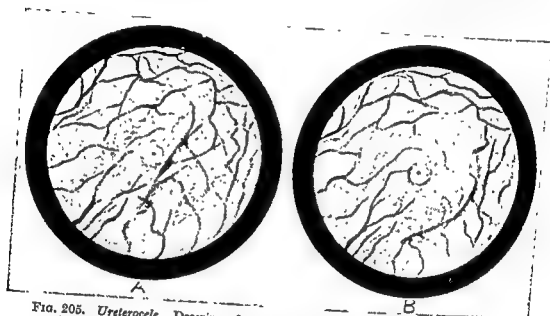


Fig. 205. *Ureterocele*. Drawing of cystoscopic view of left ureteric orifice. Note its small size. A. The ureter at rest. B. The ureter contracting and inflating the ureterocele.

**Ureteric Calculus** is the commonest disease of the ureter. In almost all cases the stone forms originally in the kidney and lodges in a normal ureter. Rarely the ureter itself is abnormal as, for example, when there is meatal stenosis. The majority of stones which enter the ureter eventually pass spontaneously into the bladder, but there are factors which often arrest them temporarily and sometimes permanently. The first factor is local spasm of the ureteric muscle which grips the stone, obstructs the ureter and causes the typical symptom, ureteric colic. In most cases the spasm soon relaxes, the obstruction is relieved and the stone passes on, but other complicating factors may arise. Oedema may quickly develop and narrow the lumen. When the stone is squeezed by the ureter pressure ulceration of the mucous membrane occurs, and in some cases this process goes on to excavate a shallow diverticulum lined by granulation tissue in which the stone lodges. When the spasm relaxes the urine flows again, but the stone remains with one side partly embedded

in the wall of the ureter and the other side washed by the stream of urine. A stone may remain in such a situation for weeks or months without causing symptoms or complications. Sometimes a stone which has been lodged in the ureter for months suddenly floats free and passes out into the bladder without obvious cause. This type of prolonged and often harmless lodgement is most likely to occur in the lower quarter of the ureter, often just outside the bladder wall; it is much less common in the upper half of the ureter where lodgement often means unyielding obstruction.

By far the most serious effect of a ureteric calculus is obstruction to the flow of urine which, if it continues unrelieved, causes progressive deterioration of renal function. The dilatation of the urinary tract above the stone is seldom great and usually affects the calices more than the pelvis and ureter. Infection is another important complication of ureteric calculus, but it often does not develop even when obstruction is severe.

The diagnosis of ureteric calculus is usually straightforward, but it is sometimes very difficult. The main and often the only symptom is pain, usually a bout of severe renal or ureteric colic. Sometimes (usually when the stone is in the lower end of the ureter) there are also symptoms referable to the bladder such as frequency, pain on micturition, or strangury. There may be hæmaturia. Sometimes the symptoms suggest unilateral pyelitis, but the pain is disproportionately severe compared with the other features. There are usually no physical signs unless the kidney is infected. Rarely the stone can be felt in the lower part of the ureter *per rectum* or (more likely) *per vaginam*. The diagnosis is sometimes settled by the patient's passing the stone and finding it in his urine, but often the passing of the stone is hardly noticed by the patient.

In most cases the diagnosis is decided by radiography. The control film shows the characteristic small irregular oval shadow in the course of the ureter with its long axis in the line of the ureter (Fig. 206). The shadow may be very small, for a calculus no bigger than a grape stone may produce severe symptoms. Other calcified objects may cause difficulty in diagnosis, particularly phleboliths near the lower end of the ureter. These are usually rounder and smoother and more definite than a ureteric calculus, but their intrinsic appearance may be insufficient to prove their identity. When a calculus lies in front of the lateral mass of the sacrum, quite a common occurrence, it may be difficult or impossible to detect in the X-ray. The excretion pyelogram often shows some degree of dilatation of the urinary tract above the stone or lack of concentration of the urine, but it may show no abnormality at all if the stone is causing no obstruction at the time. The easiest cases are those in which the ureter is overfilled down to the level of the suspected shadow and empty below it. Sometimes the ureter may not show up even when the pelvis and calices are dilated. Acute ureteric obstruction may turn an excretion pyelogram into a "nephrogram," i.e. a uniform density of the whole renal shadow due to damming back in the collecting tubules of



concentrated dye. Such a kidney is likely to recover well when the obstruction is removed. The identity of a doubtful shadow can usually be settled by X-ray after ureteric catheterisation. The catheter is run up to the kidney and X-rays are taken in two different planes: the shadow cast by a ureteric calculus overlies the catheter in both films. If the catheter can be passed up to the stone but not beyond it, the same technique may be used, but the mere fact that the catheter cannot be



FIG. 206. *Ureteric Calculus. Plain X-ray.* The stone is in the lower end of the right ureter approaching the ureteric orifice. It is near the midline and its long axis is almost horizontal. It was subsequently passed spontaneously. The oval shadow near the left ischial spine is a phlebolith.

passed beyond a certain point must not be accepted as proving the presence of a calculus; technical hitches are a commoner cause of this occurrence. Sometimes a ureterogram may be needed to prove the diagnosis. When the stone is in the lower end of the ureter, there may be characteristic cystoscopic evidence, chiefly cedema, and sometimes the stone itself may be seen peeping out of the orifice.

In the management of ureteric calculus, the first consideration is to preserve or to restore renal function. The next thing to bear in mind is that the great majority of stones pass spontaneously, and this is always the best result. If the kidney is functioning normally, the stone should be given every opportunity to pass unless there are positive reasons

against this course. Thus if the stone is obviously too large to pass naturally (i.e. over 2 cm. diameter in the lower part of the ureter and over 1 cm. in the upper) it should be removed. If it is very jagged it is unlikely to pass naturally (Fig. 207). Frequent severe pain, urinary infection and hamaturia may also be indications to intervene. Time alone provides no automatic signal for intervention, for a stone may pass harmlessly after remaining many months in the ureter, but some surgeons set an arbitrary limit, e.g. three months, as a matter of practical expediency. In any case the state of renal function should be checked regularly during the waiting period. The passage of a stone may be helped by an increased fluid intake, exercise and relaxant drugs such as propantheline (Probanthine).

When intervention is required, the treatment depends on the position and size of the stone and other considerations. A stone lying in the intramural part of the ureter can usually be helped out by endoscopic means, particularly by enlargement of the orifice by diathermy incision. Stones above this level but below the pelvic brim, provided they are less than 2 cm. in diameter and not jagged, can often be helped out by intra-ureteric manipulations. The simplest and safest of these is the passage of a ureteric catheter past the stone. The catheter may be withdrawn at once or left in place for twenty-four hours. More positive methods are the use of various types of extractor such as Howard's (corkscrew), Councill's (basket) or the looped catheter. Their use is not often necessary and is moreover a little risky. When intervention is clearly necessary, open operation is usually the quickest and safest method of removing the stone. If there is good evidence that renal function is irrecoverably lost, nephrectomy should be done.

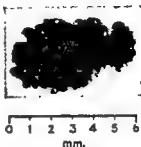


FIG. 207. *Ureteric Calculus*. Photograph of typical small oval stone with very irregular, jagged surface.

**Ureteric Tumours.** These tumours are rare. They are usually transitional-cell papillomas or carcinomas. The symptoms are hæmaturia and those due to ureteric obstruction. Sometimes these tumours are associated with others in the renal pelvis or bladder, and the likelihood of this determines the form of treatment and follow up. The diagnosis depends on radiography (pyelogram and ureterogram) which shows evidence of obstruction of the urinary tract and a filling defect (Fig. 208). The treatment is usually nephroureterectomy, but if the tumour is histologically benign and the rest of the renal tract can be pronounced clear, it is sometimes justifiable to resect the affected segment of the ureter and to replace it with a graft of ileum. Follow up is the same as for other transitional-cell tumours of the urinary tract.

**Injuries of the Ureter.** Apart from penetrating injuries which are rare in peacetime, the ureter is seldom injured except inadvertently during

operations. Owing to its slenderness and mobility, the ureter is very liable to injury during operations in the pelvis. Above the pelvic brim the left ureter lies very close to the lower part of the colon and mesocolon. The best way to avoid injury to the ureter is to identify it early in the operation. In some cases it is helpful to catheterise the ureters beforehand to render them palpable.



FIG. 208. *Papillomata of Ureter.* Ureterogram (left). Extensive blotchy filling defect. The kidney was hydronephrotic and functionless due to the ureteric obstruction. The patient had been treated for vesical papilloma two years previously when excretion pyelography had shown normal kidneys.

An injury to the ureter recognised at the time and repaired forthwith is usually followed by a good result. If the ureter is completely obstructed by a ligature the kidney usually ceases to function without other complications. If the ureter is partly obstructed (as by a lateral ligature) hydronephrosis and infection are likely to follow. Sometimes a ligature inadvertently applied to the ureter cuts through it after operation; then a ureteric fistula develops (e.g. a ureterovaginal fistula). When injury to

the ureter is diagnosed repair is done if possible, but often the ureter has to be implanted into the bladder or transplanted into the colon, or a defect bridged by a graft taken from the ileum. Nephrectomy may be necessitated by poverty of renal function or severe infection.

**Ureteric Reflux.** The normal ureter has a valvular opening into the bladder. Injury at operation, neurological disturbance (e.g. after spinal injuries), and local inflammatory disease may sometimes render this mechanism incompetent. The effect is that when the bladder contracts urine is forced up into the kidney which becomes hydronephrotic and liable to infection.

**Megalo-ureter or Hydro-ureter.** This is a condition in which the ureter is much dilated. It may be associated with obstruction or reflux, but sometimes no local abnormality of the lower end of the ureter can be found. There is usually some degree of hydronephrosis. Urinary infection is common. The pathogenesis is seldom clear and the treatment unsatisfactory.

### OPERATIONS ON THE URETER

**Exposure.** The ureter is usually approached extraperitoneally. Its upper end is reached easily by any kidney incision and particularly the standard oblique lumbar incision. In its middle reaches the ureter is approached from in front by either cutting or splitting the muscles of the abdominal wall and stripping the peritoneum medially at the appropriate level. Here it is in a relatively anterior plane and is easily accessible. The ureter is least accessible at its lower end as it lies very deep in the pelvis, and its exposure in this situation is often hampered by bleeding from small veins. An ample median or paramedian incision is employed. In the female the ureter passes close to the lateral vaginal fornix allowing an operative approach which although very restricted is sometimes advantageous.

**Ureterolithotomy.** The first essential is that an X-ray should be taken immediately before operation in case the stone has moved unexpectedly. As soon as the ureter is exposed it should be secured by passing a tape around it above the stone to prevent the stone from slipping upwards out of reach and perhaps even up to the kidney. The stone is located, stay sutures are inserted into the ureter which is incised directly on to the stone. After removal of the stone a ureteric catheter is passed upwards to the kidney and downwards into the bladder to demonstrate the patency of the lumen throughout its course. The incision in the ureter may be sutured or left unsutured, but the wound is always drained.

**Diversion of the Urine from the Bladder.** This is usually done by means of operations on the ureter. The indications include : (1) As a prelude or accompaniment of total cystectomy ; (2) inoperable carcinoma of the bladder with severe local symptoms ; (3) contracture of the bladder due to tuberculosis ; (4) intractable bladder pain due to other causes, e.g.

Hunner's ulcer, radiotherapeutic cystitis ; (5) incurable incontinence of urine, e.g. inoperable vesicovaginal fistula ; (6) ectopia vesicæ.

Diversion of the urine is a serious step, for completely normal renal function is seldom maintained afterwards and fatal renal failure may ensue.

*Ureterocolic anastomosis* is the commonest operation for permanent diversion of the urine above the level of the bladder. Provided the rectal sphincter is not paralysed, the colon provides a continent and capacious reservoir for urine, and most patients are very comfortable after the operation. The serious disadvantage of the operation is that afterwards many patients develop hyperchloræmic acidosis or renal deterioration or both. In hyperchloræmic acidosis, the plasma chloride is raised and the bicarbonate lowered. It has been shown that if urine is retained in the colon, the chloride is absorbed more rapidly than the sodium and this is probably the main cause of the hyperchloræmic acidosis. Patients who die from this condition are always found to have some degree of pyelonephritis and sometimes it is severe. This would account for the acidosis and the increased blood urea which often accompany the raised chloride level. Sometimes a very low level of serum potassium is another finding. Two complications which amply account for renal infection and deterioration are known to occur at the site of the ureterocolic junction ; in some cases stricture develops with resultant ureteric obstruction, and it has been shown experimentally that techniques which do not include direct suture of mucosa to mucosa are liable to this complication ; in other cases, although the anastomosis remains of adequate calibre, in fact partly because of this, reflux of colon contents occurs up the ureter into the kidney. The best results are therefore obtained by operations which construct a valve and make direct mucosal union. *Leadbetter's operation* meets these requirements.

A bout of hyperchloræmic acidosis is treated by an indwelling rectal tube, antibiotics for the presumed renal infection, and intravenous fluids adjusted according to the chemical estimations. In most cases M/6 sodium lactate solution will be suitable. If potassium is needed it is best given by mouth, for intravenous administration requires very strict precautions if the patient's renal function is defective.

*Cutaneous ureterostomy* is another means of diverting the urine. It is rather less damaging to the kidneys than ureterocolic anastomosis, but hydronephrosis and infection are liable to occur to some extent. The greatest disadvantage is the external fistulæ. An adhesive apparatus is best, but it requires careful and expert attention on the part of the patient if it is not to leak. A more recent method is to construct an artificial bladder from an isolated loop of ileum and implant the ureters into it. It appears to prevent hyperchloræmia and is less liable to be followed by renal infection, but it has a higher immediate mortality than ureterocolic anastomosis and furthermore there is an external incontinent fistula which requires an adhesive apparatus and bag.

## CHAPTER 28

### THE BLADDER

**Congenital Abnormalities.** These are remarkably uncommon considering the relative frequency of anomalies of the kidney, ureter and urethra. The commonest abnormalities are those of the ureteric orifices, but they are best considered under diseases of the ureter (Chap. 27). Congenital obstruction at the bladder neck does sometimes occur, but it has no clearly defined structural characteristics. Ectopia vesicæ or exstrophy of the bladder is a rare and special problem (Chap. 15). Diverticula of the bladder, even in young children, are almost always secondary to obstruction at the bladder neck, but they are very occasionally of developmental origin. Failure of the urachus to close may rarely be the cause of a diverticulum, a cyst or an abdominal urinary fistula (Chap. 15).

**Injuries.** Injuries of the bladder may be either closed or penetrating ; in either case the wound in the bladder may be extraperitoneal or intraperitoneal. In fractures of the pelvis, the bladder may be torn by a jagged fragment of bone. In crushing injuries without fracture, a distended bladder is more liable to rupture than an empty one. The bladder is liable to accidental injury during surgical operations in its vicinity, particularly hysterectomy and operations for inguinal and femoral hernia. Obstetric injuries may occur with or without instrumentation. Perforation of the bladder may be caused by endoscopic operations on it. If rupture of the bladder is suspected, it can usually be confirmed or excluded, and it should always be suspected in any injury, open or closed, of the lower abdomen or pelvis including penetrating wounds of the thigh and buttock. If a patient has spontaneously passed clear urine since the accident, rupture of the bladder can be excluded, but in serious injuries shock and reflex retention often prevent micturition so that a catheter must be passed. If clear urine is obtained, the bladder can be considered *intact* ; if no urine or blood-stained urine is withdrawn rupture must be diagnosed provisionally and cystoscopy done for confirmation. A cystogram may give a doubtful result and even more equivocal may be the outcome of running into the bladder a measured volume of fluid and attempting to check its return. If ever there is doubt the bladder should be explored. In fracture of the pelvis, it may be difficult to distinguish avulsion of the membranous urethra from rupture of the bladder. Blood-stained urine after trauma may be due to renal injury ; if there is doubt about the origin of the bleeding, cystoscopy will settle the matter. Rupture of the bladder requires operation immediately or as soon as the patient is fit enough. The rent should be closed whenever possible and the bladder drained suprapubically. Accidental injury of the bladder

during operation (e.g. femoral herniotomy) is treated by immediate repair and in such cases drainage by urethral catheter usually suffices. The important thing is to recognise that the bladder has been injured.

### CYSTITIS

Acute cystitis is usually due to infection of the bladder with bacteria, most commonly *Bact. coli*. The infection usually reaches the bladder from some other part of the urinary system, either from the kidneys or the urethra and its surrounding glands. The short urethra in the female is a common route of infection. In the male, the infection often spreads from the prostate. Cystitis may also arise from adjacent infective foci, e.g. in the colon or uterine cervix. The symptoms are painful frequency of micturition, often strangury and sometimes retention of urine. *Strangury* is the frequent painful passage of small amounts of urine which does not relieve a continuous desire to micturate. The urine may be cloudy or thick due to the presence of pus or mucus or both, and hæmaturia is common, occurring typically at the end of the stream. There are usually no abnormal physical signs and the temperature is often not raised unless there is an associated pyelitis or prostatitis. Microscopy of the urine shows pus cells and usually red cells; culture yields the causative organism. Instrumentation of the bladder should generally be avoided in acute cystitis but occasionally cystoscopy must be done. It reveals a generalised redness of the mucous membrane so that the pattern of individual vessels is lost. There is often œdema or even shallow ulcers coated with fibrin or mucus. The bladder is very irritable so that its capacity is small. It bleeds easily. The treatment of acute cystitis is the same as that for pyelitis (*q.v.*) and the response is usually quick.

Chronic cystitis is not a disease entity but comprises an assortment of widely differing conditions which may be divided into two main classes, primary or specific and secondary or non-specific.

**Primary or Specific Chronic Cystitis.** (i) *Tuberculosis* (Chap. 32).

(ii) *Schistosomiasis*.—This condition is very rarely seen in Great Britain and then only in visitors from countries where it is endemic; its effects on the urinary system are remarkable and characteristic. The condition is due to infestation with a small trematode or fluke, *Schistosoma hæmatobium* (*Bilharzia hæmatobia*), which inhabits the smallest tributaries of the pelvic veins, particularly those at the base of the bladder. A description of its complicated, fantastic life history can be found in any work on tropical diseases. The female fluke deposits ova in the submucous tissue of the bladder where they cause an intense and characteristic inflammatory reaction and are from time to time discharged into the urine in large numbers providing an excellent opportunity for diagnosis, for the ovum with its terminal spine is easily recognised without staining under the  $\frac{1}{6}$  inch objective. Cystoscopy also provides a very good means of diagnosis, for some of the changes are characteristic. The earliest change







FIG. 209. *Schistosomiasis*. Cystoscopic view of papillomatous lesions in bladder. Note the "sandy patch" above the largest papilloma. The patient was an Iraqi student in London.

is a small area of congestion followed later by the typical bilharzial tubercles. They are smaller than a pin's head and are greyish-yellow, surrounded by a ring of erythema. Concentrations of these granules are known as "sandy patches." Various secondary changes occur, including ulceration and bullous oedema. Secondary infection is common; fibrosis may occur and obstruct the ureters. One of the most remarkable developments is the occurrence of bilharzial papillomata which resemble extremely closely the familiar neoplastic papilloma (Fig. 209). The fact that these bilharzial tumours are indeed neoplastic is strongly supported by the common development of malignant changes. The commonest early symptom of vesical schistosomiasis is slight terminal hæmaturia. Thereafter all the symptoms of chronic cystitis may develop. The standard treatment of schistosomiasis is intravenous injections of tartar emetic (sodium antimony tartrate). The injections are given on alternate days, the initial dose of 30 mg. being increased by 30 mg. a time up to 150 mg., and the course concludes when a total of 1.2 gm. has been given. Local complications such as infection, stone formation, stricture and carcinoma require the appropriate remedies.

(iii) *Hunner's Ulcer*.—This is a fairly uncommon condition occurring chiefly in middle-aged women. It was described by an American (whose name rhymes with runner) and is also called interstitial cystitis. Its cause is unknown and the urine is characteristically clear (without pus) and sterile. The disease attacks sharply limited areas of the bladder, usually in the vault, where it produces a linear or branching ulcer and fibrosis of the underlying muscle. There is very painful, frequent micturition continuing sometimes for months at a time. The cystoscopic picture is typical. A linear ulcer is seen which, as the bladder fills, seems to split; a fine ooze of blood then issues from it. No effective treatment is known and consequently a multitude of measures have been tried. Fulguration by *per urethral* diathermy often brings some relief. In severe and resistant cases, diversion of the urine may be justifiable. Hopes that cortisone would prove effective have not been realised.

(iv) *Abacterial Pyuria*.—The aetiology of this condition is not known, but there is some evidence that it is related to non-specific urethritis, for the bladder symptoms are sometimes preceded by a urethral discharge in which no organisms are found. The dominant symptom is frequency of micturition, sometimes with strangury and pain. The findings, both clinical and laboratory, are negative except for pyuria, and the diagnosis is made chiefly by exclusion. The cystoscopic picture is variable and non-specific, but there is clearly cystitis present and there may be ulceration. The most important differential diagnosis is tuberculosis. The treatment is empirical and many cases respond to the organic arsenicals. *Novarsenobillon* (neoarsphenamine, B.P.) 0.5 gm. intravenously weekly for three or four weeks is a standard course. Sometimes Aureomycin is effective.

**Secondary or Non-specific Chronic Cystitis.** Chronic infection of the

bladder, or the failure of acute cystitis to clear completely, is almost always due to the presence of some other underlying disease of the urinary tract or an adjacent organ. The symptoms are sometimes insignificant, but there is usually some frequency of micturition and there may be painful micturition. The urine often contains pus, visible to the naked eye, and on culture a variety of organisms will be found, most frequently *Bact. coli*, but also quite often *Proteus* and *Pseudomonas pyocyanea*. Such a condition requires full investigation of the urinary tract and particularly cystoscopy and urethroscopy. The underlying cause may be one of a great variety of conditions; common examples are chronic prostatitis, carcinoma of the bladder, obstruction at the bladder neck, diverticulum of the bladder, vesicocolic fistula. There is, of course, no specific treatment of the cystitis itself; it is the causal condition which demands treatment.

**Cystitis with Metaplasia.** It is common to find on cystoscopy for recurrent urinary infection, particularly in women, an irregular surface of the mucosa, especially near the bladder neck, somewhat resembling cobblestones. In some cases, these cobbles project sharply from the mucosal surface and have a yellowish opaque appearance. This is cystitis cystica. The cysts are caused by down growths of epithelium in which central cystic changes develop. More advanced changes are sometimes found in which there is tissue of glandular type. In themselves these conditions have no importance, but very rarely they may be the site of development of adenocarcinoma in the bladder.

---

**Vesical Calculus.** (i) *Endemic lithiasis* still exists in some parts of the world (e.g. the Middle East, North-Western India, Southern China) but died out in England 100 years ago; it affects predominantly the lower urinary tract. An important factor in its ætiology is lack of vitamin A and another is probably the mineral content of the drinking water. Apart from complications caused by the stones, the bladder in this type of lithiasis is normal.

(ii) *Sporadic Vesical Calculus.*—Except in endemic areas, stones are found in the bladder far less frequently than in the kidney or ureter, and they are almost always secondary to some other disease of the bladder, usually some condition which prevents its normal emptying. Two other important factors are infection and foreign bodies. The composition of vesical stones is similar to that of renal stones but their shape is different. Usually they are flattened oval or roughly spherical. Sometimes the picturesque jackstone is found. A stone fixed in a diverticulum or in the bladder neck is often moulded to the shape of the retaining cavity.

The symptoms of stone in the bladder are very variable and include painful frequency, strangury, hæmaturia, and difficulty in micturition if the stone engages in the bladder neck. All the symptoms are worse when

the patient is up and about and are much aggravated by jolting and vibration. In addition, there may be the symptoms of the underlying condition and sometimes these predominate. Usually there are no physical signs. The diagnosis is made by X-ray examination (the stones are very seldom radiotranslucent) and confirmed by cystoscopy which also reveals the underlying condition, e.g. prostatic enlargement (Fig. 210). It is quite common for vesical stones to be found incidentally during the investigation or treatment of the causal condition.

The treatment of bladder stones is to remove them and to correct the



FIG. 210. *Vesical Calculus.*  
Plain X-ray. Note position  
of shadow in centre of pelvic  
circle. The laminated struc-  
ture of the stone can be seen.

primary disorder whenever there is one. The stones are removed preferably by *litholapaxy* (i.e. crushing them and evacuating the fragments *per urethram*), but there are some well-recognised contraindications to this method (Fig. 211). Thus if the treatment of the primary condition necessitates opening the bladder (e.g. transvesical prostatectomy) there is no point in crushing the stones, for they can be picked out of the bladder when it is opened. A stone may be too large or too hard to be crushed, or it may be so small that it can be evacuated without crushing it. Some stones (usually in infected urine) are so soft that they cannot be fragmented by the lithotrite but are merely squashed and cannot therefore be washed out of the bladder. A stone partly in a diverticulum should not be crushed owing to the danger of damaging the bladder wall. A stone which has formed round a foreign body is often more safely removed by lithotomy (i.e. by opening the bladder). Any condition of the urethra or

bladder neck which prevents the passage and free manipulation of the lithotrite also necessitates lithotomy. Nowadays lithotomy is always done by the suprapubic route, but the historic route is the perineal, hence the term "*lithotomy position*."

**Foreign Bodies.** Foreign bodies inserted into the urethra, either accidentally or deliberately, may find their way into the bladder. A foreign body may be left in the bladder during a surgical operation; the commonest example is a stitch of unabsorbable material. For this reason stitches involving the lumen of the urinary tract should be of

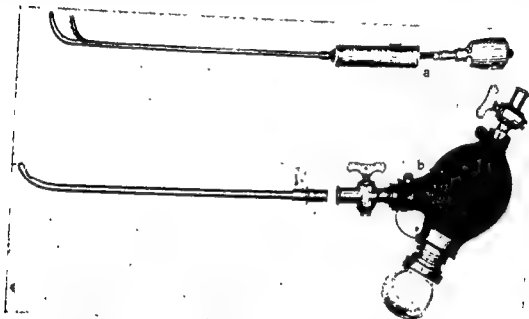


FIG. 211. Instruments for Litholapaxy. (a) Lithotrite. The serrated jaws are shown slightly open. They are closed by screwing the handle of the lithotrite clockwise. (b) Canula and evacuator.

plain catgut. The effect of a foreign body in the bladder is usually to cause infection of the urine often with alkalinising organisms, and in due course the foreign body becomes coated with phosphatic concretion. The symptoms are those of cystitis or stone, but a straightforward history may be difficult to obtain as the patient may be tempted to suppress the facts. When the foreign body is coated with phosphate or is itself radio-opaque it shows in the X-rays. Calculi of unusual shape or density should be suspected of containing a foreign body. Cystoscopy completes the diagnosis, but care must be taken to include the anterior part of the bladder in the search, for the foreign body may be lighter than urine (e.g. wax) and so float. Foreign bodies should be removed *per urethram* whenever it is safe and feasible, but in all doubtful cases cystotomy is preferable.

**Fistula.** Fistula of the bladder may arise as a result of inflammatory or malignant disease, or it may be due to trauma (operative or obstetric).

Overdosage in radiotherapy may cause necrosis with resultant fistula formation. A special case is the deliberate suprapubic cystostomy which does not close when intended. Inflammatory causes of a fistula include diverticulitis of the colon, tuberculosis of the bladder and Crohn's disease (Chap. 8). In these cases the fistula is between the bladder and the intestine. Carcinoma of the colon, bladder or uterine cervix may all cause a fistula, and the bladder then communicates with the vagina, colon, or more rarely the rectum. Hysterectomy, colporrhaphy and obstetric trauma may cause injury to the bladder with a resultant vesicovaginal fistula.

The symptom of a vesicovaginal fistula is a continuous urinary leak. There may in addition be bouts of urinary infection, and the decomposition of the spilled urine causes the patient to stink. A vesicocolic fistula on the other hand may cause surprisingly little disturbance to the patient. There may or may not be symptoms of cystitis but the urine is usually turbid; it may contain gross debris and on microscopy and culture is shown to be infected. The most characteristic symptom is *pneumaturia* which is pathognomonic of the condition, and the patient's description is usually unmistakable. Less frequently the traffic through a vesicocolic fistula is in the opposite direction; patients notice a diminution in the amount of urine passed *per urethram* and are aware of the discharge of urine *per rectum*. There may be attacks of pyelitis and sometimes epididymitis. The diagnosis is confirmed by cystoscopy. A fistula into the bowel is likely to have a marked inflammatory reaction around it with bullous oedema, although the track itself may not be visible: it is usually situated in the upper and posterior part of the bladder.

A fistula of the bladder is closed by operation whenever possible and any associated pathology dealt with *secundum artem* (e.g. resection of the colon for diverticulitis). Fistulae into the bowel are comparatively easy to cure, but vesicovaginal fistulae are notoriously liable to recur and require very free mobilisation, careful suture and prolonged, uninterrupted post-operative drainage of the bladder. The usual approach is abdominal and through the bladder. An inoperable vesicovaginal fistula, particularly when it is due to malignant disease, may justify diversion of the urine (e.g. by ureterocolic anastomosis). An inoperable vesicocolic fistula would not justify colostomy unless the symptoms were unusually severe.

In most cases a temporary artificial suprapubic fistula of the bladder (cystostomy) heals quickly when the tube is removed and normal micturition allowed to take place. Failure of healing may be due to a variety of causes, but the first one to consider is obstruction to the normal outflow. If there is no obstruction there the condition of the fistula is likely to be responsible. In a fistula of long standing an absolute bar to healing could be a complete lining of epithelium. In other cases gross fibrosis round the fistula prevents its closure by the natural process of contraction. Rigidity of the fistula is even greater if the cystostomy has been made too low so that it becomes adherent to the symphysis pubis. In all these

cases the fistula must be completely excised; the bladder is carefully closed and drained through a urethral catheter for five to ten days. The healing of a fistula is always delayed by infection, by the presence of debris and concretions, or by the poor general condition of the patient.

**Diverticulum of the Bladder.** A diverticulum of the bladder is a herniation of the mucous membrane through the muscular coat. It is unable to contract and so cannot empty itself. In most cases the cause is clearly some form of obstruction to the normal urinary outflow; the increased pressure in the bladder forces the mucous membrane out between the interlacing strands of bladder muscle. There is usually associated trabeculation. Although senile enlargement of the prostate is one of the causes of diverticulation, the obstruction in most cases is due to other forms of bladder neck disease. Diverticula are often multiple, but usually one is much larger than the others. The common site is just lateral to one of the ureteric orifices.

The effect of a large diverticulum is to cripple the bladder's action, making it like a syringe with a leaking piston, for when the bladder contracts urine flows not only out through the bladder neck but also into the easily distensible diverticulum. This results in stagnant residual urine, infection and sometimes stone formation. Complete retention of urine may occur when the diverticulum burrows under the trigone, for when the bladder contracts it distends the diverticulum which then thrusts the bladder neck upwards and forwards and increases the obstruction. Occasionally a carcinoma develops in a diverticulum. This is a dangerous complication which presents a difficult surgical problem.

There are no specific symptoms of a diverticulum. The classical symptom of the passage of urine in two lots, one clear and then one purulent, is seldom seen. The symptoms are in general those of bladder neck obstruction and chronic cystitis. Sometimes a distended diverticulum can be felt on abdominal examination, but usually it is too lax and is masked by the hypertrophied and imperfectly emptying bladder. The diagnosis is made by radiography and cystoscopy. The bladder film in an excretion pyelogram often reveals the diverticulum, but much more precise information can be got from a cystogram in which the bladder is drained by catheter and then filled with iodide solution. Oblique views are taken so as to show the neck of the diverticulum in profile. A final picture is also taken after the catheter has been allowed to drain to show the amount of residual fluid in the diverticulum. The smooth outline of the passive diverticulum contrasts strongly with the crenated outline of the muscular bladder (Figs. 212 and 213).

In the treatment of diverticulum the first consideration is to remove the cause and this usually means some form of prostatectomy or bladder neck resection, but before this is done a decision must be made whether the diverticulum itself needs removal, for it is best to deal with the diverticulum at the same time as the obstructing lesion if this requires an

abdominal approach. The decision whether or not to remove the diverticulum depends mainly on its size. If its diameter in the cystogram is more than 2 inches it probably needs to be removed. In doubtful cases a satisfactory plan is to treat the obstruction by perurethral resection and then to review the situation in three or six months. If the bladder empties satisfactorily and the urine is free from infection nothing further need be



FIG. 212. *Diverticulum of Bladder. Cystogram. Anteroposterior view.* The smooth outline of the diverticulum contrasts strongly with that of the bladder which is irregular because of trabeculation and the protrusion of saecules.

done. The operation of diverticulectomy may be done through the opened bladder, extravasically or by a combination of these two approaches. The operation is sometimes difficult and the ureter is always in danger so that it is usually best to catheterise it first.

**Tumours of the Bladder.** Bladder tumours are extremely common. They are usually composed of transitional cells (with which the bladder is lined), but they are often anaplastic and pleomorphic when the tumour is malignant. A small minority of vesical tumours are squamous or even glandular, and there are also very rare connective tissue tumours, both benign and malignant.



Although bladder tumours may be classified on cystoscopic and histological appearance into benign and malignant groups, this distinction must not be given too great a significance, and many surgeons prefer simply to speak of "bladder tumours." The reason for this is that even histologically there are many border-line tumours whose designation as

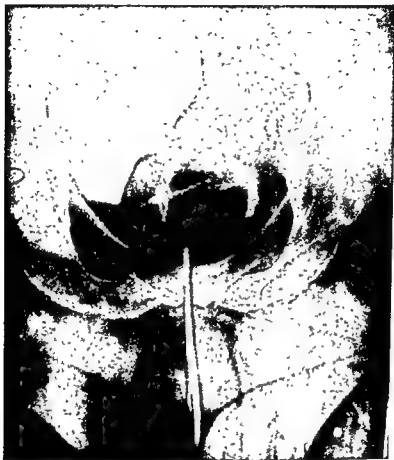


FIG. 213. *Diverticulum of Bladder. Cystogram.* There is a large diverticulum on the right side of the bladder (reader's left). The bladder is mostly occupied by the round bag of a Foley catheter. The great thickness of the hypertrophied bladder wall is represented by the space separating the outline of the diverticulum from that of the bladder. Note that the contrast medium, although injected into the bladder, has flowed straight into the diverticulum; this is because the thick-walled bladder resists distension.

benign or malignant is doubtful or whose histology varies in different parts of the tumour. More important is the biological behaviour of bladder tumours. For reasons which are not understood, even tumours which are histologically benign may "recur"; that is to say a fresh tumour may appear in the bladder from which such a "benign" tumour has been removed. This is common, and the subsequent tumours in such cases often become histologically progressively less benign as they "recur." There is not yet sufficient evidence to decide whether these "recurrences" are the result of implantation of cells of the original tumour, whether they

are due to multicentric neoplastic activity, or whether they are caused by the continued action of a carcinogenic agent in the urine. It is of course known that certain compounds (e.g.  $\beta$ -naphthylamine) if excreted in the urine will produce transitional-cell tumours in the human bladder. Workers with aniline dyes are subject to this risk and develop tumours much more often than others.

The most benign form of bladder tumour, the papilloma, grows on a fine stalk and has delicate fronds which float gracefully in the bladder



FIG. 214. *Papilloma of Bladder.* Drawing of cystoscopic view of typical stalked and fronded growth.

fluid (Fig. 214). It has a good blood supply and looks characteristically pink against the somewhat yellow bladder mucosa. It is very soft and friable. The most malignant form of bladder tumour is the irregular excavating ulcer with an indurated base, clearly malignant both macroscopically and microscopically. In between these two extremes there is a gradation of tumours, some with short coarse fronds, some with thick stalks, some widely sessile and bald (Fig. 215). They may be single or multiple and are most often found near the ureteric orifices. The frankly malignant tumours may infiltrate and obstruct the ureter or involve the bladder neck and so cause retention of urine. They spread through the whole thickness of the bladder wall and infiltrate the paravesical fatty tissue and invade the regional lymph glands, or in some cases they produce more distant dissemination in lymph glands, bones, liver or

lungs. Ulceration and obstruction frequently lead to infection and sometimes stone formation. Above all there is hæmorrhage.

The pathological classification of bladder tumours is made partly on their histological appearance and partly on their extent of spread. Four

stages have been suggested by Dukes and Masina: (1) Tumours confined to the bladder wall (subdivided into (1a) tumours confined to the mucosa and (1b) tumours involving the bladder muscle); (2) tumours involving the paravesical tissue but not yet invading the lymph glands; (3) tumours invading the lymph glands adjacent to the bladder; (4) metastatic lesions which cannot be removed in continuity with the tumour. Although tumours cannot be accurately fitted into this scheme except by examination of specimens removed by radical operation, a preoperative classification should be attempted as it is a sound basis for assessment of operability and prognosis.

Hæmaturia alone is the presenting symptom of bladder tumours in three cases out of four, and occurs with other symptoms in a much higher proportion. The bleeding is characteristically profuse and involves the whole stream. It appears

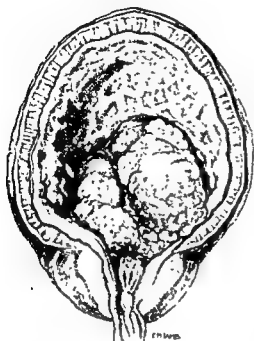


FIG. 215. *Carcinoma of Bladder.* Large papilliferous growth partly obstructing bladder neck. Note trabeculation. The lower picture shows how the growth has infiltrated through the muscle of the bladder wall (stage 2 of the Dukes and Masina classification). Cystectomy specimen.

without any warning, recurs perhaps on two or three successive urinations and may then disappear completely for weeks, months, or even years. In some cases, usually with frankly malignant tumours, there are other symptoms, often those of cystitis, or obstruction of the bladder neck. The absence of these additional symptoms does not at all exclude a malignant, even inoperable tumour. There are usually no abnormal physical signs even with malignant tumours. However, if a tumour

can be felt, either abdominally or *per rectum*, it is almost certainly malignant.

The diagnosis is made by *cystoscopy*. The situation, number and characteristics of the tumours are noted and a provisional diagnosis made as to their innocence or malignancy. *Biopsy*, which is usually helpful and sometimes decisive, should be done whenever possible, but it is important to obtain sufficient tissue and to take specimens from various parts of the tumour, particularly from its base if this is feasible. Finally a thorough *bimanual palpation* under full muscular relaxation is essential for the assessment of a tumour, as it gives information about infiltration and fixity outside the bladder obtainable in no other way. An excretion pyelogram should be done to exclude associated tumours of the renal pelvis or ureter, and also to reveal any evidence of ureteric obstruction which always implies malignant infiltration.

In the differential diagnosis the only common problem is to distinguish localised inflammatory lesions from neoplasms. In most cases, experience in cystoscopic interpretation enables this to be done, and biopsy finally settles the matter. Sometimes a local inflammatory mass very closely simulates a tumour, e.g. the patch of induration and œdema, sometimes with ulceration, caused by the pressure of the tip of an indwelling catheter. Chronic cystitis with metaplasia may sometimes present a puzzling picture on cystoscopy.

*Treatment.* The various forms of treatment available are perurethral fulguration, perurethral resection, suprapubic (open) diathermy, partial cystectomy (segmental resection), total cystectomy, radiotherapy, and palliative measures. For small tumours obviously confined to the mucosa, and especially the typical villous papilloma, perurethral fulguration is very successful and is a simple method requiring only a catheterising cystoscope and diathermy equipment. For larger (10 gm. or more), but still benign tumours, fulguration can also be used but is tedious. For these tumours perurethral resection is very satisfactory. For all benign tumours too large to be treated *per urethram*, suprapubic cystostomy with diathermy resection is used. With this method precautions must be taken against implanting tumour cells in the wound (chiefly by protecting the exposed surfaces with towels). Rarely a very extensive tumour, even though it is benign in the sense of being confined to the mucous membrane, justifies total cystectomy.

The results of treating infiltrating tumours are poor, although published results are difficult to compare owing to lack of uniformity in the criteria of malignancy, operability and degree of spread. A 21 per cent. five-year survival rate for infiltrating tumours was recorded in 1950 by the Christie Hospital, Manchester. The method used was open diathermy and the implantation of radon seeds. Results obtained by other methods are similar. Total cystectomy has the great advantage of removing the whole of the bladder intact so that no local recurrence is possible and no

implantation of tumour cells can be made in the wound. It has, however, the enormous disadvantage of requiring diversion of the urine, a procedure which carries a grave danger of causing severe renal damage. Nevertheless, total cystectomy is a good method of treatment for extensive tumours with only superficial infiltration. Partial cystectomy gives fairly satisfactory results for infiltrating tumours in the upper half of the bladder lying well clear of the ureteric orifices and internal meatus. It has the great advantage over total cystectomy of leaving a bladder which functions normally for the needs of the kidney and almost normally for the convenience of the patient. If it is necessary to remove a part of the bladder including a ureteric orifice, partial cystectomy may still be done but entails reimplantation of the divided ureter into the remains of the bladder. The scope of both partial and total cystectomy may be extended by including a dissection of the iliac lymph nodes. This increases the severity of the operation, and it is not generally agreed that there is a compensating improvement in the results obtained. Perurethral diathermy resection is used successfully for tumours which do not infiltrate the full thickness of the bladder wall and is particularly suited for frail subjects. Its main disadvantage is that considerable experience of the technique is required if the resection is to be both safe and thorough.

*Radiotherapy* may take one of three main forms : deep X-rays ; direct implantation into the tumour of radium or some similar source of radioactivity ; intracavitary radiation. Intracavitary radiation may be applied in two ways. The bladder may be filled with a solution of a radioactive isotope either directly or contained in a thin rubber bag which is inserted (in the male) through a perineal urethrostomy. The particular limitation of this method is that it achieves a shallow depth of penetration and so can only be used for tumours of superficial infiltration. The other method is to use a point source of radiation held at the centre of the distended bladder. By this means adequate penetration can be achieved, but the great disadvantage is that the slightest displacement of the source from the centre of the bladder dangerously increases the dose of radiation in one part of the bladder and leaves another part of the bladder without adequate treatment. Intracavitary radiation has one special theoretical advantage which has also been the cause of a serious complication. It irradiates the whole of the bladder mucosa and so destroys the whole of an overt carcinoma, no matter how extensive, and may possibly also be expected to inhibit the development of tumours in other foci of precarcinomatous activity which were destined to become "recurrences." This very advantage, total irradiation, carries with it the danger of widespread fibrosis and consequent bladder contracture. For these reasons among others, intracavitary radiation is not used much at present.

Direct implantation of a radioactive source is the commonest form of radiotherapy applied to those bladder tumours which are considered curable. It is extremely important that the dose and its distribution

should be correct. In the past, radium has often given poor results. Severe intractable cystitis due to overdosage, and recurrence of the tumour might coexist in the same patient. It is difficult to say which of these two disasters causes the greater suffering. Improvement of the results of interstitial radiation has been due partly to close collaboration between surgeon and radiotherapist or physicist. The correct dose and its distribution are worked out from the size and shape of the tumour, and the distribution of the needles or seeds is checked after operation by X-ray so that the time of removal can be altered if necessary. In interstitial radiation the source is usually applied through the opened bladder after cutting away the projecting part of the tumour with diathermy. Radium needles have the disadvantage of requiring to be removed when the dose has been delivered, and this entails suprapubic drainage. A modern variant is radioactive tantalum wire. Although this also needs to be removed, its suppleness enables it to be removed through the urethra. Its suppleness also allows it to be laid along the curve of the bladder wall and the contour of the tumour. Radon seeds have the advantage of not requiring removal, but they are foreign bodies which, although they are no longer radioactive after ten days, may cause enough reaction to confuse the interpretation of the cystoscopic findings in the follow-up period; furthermore they may cause stone formation.

In the past, deep X-ray therapy has usually given poor results, but new supervoltage machines, accurate localisation of the tumour, and rotation techniques have done much to increase the effectiveness of the tumour dose and have reduced the harmful effects on adjacent organs and the skin. The method is still mainly used to relieve symptoms in inoperable cases, but recent results obtained in some centres suggest that deep X-ray therapy may become an alternative to surgery and interstitial radiation as a method of cure.

Palliative treatment is often necessary in bladder tumours, for advanced cases are common and the patients are often old and frail. Apart from X-ray therapy, the measures available include the control of urinary infection (cystitis always aggravates the symptoms), and, when the symptoms are severe, diversion of the urine (e.g. by ureterocolic anastomosis). It is very seldom justifiable to remove the bladder merely as a palliative measure.

An essential part of the management of bladder tumours is periodic post-operative cystoscopy. At first this should be done three-monthly, then six-monthly, and finally yearly for the rest of the patient's life. The more benign the original tumour appeared to be, the more justifiable is this periodic cystoscopy, for fresh tumours are commonly found; they are easily destroyed by cystodiathermy, as an out-patient procedure when the tumours are small, and as these "recurrences" are usually symptomless in the early stages, routine cystoscopy is the only means of nipping them in the bud.

**Suprapubic Cystostomy.** Local anaesthesia may be used, but unless there is some contraindication a general anaesthetic is more convenient. The operation is easier and quicker with the bladder full, particularly in obese patients. Therefore when it is not done for retention of urine it is usual to fill the bladder by catheter. Enough sterile water should be run in to make the bladder easily palpable, preferably halfway up to the umbilicus. It is important that the drainage track should be well clear of the symphysis pubis and the incision should be centered midway between the symphysis and the umbilicus. If the track is made too low it may become adherent to the symphysis pubis and it causes difficulty in the wearing of a suprapubic urinal. A vertical incision is quick and simple to make, but may be followed by an incisional hernia if it is long or becomes infected. A 2-inch incision is adequate for most cases. It is carried down through the aponeurotic layer exactly in the midline and the rectus muscles are separated. This exposes the transversalis fascia which is incised vertically. The wound edges are retracted and a finger inserted downwards immediately deep to the fascia, pressed backwards and drawn up, stripping before it the extraperitoneal fat and peritoneum to lay bare the bladder which is recognised by the fine vertical fibres of the detrusor muscle and the perivesical veins on its surface. The level of the peritoneal sinus (reflexion) is extremely variable and it is important to identify the bladder wall with certainty before proceeding further. A stay suture is placed in the bladder each side of the midline. A self-retaining catheter is selected (e.g. a de Pezzer catheter) and stretched on an introducer so as to flatten the projecting head. A very small stab is now made through the bladder wall, and before the blade is withdrawn the catheter on the introducer is slipped into the bladder. If when the introducer is withdrawn there is any leakage around the catheter a catgut stitch is introduced to draw the bladder together round the catheter. The aponeurotic layer is now sutured with a few interrupted chromic catgut stitches so that the catheter emerges through the upper end of it, making an oblique track. When the skin has been sutured, the catheter is retained only by its self-retaining head but this must never be relied on exclusively. Therefore a stitch in the skin is tied snugly round the catheter, but not tightly enough to obstruct it. A small dressing is placed on the wound and then, after the surrounding skin and the catheter have been dried with methylated ether, the catheter is fixed with a piece of narrow strapping to the abdominal skin. It is then connected by means of a sterile rubber tube to a sterile bottle fixed to the side of the bed. It is important to ensure that this tubing is tethered so that it does not drag on the catheter.

**Catheterisation.** When a catheter is passed for the relief of retention of urine it is usually left in to allow continuous drainage until the cause of the retention is removed. In these circumstances, a self-retaining catheter is the most convenient; the *Foley* catheter is the best (Fig. 216). For

the drainage of clear urine it has no disadvantages (except expense) provided it can be got into the bladder. A convenient size is 18 or 20 French and the bag should be filled with 20 ml. sterile water. If a Foley catheter cannot be got into the bladder and provided the cause of the retention is not a urethral stricture, the next catheter to try is a *Tiemann*, preferably made of plastic, of about the same size. It needs to be retained by external fixation. The great majority of cases of retention of urine can be relieved by one or other of these catheters. In some patients with large prostates when these two types of catheter fail, a plastic (or, failing

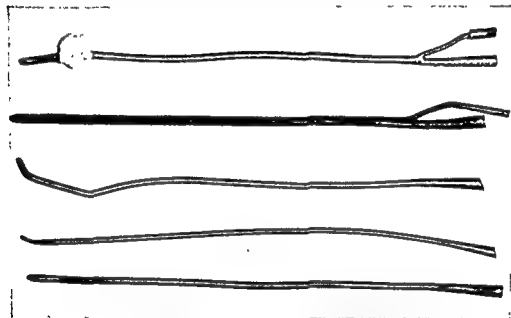


FIG. 216. *Catheters.* The top two are Foley catheters, one of them with the self-retaining bag inflated. The lower three are plastic catheters: from above downwards bi-coudé (French style), Tiemann and whistle-tip. All these catheters may be sterilised by heat.

that, gum elastic) bicoudé catheter may succeed. In strictures and in some cases of obstinate spasm of the external sphincter a fine olivary or Tiemann catheter should be tried.

The avoidance of infection in the relief of retention of urine is so important that every aseptic precaution must be taken. The pubes must be shaved, the penis cleaned with sterile Cetavlon and sterile towels used. Normally no anæsthetic is needed, but in very nervous patients the instillation into the urethra of Xylocaine gel is helpful. No part of the catheter should be touched by hand. In many cases it can be successfully handled through a sterile swab or with forceps, but when manipulation is difficult sterile gloves should be worn.

Little manipulation is possible with a soft catheter such as the Foley,



but with a curved and slightly rigid catheter such as the Tiemann a lot depends on the way it is passed. The penis is held stretched forwards and the tip of the catheter is introduced into the fossa navicularis with its curve directed towards the under surface of the penis. In this position it is passed down into the urethral bulb and is then turned through 180 degrees so that the curved tip is directed forwards and upwards; at the same time the penis, still on the stretch, is pulled downwards and all the time the catheter is steadily advanced so long as it goes easily. Resistance by spasm may often be overcome by steady gentle pressure exerted against it. When the catheter is in the bladder urine flows. Ten ounces are allowed to drain, of which some is sent for microscopy and culture; the catheter is then spigotted or clipped until it is connected by sterile tubing to a sterile bottle. Thereafter 300 ml. are allowed to flow every two hours until the bladder is empty and free drainage can be allowed. Although gradual decompression is unnecessary in acute retention, it is probably safer in chronic retention so that it is an advantage to use it as a routine.

When the urine is thick with blood or pus a wide internal lumen of the catheter is necessary. In these circumstances a Foley catheter is not ideal but it is still often used owing to its other advantages. It should be 24 or 26 French if the urethra will accommodate it. Perhaps the best catheter of all for the drainage of blood is the whistle-tip catheter which, when made of plastic, provides the maximum lumen and orifice for a given outside diameter. This type of drainage problem applies almost exclusively to post-operative cases.

By far the most reliable method for the sterilisation of catheters is autoclaving or boiling, and nowadays catheters of almost all types are obtainable which withstand this treatment. Catheters made of non-boilable material, such as gum elastic, may be sterilised by immersion in a 0.25 per cent. solution of benzalkonium chloride (syn. Zephiran, Roccal) for half an hour.

## CHAPTER 20

### DISTURBANCES OF MICTURITION AND DISEASES INVOLVING THE BLADDER NECK

THE bladder is the problem child of the urinary family. It frequently complains and misbehaves, but its inner workings are obscure. The fundamental difficulty is that the physiology of micturition is still imperfectly understood, although some aspects are fairly clear. The bladder is a bag of smooth muscle connected with the central nervous system. If this connection with the nervous system is completely severed, the bladder is still able to contract but not efficiently, for the nervous system controls and co-ordinates its activity. Although the bladder is innervated by both the sympathetic and parasympathetic components of the autonomic system, the sympathetic plays an unimportant and inessential part; the effective control comes through the sacral segments of the spinal cord and the pelvic nerves, and in their turn the spinal centres are under the control of the brain. The outlet from the bladder, the internal urinary meatus, is surrounded by a ring of plain muscle often called the internal sphincter, but the term is misleading; *bladder neck* is a preferable term, for continence does not depend on it. It can be freely resected without disturbance to the control of micturition. However, failure of the bladder neck to open normally certainly impedes the outflow of urine and may even cause complete retention. The *external sphincter* which surrounds the urethra as it passes through the layers of the *triangular ligament* (urogenital diaphragm) plays a most important part and certainly earns the title of sphincter. It is composed of striped muscle. If its action is weakened by direct injury or damage to its nerve supply incontinence follows.

One of the simplest ways of studying the working of the bladder is by *cystometrography*. This method gives information about the manner of contraction of the bladder as a whole but not about the mechanism of the bladder neck or the co-ordination between the bladder neck and the rest of the bladder. The bladder is emptied by a catheter which is then connected to a raised reservoir and a manometer (Fig. 217). Fluid from the reservoir is allowed to flow slowly and steadily into the bladder, and the intravesical pressure is plotted against successive increments of volume. The normal bladder, by a progressive yielding of its muscle wall, accommodates more and more fluid while maintaining an almost level (but slightly rising) pressure. This continues until normal bladder capacity is almost reached when there is a desire to void; the muscle no longer yields *pari passu* with the increasing volume and the pressure

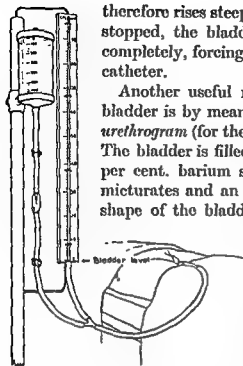


FIG. 217. Apparatus for Cystometry.

therefore rises steeply (Fig. 218). Finally, if the inflow is not stopped, the bladder contracts strongly and empties itself completely, forcing fluid out of the manometer or round the catheter.

Another useful method of studying the working of the bladder is by means of the *micturating cystogram* or *cystourethrogram* (for the urethra is usually included in the study). The bladder is filled by catheter with a radiopaque fluid (2 per cent. barium sulphate suspension). The patient then micturates and an X-ray is taken during the process. The shape of the bladder, particularly of the bladder neck and

upper part of the urethra, are then recorded. This investigation is sometimes helpful but the interpretation of the films is often difficult and controversial. A very promising development of the method is to use *cine-radiography* which is now possible following the invention of the image intensifier. This living record of the act of micturition is likely to contribute much

to the study of this difficult subject.

**Frequency of Micturition.** Many patients suffering from frequent micturition due to a variety of causes show a curve in the cystometrogram similar to the normal but shorter and steeper. In some patients evidence of any disease can be found. In young children who have attained full urinary control the curve is also short and steep, but in addition shows sudden pressure peaks of momentary duration. These contractions are due to reflexes through the spinal cord, and as the c

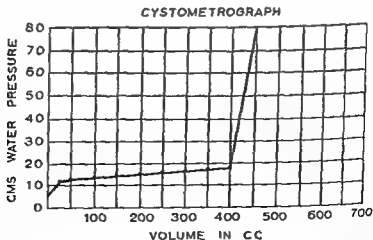


FIG. 218. Normal Cystometrogram. The pressure rises very gradually until normal maximum capacity the bladder contracts and forces up the pressure.

development proceeds they are inhibited by the higher centres of the brain. In some adults these neurogenic contractions survive or reappear due to failure of the normal inhibition of the spinal reflexes (Fig. 219). These patients suffer from frequency and urgency of micturition and even incontinence of urine, but no disease of the urinary tract can be demonstrated. Such uninhibited contractions may sometimes be abolished by ganglion-blocking agents like propantheline (syn. Probanthine : adult dose 15 to 45 mg.).

In clinical practice frequency of micturition must always be assumed to be due to organic disease of the urinary tract, usually inflammatory ; cystometrography has no place until these possibilities have been

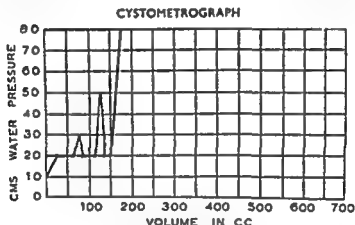


FIG. 219. *Cystometrograph of Uninhibited Neurogenic Bladder.* The curve is shorter and steeper than normal and is interrupted by peaks due to involuntary premature contractions.

excluded. Frequency in inflammatory diseases is due to increased sensory stimuli from the bladder causing reflex contraction. Frequency of micturition is often due to psychological causes. In such cases the symptoms may be strikingly variable and evidently affected by emotional stimuli, but the bladder capacity may be proved to be normal by the patient's ability to sleep through the night without voiding. In other cases the symptoms may be less characteristic and a full investigation may be necessary in order to exclude organic disease.

**Neurogenic Bladder Dysfunction.** Normal bladder function requires cerebral co-ordination and the control of the reflex centre in the sacral segments of the spinal cord (Fig. 220). Disorder of this higher controlling mechanism may result in incontinence (the uninhibited neurogenic bladder) or retention. Severance of the connection between the brain and the spinal centre of micturition results in the *reflex* or *automatic bladder* (spinal bladder) (Fig. 221). Typical causes are injury or disease of the vertebral column and tumours compressing the spinal cord.

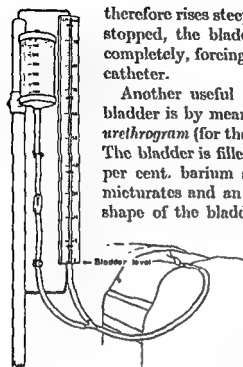


FIG. 217. Apparatus for Cystometry.

therefore rises steeply (Fig. 218). Finally, if the inflow is not stopped, the bladder contracts strongly and empties itself completely, forcing fluid out of the manometer or round the catheter.

Another useful method of studying the working of the bladder is by means of the *micturating cystogram* or *cystourethrogram* (for the urethra is usually included in the study). The bladder is filled by catheter with a radiopaque fluid (25 per cent. barium sulphate suspension). The patient then micturates and an X-ray is taken during the process. The shape of the bladder, particularly of the bladder neck and upper part of the urethra, are thus recorded. This investigation is sometimes helpful but the interpretation of the films is often difficult and controversial. A very promising development of the method is to use *cine-radiography* which is now possible following the invention of the image intensifier. This living record of the act of micturition is likely to contribute much

to the study of this difficult subject.

**Frequency of Micturition.** Many patients suffering from frequency of micturition due to a variety of causes show a curve in the cystometrogram similar to the normal but shorter and steeper. In some patients no evidence of any disease can be found. In young children who have not attained full urinary control the curve is also short and steep, but in addition shows sudden pressure peaks of momentary duration. These contractions are due to reflexes through the spinal cord, and as the child's

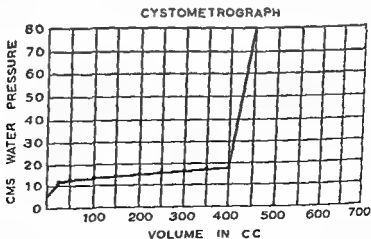


FIG. 218. Normal Cystometrogram. The pressure rises very gradually until at normal maximum capacity the bladder contracts and forces up the pressure.

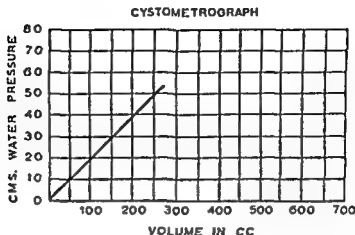


FIG. 222. *Cystometrograph of Autonomous (syn. "Cauda Equina") Bladder.* Pressure rises proportionately to volume until overflow occurs.

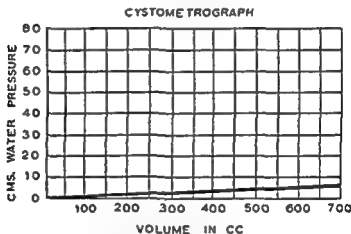


FIG. 223. *Cystometrograph of Sensory Paralytic Bladder.* The bladder is atonic and there is no micturition reflex.

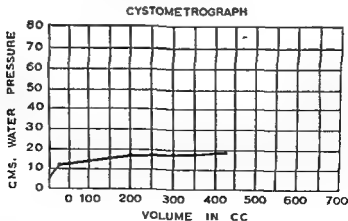


FIG. 224. *Cystometrograph of Motor Paralytic Bladder.* The curve is normal, but does not culminate in the micturition reflex.

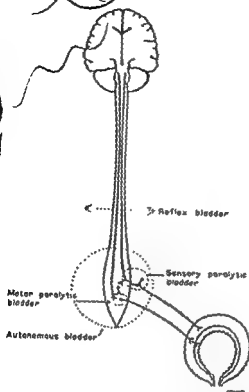


FIG. 220. *Neurogenic Bladder Disease.* Scheme showing sites of lesions in the nervous system producing the four main types of neurogenic bladder disease.

vesical wall is thin and the bladder neck may be patulous, the so-called funnel deformity. There is frequent urination or dribbling incontinence. On the other hand anterior poliomyelitis, which affects the motor neurons only, may produce the *motor paralytic bladder* which is painfully distended and ultimately overflows (Fig. 224).

Micturition is involuntary and bladder sensation may be absent. Bladder capacity is usually small and emptying is incomplete.

Destruction of the sacral centre itself or division of the nerves connecting it with the bladder (in the cauda equina or within the pelvis) produces the *autonomous bladder* (cauda equina bladder) (Fig. 222). In this condition the bladder is devoid of sensation, its walls are hypertrophied and there is hypertonicity of the bladder neck. The intravesical pressure is high, but contractions are inefficient so that there is a large residual urine and voiding is by incontinent overflow.

Sometimes lesions interrupt only one component of the reflex arc. For example tabes dorsalis, by destroying afferent neurons, may produce the *sensory paralytic bladder* which, although painless, is greatly distended (Fig. 223). The finely trabeculated

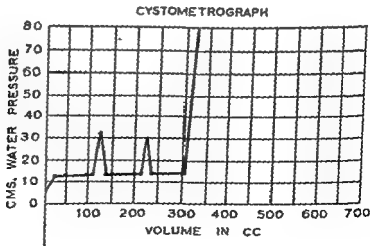


FIG. 221. *Cystometrograph of Reflex (syn Automatic, "Spinal") Bladder.* There are uninhibited contractions, but the pressure and capacity are normal.

cystoscope or urethroscope. A thick bladder neck which stands up like a collar around the internal meatus, particularly behind, is suggestive of obstructive disease. Similarly prostatic enlargement which projects into the bladder is probably obstructive.

Evidence of obstruction by the bladder neck is strongly supported when the bladder muscle is found to be hypertrophied. Hypertrophy of the bladder is manifested cystoscopically by trabeculation ; the trabeculae

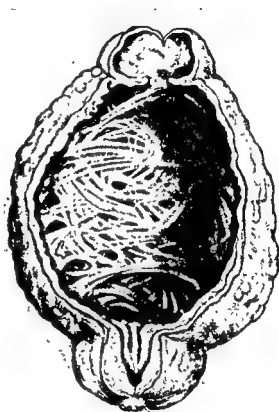


FIG. 226. *Bladder-neck Obstruction.* Trabeculation of bladder. The bladder neck is hypertrophied posteriorly and there is also some enlargement of the prostate. Note the diverticulum on the top of the bladder (an unusual site).

CR. LAMPE

(or beams) are the hypertrophied strands of the muscle mesh standing out from the bladder wall (Fig. 226). The hollows between the trabeculae vary much in depth. The deeper ones are called saccules or cellules, and sometimes these pouches are pushed out through the muscle layer of the bladder wall to form *diverticula*.

A bladder which hypertrophies in response to obstruction at the bladder neck is often able to empty completely in spite of the obstruction. Such a bladder is said to be compensated. More often the bladder is only able to force urine through the resistant bladder neck while its fibres have the advantage of being on the stretch, but as the bladder empties the muscle fibres shorten and work progressively more and more at a dis-



These are some of the main types of neurogenic bladder. Incomplete lesions of the spinal cord variously modify the effect on the bladder and sometimes the picture is complicated by concomitant disease of the bladder neck. The immediate effect of traumatic lesions is usually to produce atony of the bladder so that the typical reflex or autonomous behaviour may not develop for days, weeks or even months. This subject is further discussed under "The Spinal Cord and its Nerve Roots," Chap. 59.

**Obstruction at the Bladder Neck.** The ability of the bladder to empty completely depends on a balance between the contracting power of the

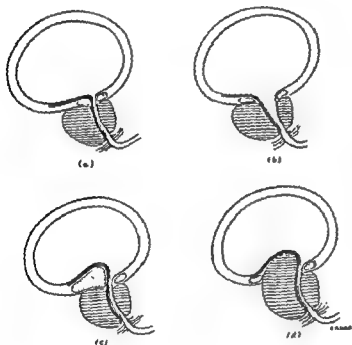


FIG. 225. *The Bladder Neck.* The upper figures show the bladder neck closed (a) and open (b) and illustrate the action of the trigonal muscle (thick black line) which by contracting straightens itself and so draws back the posterior lip of the bladder neck. The lower figures show how hypertrophy of the bladder neck (c) or intravesical projection of the prostate (d) can impede the action of the trigonal muscle.

bladder as a whole and the resistance to the outflow of urine through the bladder neck. Unfortunately cystometrography gives no information about the function of the bladder neck and no other method of study has thrown much light on it. One factor in the mechanism of opening of the bladder neck is thought to be the trigonal muscle which runs down over the posterior lip of the bladder neck into the posterior urethra. When this muscle contracts it draws back and depresses the posterior lip of the bladder neck and so opens the internal meatus (Fig. 225). This theory is not entirely proved, but it helps to explain the obstructive effect of some of the common diseases of the bladder neck. It is easy to understand how increased bulk and rigidity of the bladder neck, whether it is adenomatous as in prostatic enlargement or muscular and fibrous as in Marion's disease, hampers this delicate mechanism. In practice, the function of the bladder neck is assessed on its appearance through the

cystoscope or urethroscope. A thick bladder neck which stands up like a collar around the internal meatus, particularly behind, is suggestive of obstructive disease. Similarly prostatic enlargement which projects into the bladder is probably obstructive.

Evidence of obstruction by the bladder neck is strongly supported when the bladder muscle is found to be hypertrophied. Hypertrophy of the bladder is manifested cystoscopically by trabeculation; the trabeculae



FIG. 226. *Bladder neck Obstruction. Trabeculation of bladder. The bladder neck is hypertrophied posteriorly and there is also some enlargement of the prostate. Note the diverticulum on the top of the bladder (an unusual site).*

(or beams) are the hypertrophied strands of the muscle mesh standing out from the bladder wall (Fig. 226). The hollows between the trabeculae vary much in depth. The deeper ones are called saccules or cellules, and sometimes these pouches are pushed out through the muscle layer of the bladder wall to form *diverticula*.

A bladder which hypertrophies in response to obstruction at the bladder neck is often able to empty completely in spite of the obstruction. Such a bladder is said to be compensated. More often the bladder is only able to force urine through the resistant bladder neck while its fibres have the advantage of being on the stretch, but as the bladder empties the muscle fibres shorten and work progressively more and more at a dis-

advantage until they are no longer able to overcome the resistance at the bladder neck and can expel no more urine. The amount of urine remaining in the bladder after micturition is called the *residual urine* and is a useful indication of what might be called the negative balance between the resistance of the bladder neck and the contracting power of the bladder muscle. In some cases there is so much residual urine that the bladder can be felt above the pubis, a condition traditionally known as *chronic retention of urine* although it differs only in degree from a smaller amount of residual urine.

The balance between the bladder's power and the resistance at the bladder neck may be upset very suddenly by an alteration in the weight on either side of the scales. For example, a mild compensated bladder neck obstruction may result in acute retention of urine due to the onset of slight prostatitis. On the other hand, in old people who become ill and confined to bed, particularly if there is any mental disorientation, the bladder may lose its expulsive force and retention ensues. Prolonged overstretching of the bladder muscle always results in some degree of atony which is an important secondary factor in some types of neurogenic bladder. The common syndrome of post-operative retention is probably due to interference with micturition reflexes by abnormal stimuli from the operation site. It generally follows operations on the genitalia, the lower part of the abdomen generally, and the perineum, including the rectum. The effect usually passes off within twenty-four hours.

**Effects and Complications of Retention of Urine.** Besides hypertrophy of the bladder muscle generally, the interureteric bar in particular sometimes becomes thick so that it stands up along the back of the trigone providing an additional obstructive factor. When uncompensated obstruction leaves stagnant urine, there is danger of urinary infection and stone formation. In fact the incidence of these complications is not high, but the danger is that once infection is introduced it has more serious effects in the presence of retention than otherwise and can hardly be eradicated until the cause of the retention is removed. The effects of urinary retention are much more serious in the kidneys than in the bladder. In chronic uncompensated bladder neck obstruction the competence of the ureteric orifices often fails with the result that the pressure in the bladder, including the peaks at micturition, is transmitted up the ureters to the kidneys and causes hydronephrosis with the usual progressive atrophy of the renal substance and insidious renal failure. If infection occurs in these circumstances it is likely to develop rapidly into a severe suppurative pyelonephritis from which recovery is unlikely, but even without infection fatal uraemia may ensue.

**Treatment of Retention of Urine.** The relief of retention should be distinguished from the removal of its cause. In some cases the two objects are achieved simultaneously, but usually the relief of retention is the sole consideration at first, and the cause is removed (if it is removable)

at a later stage. In general there are two methods of relieving retention of urine : *urethral catheterisation* and *suprapubic cystostomy*. Each has some advantages over the other. A catheter can be passed quickly and easily, and the patient need not leave the bed. No anæsthetic is needed or a simple injection into the urethra suffices. A catheter drains the bladder from below and so ensures complete emptying. The most serious objection to the catheter is that it may cause urinary infection and complications in the urethra. The seriousness of infection added to retention has already been stressed. The catheter may cause it in several ways. Firstly, organisms may actually be introduced when the catheter is passed. Secondly, the mechanical effects of an in-lying catheter may lead to the development of infection. The mere presence of any foreign body in the urethra causes an aseptic urethritis and sooner or later this will almost certainly become infected either by organisms "growing along" from the external meatus or settling from the blood stream at a point of lowered resistance. The catheter blocks the orifices of the urethral and prostatic glands, and by preventing drainage causes the infection to develop and spread. In the female, a catheter is a lesser danger than in the male owing to the absence of the prostate and the shortness of the urethra.

*Suprapubic cystostomy* involves a lesser risk of infection than a urethral catheter, for the field of operation can be sterilised more reliably than the penis and terminal urethra, and the tube lies in a track of relatively inert tissues instead of a narrow tunnel of mucous membrane and glands. It can be fixed rather more securely than a urethral catheter and can be retained indefinitely (with periodic changes) if need be. Moreover, it may be used when the urethra or bladder neck are impassable. Its disadvantages are that it entails an operation, albeit a minor one. An incision in the abdominal wall and an opening in the urinary tract are possible factors in post-operative complications (e.g. pulmonary collapse, extravasation of urine) and will to some extent queer the pitch for any subsequent anterior approach to the bladder or prostate. A cystostomy does not drain the bladder from below and so leaves a sump of stagnant urine.

A good deal can be done to minimise the complications of both methods of bladder drainage. The risk of infection due to a urethral catheter can be greatly reduced if it is passed gently and skilfully with strict aseptic precautions and is connected with a *closed* system of drainage *all the components of which are sterilised*. In most cases continuous drainage with an indwelling catheter is better than repeated catheterisation. Tidal drainage had a short vogue but is now little used. The best bladder wash is an ample flow of urine. A lot depends on the quality of the catheter which should be supple, smooth and non-irritant; it must be slender enough to lie easily in the narrowest part of the urethra; it is a great advantage if it can be sterilised by heat. The Foley catheter

fulfills all these requirements and is the most generally used, especially in view of its convenient and effective self-retaining device. Because it is soft and straight, the Foley catheter cannot always be manipulated through a deformed urethra or a spastic sphincter. The Tiemann catheter is admirably adapted for this purpose as its curved and tapering tip can be insinuated through a curved and narrow passage, and its greater rigidity allows more control in its manipulation; when it is made of plastic it is sterilisable by heat and is non-irritant. A patient with an indwelling catheter should receive small regular doses of penicillin and a sulphonamide as a further means of discouraging infection. The dis-

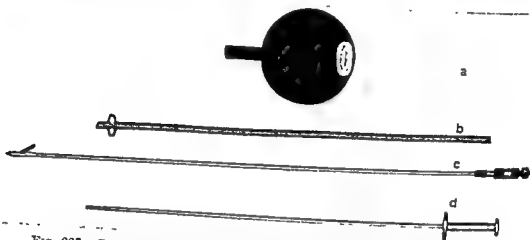


FIG. 227. *Suprapubic Cystostomy: the Riches' Apparatus.* (a) Rubber shield for securing catheter to abdominal wall. (b) Fine (16 Charrière) self-retaining catheter. (c) Special trocar. The barb just short of the tip is hinged and its angle is controlled by the milled screw in the handle. With the barb lying flat the catheter is threaded on the trocar until the barb is clear. The barb is then opened (as in illustration), the tip of the catheter is slid under it and fixed by screwing down the barb against it. The catheter is then stretched along the shaft of the trocar and stabbed into the bladder preferably after exposing the bladder wall through a small incision. By unscrewing the barb the catheter is released and the trocar is then withdrawn after screwing the barb flat. The catheter is then introduced further into the bladder by means of the special "advancer" (d).

advantages and dangers of suprapubic cystostomy can be much reduced if the wound is small, the track oblique and the catheter slender. Such a cystostomy can be conveniently and effectively made by means of the instrument devised by Riches (Fig. 227).

A different approach to the problems of urethral catheter drainage is the method of perineal urethrostomy by which a catheter is introduced through an incision in the perineum directly into the bulbous urethra and so straight up into the bladder. By this means the whole of the distal urethra with its curves and its straits is excluded. In practice, the urethral catheter is the usual means of draining the bladder in normal hospital conditions. Cystostomy is preferable if drainage must be





FIG. 228. *Senile Enlargement of Prostate.* Urethroscopic view of trilobe type of enlargement. The middle lobe is seen above the lateral lobes. This patient was treated by perurethral prostatectomy.

prolonged (as in most cases of chronic retention) or if a catheter cannot be passed. This order of preference applies to the neurogenic bladder as well as to the commoner forms of retention of urine.

There are reasons for believing that it is dangerous to empty suddenly a bladder which has been *chronically* distended, particularly if the kidneys are affected by back pressure. Sometimes hæmaturia and renal failure follow. The explanation given is that the blood vessels of the kidney, by being suddenly deprived of the support given by the urine distending the drainage system, rupture and bleed; there may also be a reflex which directly depresses renal function. The modern view is that the risk of such a complication is slight and that hæmaturia and renal failure are much more often due to infection than to the effects of rapid emptying. Nevertheless, in England rapid emptying is generally avoided in chronic retention. Slow decompression can be carried out with suprapubic cystostomy just as with urethral catheterisation.

### OBSTRUCTIVE DISEASES AT THE BLADDER NECK

Bladder neck obstruction is an extremely common condition usually affecting men but occasionally women. It is most often associated with prostatic enlargement, and in these cases the prostate itself dominates the picture and gives its name to the group which may be called *prostatic obstruction*. Those cases in which the prostate is not enlarged are placed in the group of unqualified bladder neck obstruction. This conventional distinction has some practical convenience, particularly in treatment, but there are many cases which are difficult to assign to one group or the other and there is evidence that the fundamental mechanical disorder is the same in both groups.

The exact mechanism of the bladder neck is not well understood, but there is little doubt that rigidity or increased bulk of its tissues deranges its function and may lead to retention of urine. The commonest cause of this disorder is enlargement of the prostate gland by adenomatous overgrowth. The adenomatous tissue grows chiefly in the part of the prostate immediately under the mucous membrane of the urethra and bladder; the main bulk is usually on each side in the lateral lobes but some occurs posteriorly and forms the "middle lobe" (Fig. 228). It is this posterior growth which is of special importance because it is situated rather above the lateral lobes where it is more likely to involve the bladder neck and to involve it in the place where it is most vulnerable, where the trigonal muscle retracts it on micturition (Fig. 225). Considerably increased bulk of the lateral lobes is compatible with normal micturition. There is evidence that in all types of prostatic enlargement it is the condition of the muscle at the bladder neck which is responsible for the obstruction.

**Senile Enlargement of the Prostate.** This is the common type of prostatic enlargement; synonymous terms are benign hyperplasia and



simple adenomatous hypertrophy. Histologically the hyperplastic tissue resembles that of the normal gland, but there is a greatly varying proportion of stromal elements (including plain muscle) and epithelium. The glandular acini are sometimes much dilated. The changes mostly affect the upper and central part of the gland around the urethra between the bladder neck above and the verumontanum below. The rest of the gland is compressed into a shell of varying thickness which is sometimes called the false or surgical capsule. The adenomatous tissue is separated from the false capsule by a plane of cleavage which can easily be found and opened up by the surgeon's finger, a fact which forms the basis of the usual methods of prostatectomy (enucleation). The cause of the condition is unknown, but its age incidence in a sexual organ suggests that it is an aberration of involution. The growth of adenomatous tissue is probably not indefinitely progressive; having reached a certain size by about the age of sixty the gland may remain unchanged for the rest of the patient's life. Symptoms may nevertheless develop after this time due to secondary changes in the prostate such as inflammation or congestion or due to anything affecting the efficacy of bladder contraction, and it is quite common for symptoms to develop rapidly, far more rapidly than can be explained in terms of the growth of adenomatous tissue. In other cases there is a slow and steady development of prostatic symptoms.

The early symptoms of prostatic enlargement are often irritative and take the form of frequency of micturition and sometimes urgency. The frequency may affect the patient by day as well as by night, but nocturnal frequency is often found to be due to relative nocturnal polyuria the result of an upset of the normal rhythm of urinary secretion. Whether or not there are irritative symptoms there is usually some indication of difficulty in micturition. This may take the characteristic form of delay in starting, and usually the flow when it starts is also slow, without force and sometimes intermittent. It is not helped by straining (cf. stricture of the urethra). There may be dribbling after micturition appears to be complete. Pain on micturition sometimes occurs in uncomplicated cases, but this symptom rather suggests some complication such as prostatitis or even carcinoma. Incontinence due to urgency may occur in the irritative phase, but incontinence chiefly suggests chronic retention of urine with overflow. Acute retention is a common complication. Hæmaturia quite frequently occurs, and usually the blood appears at the beginning of the stream, but there are many exceptions.

The most important general symptoms are those due to uræmia but sometimes there is dyspepsia. Hæmorrhoids and hernia are not infrequently found, especially when obstructive symptoms are severe. Incidental chronic diseases are common, for these patients are usually over sixty and often seventy or eighty. Cardiovascular conditions are particularly common, such as arteriosclerosis, hypertension, auricular

fibrillation and congestive heart failure. There may be chronic bronchitis or senile mental changes.

*Physical Signs.* Apart from the vitally important routine general examination, attention is directed particularly to the kidneys, bladder and the prostate itself. The kidneys seldom feel abnormal even when there is chronic retention of urine. A palpable bladder is a very important sign (the detection of an enlarged bladder by percussion is extremely unreliable). The prostate is examined *per rectum*. With the patient in the left lateral position the finger can be introduced rather higher than in the dorsal position, but bimanual palpation, which gives a far better indication of the size and shape of the prostate, can be done much more satisfactorily with the patient in the dorsal position. The adenomatous gland is elastic, well defined and usually symmetrical (cf. carcinoma of the prostate).

*Investigations.* As far as possible urethral instrumentation should be avoided when the prostate is known to be enlarged, for the resultant oedema due to trauma or infection may precipitate retention. When there are doubtful bladder neck symptoms and the prostate is not much enlarged diagnostic cystoscopy is justifiable, but it is usually possible to make a provisional diagnosis of prostatic obstruction without it. The urine is usually normal and sterile. A raised blood urea indicates marked renal impairment. An excretion pyelogram is an invaluable investigation for it shows not only the condition of the kidneys, but also the residual urine in the bladder and the amount of intravesical enlargement of the prostate. The final diagnosis and assessment of the prostate is made on cystoscopy immediately before operation.

*Treatment.* The mere fact of prostatic enlargement is no indication for operation, nor are some of the associated symptoms, such as frequency or hæmaturia. The only sure indication is *obstruction*, the evidence of which may be obtained from the history, the estimation of residual urine, or the signs of frank retention. The amount of residual urine is no automatic criterion and no precise figure can be set; it must be considered together with all the other data. There may even be obstruction without residual urine, but these cases can be diagnosed only by finding trabeculation on cystoscopy, although the history may be suggestive. Mild symptoms may never progress, therefore prophylactic operation is not justifiable. Moreover, much can be done to relieve mild cases by general measures. Frequency may be reduced by a moderate fluid intake timed according to need; nocturnal polyuria may be eased by pituitrin (Disipidin snuff 30 to 60 antidiuretic units) or Dexedrine (15 mg. by mouth) and urgency by propantheline (30 mg. t.d.s.). Urine should be passed as soon as possible after the desire to micturate is noticed. Exposure to cold should be avoided. In most cases hormones are useless. Such conservative measures are hardly ever justifiable when there is definite evidence of obstruction, for then operation is necessary.

The only satisfactory operation for prostatic obstruction is prostatectomy, i.e. the removal of all adenomatous tissue (Fig. 229). This inevitably involves removal of the prostatic urethra down to the top of the verumontanum and it leaves a shell of compressed prostatic tissue, the so-called *false capsule*. The posterior part of the bladder neck is also usually cut away. The raw surface left is soon covered by new mucous membrane which spreads over it from the bladder above and the urethra below and probably also from the duct orifices in the false capsule. Continence then depends entirely on the external sphincter. The two chief technical problems in the operation are to secure hæmostasis and to ensure drainage of urine until the bladder is able to empty itself efficiently again. The main operations available are: (1) Transvesical; (2) retropubic; (3) perineal; (4) perurethral (Fig. 230).

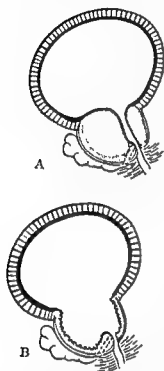


FIG. 229. Prostatectomy. Diagrams showing A the site in which the adenomatous tissue grows in senile enlargement of the prostate, and B what is removed and what remains in the standard operations of "prostatectomy." The plane of cleavage is between the adenomatous tissue and the false capsule.

In transvesical prostatectomy the bladder is opened suprapubically (the operation is often called suprapubic prostatectomy), and the "prostate" (i.e. the adenomatous tissue) is enucleated with the finger. Bleeding is controlled mainly by diathermy coagulation of the bleeding points, but sometimes sutures are used or the prostate bed is packed with gauze or rubber to produce hæmostasis by pressure. The bladder is then drained by a suprapubic tube or else closed and drained by a urethral catheter. There are several forms of transvesical prostatectomy, and they are commonly known by the names of the surgeons who popularised them. In Freyer's operation (1900) no attempt was made to stop the bleeding, and the bladder was drained by a large suprapubic tube. When this operation is done nowadays hæmostasis is helped by packing the prostate bed with gauze or Paul's tubing for twenty-four or forty-eight hours. In 1934 Harris described a method of hæmostasis by suture which enabled the bladder to be closed and drained *per urethram*. Wilson Hey's method, published in 1946, relies on the individual coagulation by diathermy of bleeding points, and the catheter must be passed (according to Hey's strict rule) down the urethra from the bladder. The bladder is closed.

*Retropubic prostatectomy* (Millin's operation, 1945) does not directly open the bladder, but the adenomatous tissue is laid bare by an incision in the capsule in front of the prostate and it is then enucleated with the

finger very much as in transvesical prostatectomy. Bleeding is controlled by diathermy and by suture of the incised capsule. Drainage is by means of a urethral catheter.

In *perineal prostatectomy*, which is far more popular in America than in England, the prostate is approached from behind, through the perineum, the capsule is incised, and the adenomatous tissue enucleated as in the anterior operations. Hemostasis is by ligature or diathermy and drainage by means of a urethral catheter.

*Perurethral prostatectomy* consists in the removal of the adenomatous tissue piecemeal by an instrument introduced through the urethra. Cutting is carried out with the "cold punch" (a cylindrical knife) or the diathermy loop (Figs. 231 and 232). Hemostasis is obtained by diathermy coagulation of individual bleeding points and drainage by means of a urethral catheter.

Any of these operations can produce very good results, and mortality figures in the region of 1 per cent. have been recorded for large series of

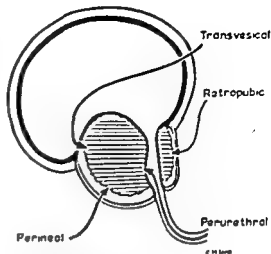


FIG. 230. *Prostatectomy*. Diagram showing the route of access to the adenomatous tissue in the four main types of prostatectomy.

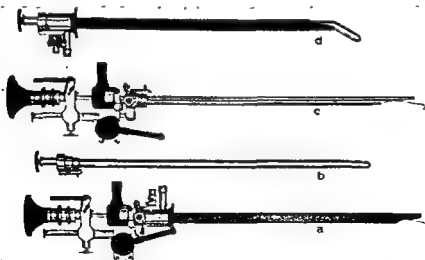


FIG. 231. *The McCarthy Electrotome ("Resectoscope")*. (a) The assembled instrument. (b) The obturator, used only for introducing the sheath into the bladder. (c) The working element, telescope and diathermy loop. (d) The sheath with obturator inserted.

cases. The choice of operation often depends on the personal preference and training of the surgeon. In general it may be said that *transvesical* prostatectomy is the easiest to perform (but not necessarily the easiest to perform well) and, if suprapubic drainage is used, the most fool-proof. The *retropubic* operation is rather more difficult but gives better access for hæmostasis and is less liable to be followed by urinary leakage from the wound. It appeals to surgical instincts and has attained an astonishingly wide popularity in a very short time. Perineal prostatectomy requires a rather specialised technique and carries a risk of permanent urinary incontinence due to damage to the external sphincter or its nerve supply. Perurethral prostatectomy imposes the least immediate strain on the patient of all the procedures, especially those using the abdominal approach. It is least suitable for the larger glands; it is rather more

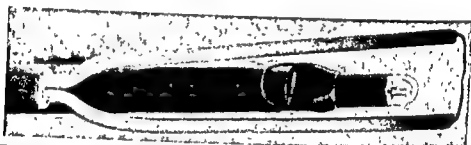


FIG. 232. The McCarthy Electrotome. Near view of lens (crescentic window), lamp and loop.

liable to cause urethral stricture than the other operations and it requires long experience before it can be done well. Immediate hæmorrhage is a difficulty with the cold punch and secondary hæmorrhage with the diathermy loop.

*Bladder Neck Obstruction without Prostatic Enlargement* ("prostatisme sans prostate" of Guyon or Marion's disease). The symptoms are those of prostatic enlargement but the pathology is not understood, and it may be that it comprises a number of different conditions having as a common factor interference with the bladder neck mechanism. The bladder neck is thickened and often rigid, especially in its posterior part. In one form, the posterior commissure of the prostate is involved, and this is called the "median bar" or "posterior commissural hypertrophy." It well illustrates the difficulty of separating pure bladder neck obstruction from prostatic enlargement. Cystourethroscopy shows that the posterior part of the bladder neck stands up and projects forward. The bladder usually shows some evidence of hypertrophy and diverticula are relatively more common than with larger prostates. Sometimes the hypertrophy of the bladder wall involves particularly the interureteric bar, and the trigone then seems foreshortened so that its raised posterior edge is near the internal meatus and blocks it, leaving a post-trigonal pouch. Although

bladder neck obstruction is commonest in men of prostatic age, it is also found in younger men and sometimes even in infants. It also occurs in women, in whom the upstanding bladder neck looks exactly like the "median bar" in men.

Diagnosis and treatment are on the same lines as for prostatic enlargement. The operative treatment differs in detail in that prostatectomy in the usual sense is not needed and the enucleation of adenomatous tissue with the finger impossible. The essential feature is the removal of the obstructing bladder neck, but it soon becomes obvious in the male that unless the prostate is also resected, at least in its upper part, it will maintain the obstruction by falling forwards and inwards. The operation can be done transvesically, but *perurethral resection* has special advantages and is the method of choice.

**Bladder Neck (including Prostatic) Obstruction with Retention of Urine.** In chronic retention of urine there is no need for hurried relief because the patient is not in pain and can pass urine. Prolonged drainage may be needed. Infection is a particularly serious complication. The safe treatment is the insertion of a suprapubic tube of fine calibre and slow decompression (e.g. 300 ml. two-hourly). The progress of the patient, and in particular the level of the blood urea, indicates when operation should be done to remove the obstruction, or whether permanent suprapubic cystostomy is indicated. This decision depends partly on the renal function and also on the general condition of the patient including the state of the heart, lungs, blood vessels and brain. A blood urea persistently over 100 mg. per 100 ml. offers a poor chance of success.

In acute retention of urine, the state of the kidneys usually does not necessitate prolonged drainage of the bladder and a urethral catheter is the best immediate treatment. The patient is in pain and needs quick relief. Prostatectomy can then be done by any preferred method as soon as the patient's condition has been assessed. It sometimes happens, however, that acute retention of urine with good renal function develops in patients with other diseases, e.g. heart failure or pneumonia. In some of these cases it is obvious that prostatectomy will not be possible for several weeks; a suprapubic cystostomy allows as long a wait as may be necessary.

Prostatectomy preceded by a cystostomy is sometimes called a two-stage prostatectomy. The presence of a cystostomy track in the operation field is a hindrance and a danger, for it obstructs the exposure of the prostate and is a source of infection. The prostatectomy may have to be modified or restricted accordingly. These disadvantages are minimised by a high cystostomy for then adequate virgin territory is left below it for a clear approach to the lower part of the bladder and prostate.

**"Immediate" Prostatectomy in Acute Retention of Urine.** Drainage of the bladder either by urethral catheter or suprapubic cystostomy is virtually certain to lead to some degree of urinary infection: prostatectomy

done after preliminary bladder drainage cannot therefore be regarded as strictly aseptic. Infection is responsible for some of the commonest and most serious complications after prostatectomy. Mainly for this reason Wilson Hey proposed that retention of urine due to prostatic enlargement should be treated by immediate prostatectomy. He advocated the transvesical operation with closure of the bladder and insisted that the ureteral catheter for post-operative drainage should be passed from the bladder downwards so as to eliminate the risk of carrying in organisms from the distal urethra. An additional argument was used to justify this treatment in patients with a raised blood urea; it was maintained that the bulging prostate itself obstructs the ureters and therefore mere drainage of the bladder does not relieve the back pressure on the kidneys; the prostate must therefore be removed forthwith. Immediate prostatectomy has been used successfully in many cases but its superiority over other methods has not been proved.

**Prostatectomy. Preoperative Management.** An adequate fluid intake and output should be assured, and the patient should not be confined to bed even if the bladder is being drained. Blood should be available for transfusion. The patient's condition is fully assessed. In most cases, cystoscopy is avoided until immediately before prostatectomy and is then done partly to discover any associated bladder pathology such as a diverticulum or papilloma, and partly to allow a more detailed assessment of the condition of the prostate itself including a final estimate of its size so that the most suitable type of prostatectomy can be chosen.

**Post-operative Management.** Usually intravenous fluid is given for about twenty-four hours to ensure adequate urinary flow and as part of the treatment of post-operative shock. Blood is given as needed. One of the most important points of all is to ensure uninterrupted drainage through the catheter; clot retention is a serious complication. Penicillin and sulphonamide are given as "cover" by the great majority of urologists, but any signs of pyelonephritis call for stronger measures in antibiotic therapy. Patients should drink as much as possible, and as soon as the urine is clear of clots they should be allowed up. When drainage is *per urethram* the catheter is removed as soon as it is judged that the bladder will empty itself naturally. This depends on the state of the urine (in regard to blood and clots), the patient's general condition and the bladder tone. Previous chronic retention requires longer post-operative drainage. Usually the catheter is removed in from two to five days. There is often some degree of residual urinary infection after catheter drainage; this is treated by specific antibiotic therapy when internal healing is complete from three to six weeks after operation.

**Complications.**—Post-operative shock, reactionary hæmorrhage and pyelonephritis are the three commonest early complications. Urinary fistula may complicate all operations on the prostate except perurethral prostatectomy. Bladder atony and secondary hæmorrhage may cause

trouble in the intermediate post-operative period. Later complications are urethral stricture, stenosis of the bladder neck, incontinence of urine, stone in the bladder and osteitis pubis. Epididymitis can almost always be prevented by vasotomy, which is usually done as a routine.

**Carcinoma of the Prostate.** Carcinoma of the prostate is common. There is evidence that it usually arises in the posterior and lower part of the gland below the plane of the ejaculatory ducts. This is not the part of the gland involved by adenomatous change, and it is the part left behind after prostatectomy. The growth spreads early. Metastasis occurs in bones (most commonly in the lumbar spine and pelvis) and in the regional lymph glands, and is often widespread. Locally the growth spreads laterally, upwards and backwards although it involves the rectum only rarely. The rest of the prostate often becomes infiltrated so that urinary symptoms may develop, and they are often indistinguishable from those due to adenomatous enlargement of the prostate, and include retention of urine. Pain on micturition is commoner than in simple enlargement, and there may be incontinence due to involvement of the external sphincter. In addition to urinary symptoms, and sometimes when they are entirely absent, there are often symptoms due to secondary deposits such as bone pains, anaemia and cachexia. On rectal examination the prostate usually has one or more of the following features: hardness, irregularity, obliteration of the median sulcus, fixity, indefiniteness of outline. Usually the prostate is not greatly enlarged. Often the local physical signs make the diagnosis reasonably certain, but fibrosis, calculi, tuberculosis and localised adenomata in the prostate may all produce changes which feel like a carcinoma. The diagnosis has usually to be confirmed by additional evidence.

*The Serum Acid Phosphatase Estimation.* The normal prostatic secretion contains acid phosphatase. In carcinoma of the prostate the enzyme may escape into the blood stream apparently due to its secretion by secondaries in the bones. There is normally a small amount of acid phosphatase in the serum derived from other sources, but the prostatic enzyme can be distinguished by its inactivation with alcohol. In practice, a serum acid phosphatase content of over 3 units per 100 ml. is suggestive of carcinoma of the prostate and over 10 units virtual proof. Unfortunately the serum acid phosphatase is often normal in prostatic carcinoma, therefore a negative result of this test is of no value.

*Radiography of the bones* may make the diagnosis certain by showing the typical dense shadows caused by osteoblastic secondary deposits of prostatic carcinoma (Fig. 233), but a difficulty may arise in distinguishing them from Paget's disease of the bone which is very common in elderly men.

Cytology of fluid obtained by prostatic massage may reveal carcinoma cells, but the technique and interpretation are very specialised. Cystourethroscopy may show changes in the prostate and bladder neck



extremely suggestive of carcinoma, but they are seldom conclusive, and carcinoma may be simulated by chronic infection or the effects of an inlying catheter.

Histology is often necessary to settle the diagnosis. Tissue may be obtained by perurethral resection or by needle biopsy through the perineum.

*Treatment.*—If the diagnosis is certain, if the growth is known to be confined to the prostate, and if the patient is fit for a severe operation,



FIG. 233. *Carcinoma of Prostate.* X-ray of pelvis showing mottled increase of bone density due to widespread osteoblastic secondary deposits.

radical prostatectomy or cystoprostatectomy may be justified. Unfortunately if the growth is early, the diagnosis is often not certain, nor even suspected, and it is never possible to be sure that the tumour has not spread outside the gland. Moreover, so-called radical prostatectomy is hardly radical in the sense in which this term is applied to cancer operations elsewhere, for there is no adequate margin between the apex of the prostate (so often the site of the tumour) and the urogenital diaphragm. Furthermore, palliative treatment is often effective for years. Therefore operation with a view to cure is seldom justifiable. There is a further difficulty, for studies of the prostate in old men suggest that there may be morphological changes indistinguishable under the microscope from carcinoma, and yet not biologically malignant growths. This may explain

the experience of many surgeons that some patients diagnosed and treated as senile enlargement of the prostate, but reported as having carcinoma on the basis of histological findings, often remain free from recurrence or metastasis although the operation done was quite inadequate for a malignant growth.

The standard treatment of carcinoma of the prostate is based on the discovery that the growth is to some extent dependent on androgens for its development and progress. The production of androgens may be reduced by castration or by administration of oestrogens, or more effectively by both methods. The usual method is to give stilbœstrol (5 mg. three times a day although very much larger doses are sometimes used). Troublesome side-effects of oestrogen therapy are painful enlargement of the breasts, dyspepsia and œdema, but a change to a slightly different œstrogenic compound may relieve them. When the administration of œstrogens does not avail or loses its effect, orchidectomy may be done in addition. In some resistant cases with severe symptoms, bilateral adrenalectomy or hypophysectomy may be justifiable. Fifteen years' experience with oestrogen therapy has shown that life is prolonged and symptoms relieved in many cases, including those with widespread secondary deposits. Bladder neck obstruction caused by carcinoma of the prostate may often not respond to œstrogens and is then best treated by perurethral resection or occasionally by suprapubic cystostomy.

Radiotherapy has not produced good results. It has been applied in the form of deep X-rays and more recently by infiltrating the prostate with a solution of a radioactive metal salt with hyaluronidase.

**Prostatitis.** Prostatitis in various forms is extremely common. *Acute suppurative prostatitis* is one of the less common forms and whereas it is still sometimes gonococcal, it is more often caused by the staphylococcus, *Bact. coli*, and other organisms. Infection may reach the prostate either by the blood stream or along the urethra. The gland becomes infiltrated with inflammatory cells and exudate, and many of the ducts are blocked. The onset may be sudden with high fever and rigors and there is deep pain in the pubis and perineum. Micturition is frequent and painful; it is often difficult, and retention may ensue. The prostate feels much enlarged and tender. The urine is likely to be infected with the causal organism. The vesicles are also very commonly involved. Complications may be retention of urine, pyelonephritis, epididymitis and prostatic abscess. The treatment is administration of appropriate antibiotics, in the first instance guided by the result of urine culture. Retention of urine or great difficulty in micturition may require an indwelling catheter. If an abscess forms it is likely to discharge spontaneously *per urethram* but rarely it may need draining through the perineum. *Chronic Suppurative Prostatitis.*—In this condition, the prostatic ducts and acini are chronically infected and dilated, and the gland substance is fibrotic. The fibrosis may result in stenosis of the prostatic urethra. Quite commonly

there is calcification in the gland some of which takes the form of stones in the ducts. This type of prostatitis may follow gonorrhœal urethritis, but in these cases the primary organisms have usually long since died out, and secondary invaders, especially Gram-negative bacilli, have taken their place. Chronic vesiculitis is also commonly present.

The main symptom of chronic suppurative prostatitis is usually chronic frequency of micturition, often with pain. There may be difficulty in passing urine. In some cases there is a chronic urethral discharge. The urine is turbid and contains threads of muco-pus. Clinical examination reveals abnormal hardness of the prostate, but its size is usually within normal limits. Prostatic massage expels cloudy fluid containing pus and epithelial cells. Radiography often shows calcification in the prostatic region. Cysto-urethroscopy completes the local investigations and the findings include rigidity of the prostatic urethra, contraction of the bladder neck, œdema or even inflammatory polypi on the prostate, dilated ducts from which pus exudes or calculi may peep.

Complications of chronic prostatitis are local, regional and general. Locally, the bladder neck and posterior urethra are affected by fibrosis which may cause obstruction. Rarely, a fistula may form involving the perineum or even rectum. Regional complications affect the rest of the genito-urinary system, and include stone in the bladder, pyelonephritis and epididymitis. The facts about general complications are less well established, but there is evidence that some rheumatic conditions including sacroiliac arthritis may be caused by chronic prostatitis.

The treatment of chronic prostatitis is difficult and the results are often unsatisfactory. Prostatic massage may bring temporary relief, and even more so urethral dilatation may be effective, particularly if there is any stenosis of the posterior urethra or bladder neck. The appropriate antibiotics should be used after sensitivity tests of the urine, but they are unlikely by themselves to eradicate completely such a deep-rooted infection. In some cases operative treatment has to be resorted to with a view to removing the chronically infected tissue. Enucleation prostatectomy is out of the question, and open surgery must take one of two forms : (a) An attempt to dissect out the glandular tissue of the prostate by a transvesical approach ; or (b) a radical removal of the whole prostate including its capsule, completing the operation by anastomosing the bladder to a distal fringe of prostatic urethra. The latter operation is formidable and carries the risk of incontinence and fistula ; the lesser operation gives indifferent results. Perurethral resection is a sound proposition, but it must be fairly radical. After all such procedures, residual infection must be swept up by all appropriate means (antibiotics, dilatation, prostatic massage).

The term *subacute non-suppurative prostatitis* is used to describe a common condition which bears some resemblance to the urethrotrigonitis of woman. It affects adults of all ages. The symptoms are variable

frequency, often with slight pain on micturition and sometimes urgency. On examination, the prostate feels normal, but sometimes an undue flow of turbid fluid can be expressed which on microscopy shows epithelial cells and amorphous debris but not pus. The urine is clear and sterile. Cystourethroscopy shows injection of the prostate, with turbid fluid flowing from the ducts; the bladder trigone is also injected. The symptoms are disproportionate to the findings. Prostate massage may bring relief and sometimes urethral dilatation may help. More radical measures are not justified.

**Prostatic Calculi.** *Corpora amylacea* are small smooth tawny bodies quite commonly found in the substance of the prostate, apparently within the ducts and acini. They have no known clinical significance and their relationship to other types of prostatic calculi is not known. *Calculi in the Prostatic Gland.*—These are extremely common especially as an incidental finding in X-rays of the urinary tract. They often cause no symptoms and are not associated with any other demonstrable disease. They may be associated with chronic suppurative prostatitis, but it is doubtful to what extent they are cause or effect. They are also found quite commonly in senile enlargement of the prostate and lie in the plain of cleavage between the adenomatous tissue and the false capsule. *Stones in the prostatic urethra* may arise from various sources. They lodge in the prostatic urethra, being prevented from passing onwards by spasm or narrowness of the membranous urethra. These stones may have come down from the kidney or bladder, or they may have formed in the ducts or lymphatics of the prostate itself. As the prostatic urethra is usually fairly roomy, stones may lodge in it without causing much obstruction, but there is often discomfort or even pain and some difficulty in micturition. The diagnosis of prostatic calculi may be suspected when the gland feels hard on rectal examination, and it is proved by X-ray (Fig. 234). The shadows may lie behind or extend well above the symphysis pubis, but they are usually easy to distinguish from those of vesical calculi which are higher still, near the centre of the pelvic ring. A stone in the prostatic urethra must be removed. This is fairly simply done by pushing it back into the bladder and then evacuating it through a large canula, with or without crushing it, depending on its size. Stones within the prostate gland require no treatment unless there is associated disease of the gland. If simple adenomatous enlargement requires operation the stones are incidentally evacuated when the adenomata are removed. If the calculi are associated with chronic suppurative prostatitis, they aggravate the problem of treatment, and any surgical attack, whether endoscopic or open, should aim also at removing all the stones.

**Urethrotrigonitis in the Female.** This is a very common mild inflammatory condition usually occurring in young married women. It affects the upper part of the urethra, the bladder neck and the trigone. These parts are hyperæmic, and the inflammatory area spreads like a fan over

the trigone. The upper limit, approaching the ureteric orifices, is sharply defined. The bladder neck is usually irregular, and just inside the upper end of the urethra are found a varying number of oedematous polypoid projections. The urine is free from pus and is sterile, but super-added



FIG. 234. *Prostatic Calculi.* Note how the shadow lies immediately above and also behind the symphysis pubis. This patient also had senile enlargement of the prostate causing obstructive symptoms.

bouts of infective pyelocystitis are fairly common. The symptoms are frequency and often painful micturition. The diagnosis is made by urethroscopy. Treatment is dilatation of the urethra. In addition, the more severe cases may need light fulguration of the urethral polyps. The response to treatment is usually not permanently satisfactory.

## CHAPTER 30

### DISEASES OF THE URETHRA AND PENIS

IN the male the urethra is subdivided into various parts, but unfortunately the nomenclature lacks definition and standardisation. A nomenclature is used here which is accepted fairly generally in England. The main dividing point is the external sphincter. Above it (proximally) lies the posterior or prostatic urethra and below it (distally) the anterior urethra. The membranous urethra is the short stretch running through the external sphincter itself; this muscle lies between the layers of the *urogenital diaphragm* (triangular ligament). The long anterior urethra is further subdivided by rather ill-defined boundaries into bulbous, scrotal and penile parts. The distal extremity of the urethra as it runs through the glans is dilated and is called the *fossa navicularis*. Most of the urethral mucous membrane is composed of columnar cells, but near the external meatus it is stratified and squamous. The lining of the urethra is pitted by the orifices of small glands and some larger lacunæ. The anterior urethra is ensheathed in the erectile tissue of the *corpus spongiosum* (*corpus cavernosum urethræ*); the posterior urethra is surrounded by the prostate. From the surgeon's point of view the calibre of the urethra is of the highest importance. Its narrowest and least distensible part is the external meatus. Often the membranous urethra may appear to be even narrower, but this is only due to spasm of the external sphincter and, although very common, can be prevented or overcome by various measures. Another part which may be narrowed by spasm is the bladder neck. The bulbous and prostatic parts of the urethra are both fairly roomy, but the penile urethra is relatively narrow. There is much normal variation in the calibre of the urethra.

**Scales of Urethral Calibration** (Fig. 235). There are three main scales, the French or Charrière (designated F. or Ch.), the Bénéiqué (designated B.) and the English (designated E.). The modern tendency is to use exclusively the Charrière scale, in which each number represents the circumference of the instrument in millimetres. In the Bénéiqué scale, the numbers are double those in the Charrière so that the gradations are twice as fine. The English scale is entirely traditional and empirical and has only sentiment to commend it, but it will die hard. It is the coarsest scale of the three. In the majority of adult patients the urethra easily admits an instrument of size 22 F. (44 B. or 12 E.), and in many it accommodates size 28 F. although in a proportion of these meatotomy is necessary. There is a fixed curve of about a right angle in the posterior end of the bulb as it turns upward to pass through the external sphincter. This

curve must be carefully negotiated when a rigid instrument is passed along the urethra. The action of gravity on the flaccid penis (the patient lying supine) produces another curve in the opposite direction at the junction of the fixed and movable parts of the shaft of the penis. This curve has little importance surgically because it can easily be abolished by holding the penis on the stretch in the appropriate direction.

The urethra has an extremely rich blood supply and bleeds easily and freely. The lymphatics of the distal part drain into the groin and those of the proximal part into the iliac glands.

In the female the basic structure of the urethra is similar to that in the male; the differences follow from the absence of the penis and prostate.

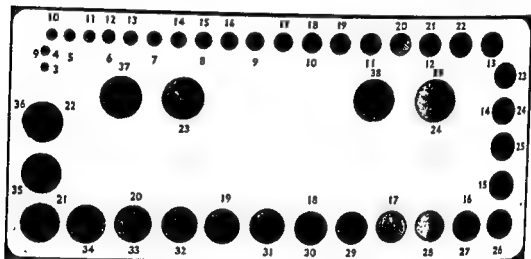


FIG. 235. *Catheter Gauge.* This shows the relation between the French (Charrière) and the English scale. The larger figure for any given size represents the Charrière scale. (Illustration  $\frac{2}{3}$  scale.)

The external sphincter is very near the bladder neck, and below it is a slightly dilated part corresponding with the bulb in the male. There is erectile tissue in the walls of the dilated part of the urethra and there are glands opening in the mucosa; there are also lacunae opening at or near the external meatus. The consistency and thickness of the tissues intervening between the vagina and the urethra vary from patient to patient, so that the findings on palpation of the urethra *per vaginam* need careful interpretation. The female urethra is rather wider than the male urethra and the external meatus is much more distensible.

**Examination of the Urethra.** Direct clinical examination is chiefly useful for the external meatus. Palpation of the rest of the urethra may reveal gross lesions such as a calculus impacted in the fossa navicularis, but the erectile tissue ensheathing the urethra is a barrier to fine palpation in both sexes. *Sounding* the urethra is a very ancient and still a very useful method of examination, used chiefly to detect and localise obstructions, particularly strictures. Sounding is also the usual way of measuring

the calibre of the urethra ; graduated sounds are passed in increasing sizes until the limit is reached. The terms *sound*, *bougie* and *dilator* are used synonymously for these instruments. Although there are many varieties and modifications of these, there are only three main types (Fig. 236) : (1) Curved metal ; (2) flexible ; (3) short, straight metal (for female urethra and distal part of male urethra).

*Urethroscopy* is by far the best method of examining the urethral mucous membrane. There are two main types of instrument. The *air-distension* urethroscope has the great advantage of not washing away discharges and is therefore used chiefly in the study of urethritis. It

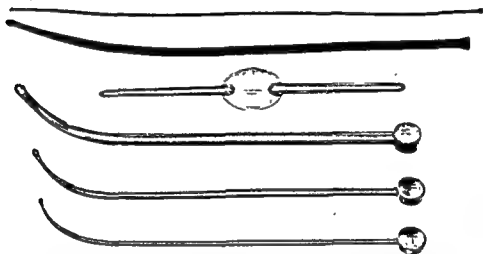


FIG. 236. *Urethral Dilators.* The two top instruments are flexible bougies (plastic). Below them is a straight metal dilator (Canny-Ryall's type) for the female or the distal end of the male urethra. The three bottom instruments are Lister's curved steel sounds. The figures are sizes in the English scale ; the greatest and the smallest calibre of each tapering sound are both marked.

cannot be used above the level of the external sphincter. The *water-distension* urethroscope is the more generally useful instrument. There are many varieties and two which are extensively used are the Swift Joly urethroscope which has direct forward vision and the McCarthy panendoscope which has foroblique vision : both magnify. *Urethrography* produces a radiographic silhouette of the urethral lumen. Its advantages are : (a) It provides an exact and permanent record ; (b) it can be used in spite of the presence of obstructions which make urethroscopy impossible (e.g. many strictures) ; (c) it does not create such artificial circumstances as does the passing of a rigid instrument through the urethra. The radiopaque fluid may be injected from below by means of a nozzle engaged in the external meatus, or it may be introduced into the bladder and then passed by the patient on micturition. The latter method is



useful chiefly in studying the bladder neck and posterior urethra whereas the former is better for investigations of the anterior urethra, particularly for strictures.

**Urethritis.** The urethra is most commonly infected during sexual intercourse, but it may also be infected via the blood stream or by the passage of infected urine along it. An important cause of urethritis is contact with or injury by instruments, particularly the indwelling catheter. The symptoms vary a good deal among the different types of urethritis, but the main symptom is a discharge of serum, mucus or pus from the external meatus. *Gonococcal urethritis* is almost invariably venereal. The infection is usually strictly confined to the part of the urethra lined with columnar cells so that it does not involve the sub-terminal urethra, nor does it spread to the bladder. Infection soon reaches the deeper layers of the urethra and may involve the corpus spongiosum. This spread in depth, due partly to involvement of the deep-lying glands, is responsible for some of the main complications, particularly persistence of the infection and stricture formation. The symptoms of acute gonorrhœa begin from two to five days after infection, with a feeling of local burning or irritation, worse on micturition; the characteristic profuse, thick yellow discharge soon follows. Sometimes there is difficulty in micturition and rarely retention of urine. Diagnosis is confirmed by the finding of Gram-negative diplococci in the cells of the discharge. It is very important to look for any other coexistent venereal infections particularly syphilis. The treatment of gonorrhœa has been revolutionalised by modern chemotherapy, for the organism is extremely sensitive to the sulphonamides and to penicillin. The danger of the latter is that the dose which cures gonorrhœa (300,000 units) is sufficient to mask the signs of concomitant syphilis, but is not sufficient to cure it so that the diagnosis of this grave disease may be dangerously delayed.

Acute gonococcal urethritis may become *chronic*; this is usually the result of inadequate treatment. Infection lurks in the urethral glands, in the periurethral tissues and in the prostate, and there is often secondary infection with other organisms. The symptoms of the chronic stage are a slight and variable urethral discharge known as *gleet*, and some pain on micturition. The urine may contain threads of pus. Gonococci may be found in the discharge but often they are sought in vain. The most important late complication of gonorrhœa is urethral stricture. When gonorrhœa involves the posterior urethra it may give rise to epididymitis, and this in turn may lead to sterility.

*Non-gonococcal (or non-specific) urethritis* is common and appears to be on the increase. It is usually venereal, but its exact cause has not been established. It may be due to any one of a variety of agents including bacteria, pleuropneumonia-like organisms, trichomonads, spirochaetes or a virus. It is sometimes followed by epididymitis ("non-specific" epididymitis) and sometimes by abacterial pyuria. It may be associated

with conjunctivitis and arthritis in the condition known as *Reiter's syndrome*. The symptoms of non-gonococcal urethritis are usually mild, and, apart from the conditions mentioned above, complications are rare. It usually responds best to broad spectrum antibiotics such as oxy-tetracycline (Terramycin) and chlortetracycline (Aureomycin).

### INJURIES OF THE URETHRA

(a) **External Trauma.** (i) *Rupture of the Bulbous Urethra.*—This fairly uncommon injury is the result of falling astride something; the urethra is crushed between the pubic arch and the external object. There is severe pain, bleeding from the external meatus, bruising in the perineum and inability to micturate. The rupture may be complete (circumferential) or incomplete, or the urethra may be only contused. The history and the local signs make the diagnosis almost certain, but it should be confirmed by the passage of a catheter: inability to pass it into the bladder indicates a complete rupture. Urethroscopy may be done, but not with an air-distension urethroscope owing to the danger of air embolism. The treatment of complete rupture is exceedingly important if subsequent stricture is to be avoided. The first necessity is diversion of the urine by suprapubic cystostomy. The perineum should then be freely incised, the ruptured ends of the urethra found and carefully sutured. The wound is drained. A catheter should not be left in the urethra as it hinders healing and encourages infection. The cystostomy is discontinued after two or three weeks. Thereafter bougies must be passed periodically to ensure that a stricture does not develop, but it is extremely important not to overstretch the scar. In incomplete rupture the bladder may be drained by urethral catheter for several days if one can be got in; no local repair need then be done. If, however, there is much bruising and swelling in the perineum it is safer to treat the patient as for a complete rupture of the urethra.

(ii) *Avulsion of the membranous urethra* is caused by tearing of the prostate away from the urogenital diaphragm (triangular ligament) in fracture of the pelvis. There is severe local trauma and copious bleeding from torn veins. The integrity of the bladder neck often prevents extravasation of urine. This type of injury is caused by great violence and there are often injuries to other parts of the body. The patient is shocked, unable to pass urine and there may be bleeding from the external urinary meatus. There is clinical evidence of fracture of the pelvis. Efficient treatment is vitally important if stricture is to be avoided, but it is very difficult because the patient is shocked, the lesion is inaccessible, definition of anatomy is hindered by laceration and bleeding, and the instability of the broken pelvis makes maintenance of the position of the urethra unreliable. In most cases the only feasible solution is to thread a catheter along the urethra across the gap and draw it into the bladder. An internal splint is thus made for the ruptured urethra. Some

ingenuity is needed in carrying out this apparently simple manœuvre. The bladder is drained suprapubically. A synchronous abdominoperineal approach has been used to enable direct suture of the urethra to be done.

(b) **Internal Injury by Instruments.** The urethra is a delicate organ and can be injured by very slight force. The danger of using fine metal bougies is obvious and well known, but pliable bougies and the thicker metal instruments can also penetrate the urethral wall and produce false passages. Penetration by a straight instrument usually occurs in the posterior wall of the urethra, most commonly in the bulb; and penetration by a curved instrument in the anterior wall either below or above the external sphincter. The bougie may sometimes leave the lumen of the urethra, produce a false passage running parallel to it, and then re-enter the lumen. False passages comparatively seldom cause serious complications, but they make subsequent instrumentation more difficult. The important thing is not to make them. Instruments cause more serious injury by stretching than by penetrating the urethra, for stretching results in stricture. The narrowest part of the urethra is the external meatus and strictures are often caused here or in the fossa navicularis immediately above it either by the forcible passage of a large instrument

which splits and contuses the tissues, or by the lying in the urethra of a catheter which fits too snugly and produces urethritis and pressure ulceration. These complications can be avoided by using instruments of appropriate size for each patient or by performing an ample meatotomy if a larger instrument must be used. Another means of avoiding injury to the distal urethra is to pass the instrument through a perineal urethrostomy.

### URETHRAL STRICTURE

**Post-inflammatory Stricture.** This type of stricture usually follows gonorrhœa, but sometimes also other forms of urethritis. In gonorrhœa there is a very long latent interval between the urethritis and the manifesta-



FIG. 237. *Urethral Stricture.* Urethrogram showing diaphragm stricture just below membranous urethra.

tion of the stricture, the average being twenty-one years. The cause of the stricture is contraction of the fibrous tissue which forms as a result of suppuration in the submucous tissue and corpus spongiosum. The infection reaches this depth by invading the urethral glands, and as these are more numerous on the dorsal aspect of the urethra, the lumen is usually drawn towards the roof of the urethra. A stricture may involve any part of the anterior urethra, but most often the bulb. Strictures may be very short ("diaphragm" stricture—Fig. 237) or long ("tunnel"



FIG. 238. *Urethral Stricture.* Urethrogram (oblique view) showing tunnel stricture just below membranous urethra.

stricture—Fig. 238), and they may be multiple. Although the original gonococcal infection has usually died out, secondary infection with other organisms has usually occurred. It is relatively inactive, but is easily stirred into activity by trauma. The symptoms of urethral stricture are almost always those of urinary obstruction. The slow urinary stream is helped by straining, unlike that in most cases of prostatic obstruction; but, like that of prostatic obstruction, the stream is a sluggish dribble unless the stricture is very near the external meatus, when it may be thin and forceful. Urinary infection is much more likely to be associated with obstruction caused by urethral stricture than with that due to the bladder neck or prostate, and there may be symptoms of cystitis. Hydronephrosis or pyelonephritis may lead to uræmia.

ingenuity is needed in carrying out this apparently simple manœuvre. The bladder is drained suprapubically. A synchronous abdominoperineal approach has been used to enable direct suture of the urethra to be done.

(b) **Internal Injury by Instruments.** The urethra is a delicate organ and can be injured by very slight force. The danger of using fine metal bougies is obvious and well known, but pliable bougies and the thicker metal instruments can also penetrate the urethral wall and produce false passages. Penetration by a straight instrument usually occurs in the posterior wall of the urethra, most commonly in the bulb; and penetration by a curved instrument in the anterior wall either below or above the external sphincter. The bougie may sometimes leave the lumen of the urethra, produce a false passage running parallel to it, and then re-enter the lumen. False passages comparatively seldom cause serious complications, but they make subsequent instrumentation more difficult. The important thing is not to make them. Instruments cause more serious injury by stretching than by penetrating the urethra, for stretching results in stricture. The narrowest part of the urethra is the external meatus and strictures are often caused here or in the fossa navicularis immediately above it either by the forcible passage of a large instrument

which splits and contuses the tissues, or by the lying in the urethra of a catheter which fits too snugly and produces urethritis and pressure ulceration. These complications can be avoided by using instruments of appropriate size for each patient or by performing an ample meotomy if a larger instrument must be used. Another means of avoiding injury to the distal urethra is to pass the instrument through a perineal urethrostomy.

#### URETHRAL STRICTURE

**Post-inflammatory Stricture.** This type of stricture usually follows gonorrhœa, but sometimes also other forms of urethritis. In gonorrhœa there is a very long latent interval between the urethritis and the manifesta-



FIG. 237. *Urethral Stricture.* Urethrogram showing diaphragm stricture just below membranous urethra.

Some contract again very quickly and need dilatation every few weeks. Some patients find the ordeal of periodic dilatation very trying. Strictures are sometimes so tough that they can never be adequately dilated. In other cases, dilatation always produces a severe general reaction such as "catheter fever" or "urethral shock." When dilatation is unsuccessful or inadvisable, reconstruction of the urethra may be done. A method based on the Denis Browne operation for hypospadias (Chap. 24) is being increasingly used and gives good results. The principle that underlies this method is to convert the stricture into a hypospadias at one operation, and at a subsequent operation to repair the hypospadias. The operation of internal urethrotomy is seldom used nowadays and is merely an adjuvant to dilatation. Excision of post-inflammatory strictures with direct suture of the divided ends of the urethra is frequently followed by recurrence of the stricture due to reactivation of dormant infection. Very occasionally the poor general condition of the patient, or his refusal to submit to other forms of treatment, necessitates a permanent suprapubic cystostomy.

**Post-traumatic Stricture.** This is best prevented by correct treatment of the original injury. If a stricture does develop it is often most conveniently treated by dilatation. Unlike post-inflammatory stricture, traumatic stricture may be treated by excision and suture with fair prospect of a good result, but the modified Denis Browne procedure will often be preferable to excision. For strictures very near or actually involving the urogenital diaphragm excision or the Denis Browne procedure are not feasible, but an ingenious pull-through operation has been described for their treatment by Badenoch. In this operation the urethra is divided just below (distal to) the stricture and mobilised in the perineum for about 2 inches. By means of a bougie passed into the urethra from above (through the opened bladder) a passage is cleared through the fibrotic region of the stricture into the perineum and this passage is dilated until the mobilised distal urethra can be drawn up through it to the bladder.

**Instrumental Stricture.** This type of stricture may be the result of a single injury to the urethra, usually over-stretching, or it may be due to the more prolonged effects of an indwelling catheter. There are probably several factors in the production of stricture by a catheter. The most important is pressure due to the use of a catheter that fits too tightly. This pressure causes ulceration of the mucosa, and, by blocking the orifices of the glands, causes infection to develop in the deeper layers of the urethra from where the infection may spread to the periurethral tissues and the corpus spongiosum. Such infection is more likely to develop if there is any fault in the aseptic technique of passing the catheter or in maintaining aseptic conditions thereafter. There is also probably another factor, namely the composition of the catheter, for there is evidence that certain substances, particularly some types of rubber,

There are commonly no physical signs of a stricture. In the penile urethra the periurethral fibrosis may be palpable, but in the bulb the bulbocavernosus muscle obscures it. The urine may be infected with a variety of organisms. Urethroscopy shows a stricture as a whitish area with a small eccentric hole in it. In most cases, however, the diagnosis is made by sounding. An average size sound is held up at the site of a stricture, and when a small enough sound can be got through the stricture it seems to be gripped. The two most likely difficulties in differential diagnosis are caused by spasm of the external sphincter and some form of prostatic or bladder neck obstruction. In spasm of the external sphincter (which is extremely common), if a sound can ultimately be got through it never feels to be gripped; the spasm relaxes around it. In prostatic obstruction the site of arrest may be obviously above the external sphincter and a large sound can sometimes be got through more easily than a small one, and a curved sound more easily than a straight; and there is no "gripping" unless the gland is carcinomatous. In all doubtful cases the diagnosis can be settled by urethroscopy. Another valuable means of investigating strictures is urethrography, particularly in long, multiple and impassable strictures. These additional means of investigation are not usually necessary if the diagnosis is clear and treatment is to be by dilatation.

The complications of urethral stricture include periurethral abscess, extravasation of urine, urethral fistula, impaction of calculi above the stricture, retention of urine (acute or chronic) and uræmia.

**Treatment of Post-inflammatory Strictures.** When there is chronic retention of urine or uræmia, preliminary suprapubic cystostomy is needed, but in most cases the stricture can be treated directly. The standard method is still the old-fashioned one of graduated, periodic dilatation, and in most cases it works well. The difficulty is usually in beginning, for a stricture has often become very narrow before the patient seeks advice and then only the finest bougies can be got through. The first session is helped by general anaesthesia, by patience, gentleness and skill on the part of the surgeon, and by bougies of good quality and the right sizes. At the first treatment, the dilatation must not be forced. At subsequent sessions, the stricture is progressively dilated until the limit of dilatation is reached; this varies from patient to patient. In some, the stricture can quickly be dilated to the normal urethral calibre, whereas in others nothing larger than a 12 F. bougie can ever be passed. The lumen is maintained by periodic dilatation as often as may be necessary, but never less often than once a year, for there is a wise saying "Once a stricture, always a stricture," and the abandonment of treatment, even in cases which respond favourably, often leads to recurrence of symptoms, and symptoms usually indicate severe contraction of the stricture.

Periodic dilatation is not a satisfactory treatment for all strictures.

**Urethral Calculi and Foreign Bodies.** Stones which enter the urethra from the bladder may be arrested by a stricture or by some anatomical narrowing, usually the external meatus (Fig. 239). There is sudden pain and stoppage of the stream. If the stone is at the external meatus it can easily be felt and often seen. It is delivered, if necessary after meatotomy. A stone arrested by a stricture may sometimes be released simply by dilating the stricture. External urethrotomy may be necessary, but should be avoided if possible in the penile urethra as a fistula may be caused. Stones may actually form *de novo* above a stricture due to



FIG. 239. *Urethral Calculi.*  
Plain X-ray. The stones are impacted behind a stricture of the external meatus.

stagnation and infection. Such stones are almost always phosphatic. Foreign bodies are sometimes introduced into the urethra and lodge there, either because they penetrate the wall or because they are too rigid to round a bend of the urethra. They are removed if possible through the lumen but urethrotomy may be necessary.

**Carcinoma of the Penis.** This is a squamous cell neoplasm arising on the glans or on the inner surface of the prepuce (Fig. 240). It is almost unknown in the circumcised and is commonly associated with phimosis especially when there is chronic balanitis. It is particularly likely to be found in dirty and neglected old men. The growth spreads by direct extension (and so may involve the urethra) and by the lymphatics to the inguinal glands. The history is of a sore or tumour on the penis or of a discharge from within an unretractable foreskin. When the prepuce is retractable, the diagnosis is usually obvious, but when the growth is flat and ulcerated it may have to be distinguished from inflammatory ulcers



are directly injurious to the urethral mucosa. Latex rubber and plastics appear to be the safest materials. Another very obvious factor in the production of instrumental stricture is the length of time the injurious effect lasts; an indwelling catheter should always be removed as soon as possible. The treatment of instrumental strictures is almost always by dilatation and they usually respond well.

---

**Periurethral Abscess.** This complication may develop during the course of an acute urethritis or many years after by the lighting up of a long dormant infection. An abscess forms outside the urethra, sometimes near a stricture, and usually points in the perineum or on the ventral surface of the penis. A brawny swelling appears which may discharge spontaneously. There is local pain, fever and sometimes retention of urine. The physical signs of a deep-seated abscess in relation to the urethra, particularly if there are urinary symptoms, often make the diagnosis obvious, but when the swelling is in the perineum the differential diagnosis includes infections spreading from the rectum and anus, ischiorectal abscess, anterior pilonidal sinus and furunculosis. Urethroscopy settles the diagnosis in doubtful cases. The treatment consists of free incision of the abscess where it is pointing, appropriate chemotherapy, and dilatation of any stricture. Retention of urine requires either an indwelling catheter or in some cases suprapubic cystostomy.

**Extravasation of Urine.** This is a most dangerous complication of urethral disease. It may arise from a stricture or from a periurethral abscess in the wall of which a weakness develops. Urine escapes and collects in the areolar tissue contained within Colles's fascia. It then spreads in this layer over the penis and scrotum, back to the perineum and upwards over the lower abdomen and groins. Virulent infection develops with extensive suppuration and often gangrene. The patient is extremely ill and toxic. Treatment consists in suprapubic cystostomy, free incision and drainage of the perineum, scrotum, groins and lower abdomen, and appropriate chemotherapy. If the patient survives, the local lesion of the urethra (often a stricture) will require treatment.

**Urethral Fistula.** This may follow a periurethral abscess or penetrating wounds. In the distal part of the urethra it may be due to necrotic lesions such as soft chancre. It is sometimes associated with carcinoma of the urethra or penis. Behind the penoscrotal junction, fistulae tend to heal spontaneously because of the thickness of tissue over the urethra, unless there is obstruction distally. In the penile urethra, a fistula quickly becomes lined with epithelium and therefore cannot heal without operation; moreover, unless the repair is very thorough the fistula will recur.

In the female the commonest causes of urethral fistula are injury during operations on the vagina, particularly colporrhaphy, and injury at childbirth.

to treat them only if there is positive clinical evidence of involvement, and then they are removed by block dissection.

**Penile Warts.** The warts are commonly due to a virus, and may be acquired in sexual intercourse. They are also known as *condylomata acuminata*. They are most often found in the coronal sulcus and vary much in numbers and size. Their surface is not ulcerated, but if they become sodden (as when under the prepuce) they bleed easily. There is no induration of the base. Usually the most effective treatment is the local application of podophyllin (25 per cent. suspension in paraffin) which often causes them to disappear within a day or two. In moist regions podophyllin causes an intense reaction and alternative methods of treatment are local destruction of the warts by diathermy, or, when they are confined to the prepuce, removal by circumcision.

**Penile Herpes.** This is a fairly common condition. Crops of vesicles form on the glans or prepuce. They soon break and weep, and if secondary infection ensues ulceration may occur. The symptoms are mild, but the patient may be worried by the fear of venereal disease.

**Syphilitic Chancre.** The fact that syphilis is becoming much less common increases the danger of failing to diagnose it in the early stages. The hard or Hunterian chancre develops about three weeks after infection, and is usually found on the glans or prepuce. It is a papule with a very firm base and a raw surface exuding clear serum. Commonly the inguinal glands are enlarged. The diagnosis is confirmed by finding the *Spirochaeta pallida* (*Treponema pallidum*) in the discharge. The microscopic examination is done with dark ground illumination and without staining. The Wassermann reaction does not become positive until two or three weeks after the appearance of the chancre. Once the diagnosis has been made, the local treatment of a syphilitic chancre is unimportant, for the disease is already generalised and must be eradicated by a complete course of anti-syphilitic treatment. Before the diagnosis is settled, it is important not to apply anything which might kill the spirochaetes in the discharge and so prevent their being found in the diagnostic smear. It is equally important never to give penicillin while the diagnosis of syphilis remains a possibility, for it may mask the signs without curing the disease.

**Chancroid or Soft Chancre.** This is a local disease due to infection with Ducrey's bacillus acquired in sexual intercourse. The lesion appears a few days after infection as a papule which soon becomes a pustule and then an ulcer with ragged undermined edges. The sores are commonly multiple, and differ from the syphilitic chancre in two striking ways: they are painful and they are not indurated. The glands of the groin are commonly enlarged and tender; they may suppurate. The infection responds to the sulphonamides, to streptomycin and to Aureomycin.

**Balanitis.**—This is inflammation of the glans penis. It almost always occurs in the uncircumcised, and involves the prepuce as well; the term

including syphilis, soft chancre and penile herpes. In doubtful cases biopsy is essential. Any discharge from under an unretractable prepuce or a lump palpated through it necessitates a dorsal slit so that the lesion can be uncovered and diagnosed. The treatment of carcinoma of the penis is by surgery or radiotherapy. For the small lesion not involving the urethra nor penetrating deeply, radiotherapy (by means of a radium

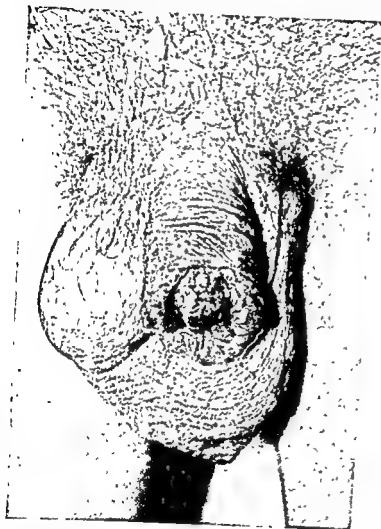


FIG. 240. *Carcinoma of Penis.* There is ulceration of the dorsum of the glans. The swelling of the prepuce is due to reaction following the operation of dorsal slit necessitated by phimosis. This carcinoma was treated by partial amputation of the penis.

mould or interstitial needles) gives a very good chance of cure and leaves a relatively intact penis. For all other types surgery is preferable. Whenever possible a partial amputation (i.e. through the shaft of the penis) should be done, because this leaves sufficient penis for almost normal male micturition. Extensive growths require radical amputation. In this operation the penis is removed from its origins on the pubic arch and the urethra is brought out into the perineum. For hygienic reasons, to avoid the excoriation and stink from urinary soiling, it is often best also to remove the scrotum and testicles. It is a fairly severe operation for a feeble old man. The usual procedure for the glands in the groin is

to treat them only if there is positive clinical evidence of involvement, and then they are removed by block dissection.

**Penile Warts.** The warts are commonly due to a virus, and may be acquired in sexual intercourse. They are also known as *condylomata acuminata*. They are most often found in the coronal sulcus and vary much in numbers and size. Their surface is not ulcerated, but if they become sodden (as when under the prepuce) they bleed easily. There is no induration of the base. Usually the most effective treatment is the local application of podophyllin (25 per cent. suspension in paraffin) which often causes them to disappear within a day or two. In moist regions podophyllin causes an intense reaction and alternative methods of treatment are local destruction of the warts by diathermy, or, when they are confined to the prepuce, removal by circumcision.

**Penile Herpes.** This is a fairly common condition. Crops of vesicles form on the glans or prepuce. They soon break and weep, and if secondary infection ensues ulceration may occur. The symptoms are mild, but the patient may be worried by the fear of venereal disease.

**Syphilitic Chancre.** The fact that syphilis is becoming much less common increases the danger of failing to diagnose it in the early stages. The hard or Hunterian chancre develops about three weeks after infection, and is usually found on the glans or prepuce. It is a papule with a very firm base and a raw surface exuding clear serum. Commonly the inguinal glands are enlarged. The diagnosis is confirmed by finding the *Spirochæta pallida* (*Treponema pallidum*) in the discharge. The microscopic examination is done with dark ground illumination and without staining. The Wassermann reaction does not become positive until two or three weeks after the appearance of the chancre. Once the diagnosis has been made, the local treatment of a syphilitic chancre is unimportant, for the disease is already generalised and must be eradicated by a complete course of anti-syphilitic treatment. Before the diagnosis is settled, it is important not to apply anything which might kill the spirochætes in the discharge and so prevent their being found in the diagnostic smear. It is equally important never to give penicillin while the diagnosis of syphilis remains a possibility, for it may mask the signs without curing the disease.

**Chancroid or Soft Chancre.** This is a local disease due to infection with Ducrey's bacillus acquired in sexual intercourse. The lesion appears a few days after infection as a papule which soon becomes a pustule and then an ulcer with ragged undermined edges. The sores are commonly multiple, and differ from the syphilitic chancre in two striking ways: they are painful and they are not indurated. The glands of the groin are commonly enlarged and tender; they may suppurate. The infection responds to the sulphonamides, to streptomycin and to Aureomycin.

**Balanitis.**—This is inflammation of the glans penis. It almost always occurs in the uncircumcised, and involves the prepuce as well; the term

*balanoposthitis* is therefore more complete. *Balanitis* is caused by accumulation of smegma and dirt under the prepuce, and this in turn is due either to want of proper hygiene or to *phimosis*. Additional factors may be specific infections such as syphilis, or tumours. Treatment is by circumcision whenever there is *phimosis*, and in all cases by attention to hygiene. Sometimes the infection is so severe that a dorsal slit of the prepuce is necessary in order to allow free drainage before circumcision can be considered. Although *balanoposthitis* is usually acute and often associated with suppuration, it may occur in a chronic form of low-grade activity, without suppuration or any discharge. This type may result in slow fibrous contracture of the external meatus or preputial orifice. There is a precarcinomatous form of this process known as *kraurosis*.

**Phimosis.** In adults, this may be due to the persistence of a primary infantile *phimosis* or to the narrowing of the preputial orifice by secondary causes such as suppurative *balanitis* or *kraurosis*. Rarely the narrowing is sufficient to obstruct urination, but the common symptom is trouble in sexual intercourse due to difficulty in retracting the foreskin or in reducing it. Indeed it is important to recognise that the preputial orifice is adequate only if it allows the prepuce to be withdrawn and reduced again when the penis is erect. For this reason the patient's own account is of more importance than the physical signs in the consulting room. In many cases *phimosis* is symptomless, particularly in older men. The treatment of *phimosis* which has caused symptoms or complications in the adult is circumcision. The operation is easier to do in the adult than in the infant, and is less liable to be followed by complications, except those due to the occurrence of erections in the early post-operative period. This painful and troublesome sequel, which may tear open the wound and cause bleeding, may to some extent be prevented by the administration of sedatives and oestrogens.

**Paraphimosis.** This term is applied to strangulation of the end of the penis by a tight foreskin which has been retracted behind the glans. The tightness of the foreskin prevents a free return of blood from the penis distal to it; this causes swelling so that the foreskin constricts more tightly still, and this in turn causes more swelling and so a vicious circle is set up. *Paraphimosis* usually occurs during an erection. The striking feature is the great swelling of the foreskin distal to the constricting ring (which is the preputial orifice). This loose tissue is capable of gross distension by oedema, and it forms a bulging ruff round the neck of the glans, completely burying the constriction ring behind it. The strangled tissues are cyanosed, sometimes ulcerated and rarely gangrenous. The treatment of *paraphimosis* has two aspects. The immediate consideration is to release the strangled tissues. This is usually done by manipulation after an attempt to reduce the oedematous swelling by expression. If this method fails the constriction ring is divided on the dorsal aspect, and reduction is then always possible. A gentler method has recently been

described in which the swelling is dissipated by the injection into it of a small amount of hyaluronidase (500-1,000 units in 2 ml. of 1 per cent. lignocaine); reduction may then be easy. The second consideration in the treatment of paraphimosis is the prevention of recurrence, and this almost always means circumcision. Whenever possible this should be done at the time the paraphimosis is reduced, but gross sepsis may make it inadvisable until later. If a dorsal slit has been made to obtain reduction, recurrence of the phimosis is likely; circumcision should therefore still be done. Paraphimosis in children is discussed in Chap. 24.

**Priapism** (named after the god Priapus). This is a condition of painful and prolonged erection, unaccompanied by sexual desire. It may be due to injury or disease of the central nervous system, or to metastatic malignant conditions in the penis, including leukaemia. Sometimes no cause can be determined. Treatment is unsatisfactory but, if symptoms are severe, an attempt should be made to reduce the turgidity of the penis by aspirating the corpora cavernosa with a wide bore needle.

**Fibrous Cavernositis or Peyronie's Disease.** This is a condition in which plaques of fibrous tissue develop in the sheaths of the corpora cavernosa. The cause is unknown. The effect is to make erection crooked and sometimes painful. It usually occurs in elderly patients in whom sexual efficiency is likely to be unimportant so that no treatment is required. In younger men, the disability may be troublesome. Treatment is unsatisfactory. The plaques may be excised, but haemorrhage is severe and the results are poor. Recently some favourable results have been reported by injecting the plaques with hydrocortisone (5 mg. in 2 ml. of saline under general anaesthetic).

**Operations on the Urethra and Penis.** *Circumcision.*—Local anaesthesia may be used (annular block round the base of the penis), but general anaesthesia is more convenient. If the prepuce can be retracted, its inner surface and the glans can be cleaned as well as the exterior surface of the penis. In any case the operation is done with the prepuce drawn forwards. Three haemostats are applied to the end of the prepuce, at one, six and eleven o'clock (Fig. 241). The dorsum of the prepuce is then slit open

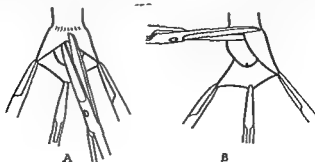


FIG. 241. Circumcision. A. Three haemostats applied to the prepuce. B. Circumcision partly done.

longitudinally down the middle until the corona of the glans is exposed. This is done with scissors after ascertaining that there is a clear space between the prepuce and the glans. The prepuce is now cut away on each side separately, beginning at the top of the original midline incision but leaving the frenum intact until both sides have been done, so as to make use of the traction provided by the hæmostat holding it. The frenum is divided as soon as the rest of the prepuce has been cut away. There should now remain a fringe of the inner lining of the prepuce (the "mucosa") not more than  $\frac{1}{4}$  inch long all round the base of the glans. The bleeding points are picked up and tied (there are often not more than three of them), and then the cut edges of the skin and "mucosa" are united with interrupted plain catgut stitches, a mattress stitch being used at the frenum. A dressing of Whitehead's varnish or Nobecutane is applied.

*Meatotomy.*—The floor of the fossa navicularis is crushed between the jaws of a fine hæmostat, and the crushed line is then cut along with scissors, so laying open the fossa navicularis for the desired distance. A short straight sound is then passed to ascertain that sufficient enlargement of the external meatus has been obtained. The preliminary crushing of the tissues minimises hæmorrhage and to some extent prevents the cut edges from reuniting. The danger of reunion can be further avoided by inserting a stitch so that the knot lies in the apex of the incision between the cut edges; the distal ends of the cut edges can also be held away from each other by a stitch placed in the cut edge and tied and then entered again in the corona and tied there also. Regular dilatation at increasing intervals will be needed to maintain the enlargement.

## CHAPTER 31

### DISEASES OF THE TESTIS AND ITS APPENDAGES

THE *testis* varies much in size between one individual and another. Except as a result of disease, it varies less in consistency. The tough *tunica albuginea* prevents much swelling of the testis. The arterial blood supply of the testis is derived mainly from the upper abdominal aorta through the *testicular (internal spermatic) artery* but partly also from the external iliac through the *cremasteric artery* (a branch of the inferior epigastric). This dual supply prevents atrophy of the testis if the testicular artery is divided above the inguinal canal. The venous drainage is through the *testicular veins* into the renal vein on the left side and directly into the inferior vena cava (but sometimes the renal vein) on the right side. The lower reaches of this venous stream are formed by the network of the *pampiniform plexus* in the spermatic cord. The lymphatics of the testis drain along the course of the blood vessels into the lateral aortic and pre-aortic lymph glands which lie topographically in the epigastrium. The *epididymis* is a soft and slender structure which runs usually down the back of the testis. A fairly common variation is for the epididymis and vas to run down the front of the testis. The upper end of the epididymis (also called the *head* or *globus major*) is the larger and the easier to feel. The epididymis is capable of much greater enlargement than the testis. Its arterial supply is provided by a branch which leaves the testicular artery a variable distance above the testis and runs laterally and downwards to the upper part of the epididymis. This relatively separate blood supply makes the operation of epididymectomy possible without compromising the blood supply of the testis. The sac formed by the *tunica vaginalis* covers all except the back of the testis (where the epididymis is attached), and usually extends for some distance upwards over the spermatic cord. The layers of fascia and the cremaster muscle which suspend and surround the testis and epididymis vary considerably in thickness even in health; in disease, particularly inflammatory disease, these fascial coats often become much thickened and indurated by inflammation, infiltration and œdema; and this may make examination of the scrotal contents and especially interpretation of the findings very difficult. The normal *vas deferens* varies much in thickness. A generalised increase in thickness above the average is commonly not the result of disease. The special feature of the *scrotum* is that its skin is rugose and vascular and lies directly on the *dartos muscle* without the interposition of subcutaneous fat. The very loose areolar tissue in the scrotum allows it to be distended very easily, and there is very little



tissue tension to stop the formation of a large post-operative hæmatoma if hæmostasis is not meticulous.

**Torsion of the Testis.** (See Chap. 14.)

**Epididymitis.** This is by far the commonest inflammatory condition occurring in the scrotum. In the great majority of cases it arises by infection travelling along the lumen of the vas from the posterior urethra,

vesicles or prostate (Fig. 242). The passage of such an infection leaves no trace in the vas. On the other hand, the vas is quite often involved by a secondary process in which inflammation spreads upwards along it from the inflamed epididymis. The inflammation may likewise spread forwards from the epididymis and so involve the testis (*epididymo-orchitis*). The epididymis may rarely be infected through the blood stream as in septicæmia and the acute specific fevers. It is probable that the lymphatics never convey infection from the central end of the vas to the epididymis, although the lymphatics from the prostate and vesicles do run up along the vas for the first part of its course in the pelvis. Epididymitis may be caused by any type of infection occurring in the posterior urethra or prostate. Gonorrhœa used to be a common cause but now it is rare. Acute infections of the urinary tract are a common cause and the organism is frequently the *Bact. (Esch.) coli*. Tuberculosis is another important cause, and arrives by the same route as the other infections, i.e. along the lumen of the

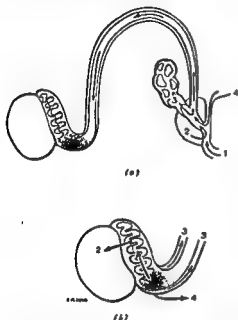


FIG. 242. *Epididymitis.* Diagrams representing arrival and spread of infection. (a) The initial invasion of the lower pole by organisms travelling along the lumen of the vas from (1) the urethra, (2) the prostate, (3) the vesicles, and (4) from urinary infection. (b) The various directions along which inflammation may spread from the original focus: (1) Along the epididymis towards its upper pole (very common); (2) into the testis (uncommon); (3) back in the walls of the vas (common for a short distance); and (4) into the scrotal tissues (common in tuberculosis).

vas. In all these conditions suppuration may ensue. In acute infection, the pus usually points anteriorly and is secondary to involvement of the testis itself, whereas in tuberculosis a sinus forms directly in relation to the epididymis and is therefore situated posterolaterally and commonly near the lower pole. There is a common form of acute or subacute epididymitis, usually called *non-specific*, in which suppuration does not occur. Sometimes there is no obvious primary condition such as urinary infection or disease of the prostate, vesicles or urethra, although there may be a history of non-specific urethritis in the previous few weeks.

The symptoms of epididymitis are painful swelling in the scrotum

and often also pain in the groin. The onset is sometimes sudden, and the pain may be severe, particularly when the testis is secondarily involved. There may be fever. There is a tender swelling in the scrotum over which the skin may be red. Owing to the inflammatory infiltration of the coverings of the cord and testis, it is often impossible to distinguish the anatomical components of the swelling, but when the acute phase has subsided the epididymis stands out as a well-defined swelling of characteristic shape. It is elongated, slightly curved, and in its anterior concavity nestles the testis. Below the testis juts the lower pole of the epididymis, which is usually the largest part of the swelling (Fig. 243). The lower part of the spermatic cord is thickened and so also may be the part of the vas running upwards from the lower pole of the epididymis. In subacute and chronic epididymitis, the anatomy of the swelling may be clearly defined from the start, and frequently only the lower part of the epididymis is involved. When the epididymis itself can be felt to be enlarged and tender, the diagnosis of epididymitis is simple. The difficulties arise mainly when inflammation obscures the anatomical features so that primary disease of the testis cannot be excluded, particularly tumour



FIG. 243. *Acute Epididymitis.* Photograph of section of operation specimen. The lower pole of the epididymis is greatly enlarged, equal in bulk to two-thirds of the testis. The testis is normal. There is a small secondary hydrocele. The cause was prostatitis. Orchidectomy is occasionally justifiable when severe epididymitis occurs in the elderly.

which may simulate inflammatory disease. Other conditions which have to be considered in the differential diagnosis are torsion of the testis and strangulation of a scrotal hernia. When there is very little pain or tenderness, it is sometimes difficult to distinguish epididymitis from non-inflammatory diseases of the epididymis, in particular cystic degeneration. Once epididymitis has been established its cause must always be determined by clinical examination of the prostate and vesicles and by laboratory examination of the urine.

*Treatment.*—This has four aspects: (1) *Prevention* is possible by division of the vas (*vasotomy*), and this is most commonly done as an addition to the operation of prostatectomy which otherwise is followed by epididy-

mitis in 5 to 15 per cent. of cases. Division of the contralateral vas is sometimes carried out in tuberculous epididymitis. (2) *Eradication of the cause* applies chiefly to tuberculosis, but in all acute cases the treatment is given which is appropriate to the organism known or assumed to be responsible, although acute epididymitis usually responds poorly to systemic measures against infection. (3) *Symptomatic treatment* is important in acute cases, and consists chiefly of rest, analgesics and elevation of the scrotum. (4) *Operation* is most commonly done in tuberculosis, and consists usually of epididymectomy. In most other types, operation is confined to the occasional evacuation of pus in acute suppuration, but in some cases of severe epididymitis in elderly men early orchidectomy brings quick relief by removing an organ for which the patient has little further use.

The most important late effect of epididymitis is sterility, caused by obstruction of the epididymis or vas as the result of scar contraction. If the testis is involved it may slough and shrivel.

**Orchitis.** This disease is rare except as a complication of epididymitis. It is probable that the epididymis, unlike the vas, cannot transmit infection without itself being infected and inflamed so that orchitis without epididymitis is almost always blood borne. It occurs in the acute specific fevers, particularly *mumps*, and in *septicæmia* and *pyæmia*. Sometimes hæmatogenous orchitis suppurates and forms an abscess of the testis. If such an abscess ruptures in the acute phase and continues to discharge, an ulcer forms and the testicular tissue thus exposed in the floor of the ulcer can be recognised by the fine thread-like tubules which can be drawn from it; this condition is sometimes called *hernia testis*. *Syphilitic orchitis* has become very rare. It may take the form of *diffuse fibrosis* or still more rarely of a *gumma*. In all forms of orchitis a small secondary hydrocele is found but it is inconspicuous. In syphilis, on the other hand, secondary hydrocele is a prominent feature. The diagnosis of orchitis requires great care and caution. Any swelling of the testis itself (as opposed to the epididymis), whether it appears to be inflammatory or not, must always be considered to be neoplastic until proved otherwise, and it must never be forgotten that orchitis is rare except when associated with epididymitis. The association of mumps may give an obvious clue, and it is hardly ever justifiable to diagnose mumps orchitis without *sialadenitis*. A positive Wassermann reaction is circumstantial evidence in favour of syphilitic orchitis but does not prove it. *Torsion of the testis* and *acute hæmatocele* may both be mistaken for orchitis. In any doubtful case the testis should be explored.

**Hydrocele.** This is an abnormal quantity of clear fluid in the tunica vaginalis. By far the commonest variety is the so-called *idiopathic* or *primary hydrocele*. The cause of this condition is not known, as its name implies. It occurs mostly in middle and old age, and is extremely common. It develops slowly and insidiously, and becomes noticeable on account

of the weight or bulk of the scrotal contents. It is often bilateral but usually one side is larger than the other. The fluid in a hydrocele is clear and straw coloured. It has a protein content, mainly albumen, of about 5 per cent. The tunica vaginalis is sometimes thickened and plaques of fibrin may be deposited on its serous surface: rarely it may be calcified. Hyaline loose bodies (*corpora libra*) are sometimes found in the fluid. Occasionally the cavity of a hydrocele is loculated by adhesions between the testis and the tunica vaginalis. An untreated hydrocele is a very harmless condition. Complications are rare and include infection, the formation of a hæmatocele and rupture. In young men a hydrocele may possibly cause sterility, for the fluid is a thermal insulator and may prevent the normal cooling of the testis on which fertility partly depends. A hydrocele is an oval fluctuant swelling and the examining fingers can be made to meet above it, thus distinguishing it from a hernia. Usually it is confined to the scrotum, but sometimes it runs up along the cord into the groin, and rarely it may extend into the abdomen. Most hydroceles can be transilluminated, but it is important when doing the test to stretch the skin of the scrotum tightly over the hydrocele and to hold the source of light against the stretched skin. Not uncommonly the thickness of the tunica vaginalis prevents transillumination. In a tense hydrocele, the testis cannot be felt but its position can usually be mapped by transillumination. In a lax hydrocele, the testis can be felt in the posterior and lower part, but if the upper part of the hydrocele is squeezed, the tension rises in the lower part and the testis can no longer be felt clearly. The fascial coverings of a hydrocele vary a good deal in thickness, and the thickness sometimes varies from place to place so that a hydrocele may appear to herniate through the weak places in its coverings, giving it an irregular or lobulated surface.

*Treatment.* If a hydrocele is symptomless there is no indication to treat it except in the doubtful case of impaired fertility in a younger man. The great majority of hydroceles are never treated. If treatment is needed three methods are available, namely tapping, injection of sclerosing fluids, and operation. *Tapping* is a simple, effective and time-honoured treatment. Its great disadvantage is that it needs to be repeated indefinitely. *It should never be used when there is an associated scrotal hernia.* The death of the historian Edward Gibbon was due to peritonitis which followed the tapping of a hydrocele which was associated with a hernia. In the tapping of a hydrocele a trocar and cannula is more satisfactory than a syringe and needle. It is important to locate the testis reliably. The hydrocele is then held firmly and squeezed so that the fluid is forced into the inferior part of the sac and the trocar and cannula are thrust briskly well into the fluid in a direction which avoids the testis. In most cases the point of entry is antero-inferior and the direction of the thrust upwards and slightly backwards. This little operation is usually done without any anæsthetic, but sometimes the infiltration of a little 2 per cent.

Xylocaine is advisable. *Injection of sclerosing fluid* is an old method which has in general been superseded by operation, but it may still be used. More than one injection may be needed, and the reaction may be painful. If cure is not achieved subsequent operation may be difficult. The solutions used are sodium morrhuate, Ethamoline and quinine urethane. *Operation* is the method of choice in all active patients whose general health is good. It consists either of the removal of the tunica vaginalis or of its eversion. The former is the better operation and it is followed by very few recurrences. Eversion is a less certain cure and, although it is a lesser operation in that it does not entail cutting away the tunica vaginalis, it nevertheless involves complete mobilisation of the tunica from the scrotum, and it is this process which is so likely to lead to the reactionary swelling or hæmatoma which are the chief complications of such operations on the scrotal contents. In any case meticulous hæmostasis and gentleness are essential if good results are to be obtained.

Other types of hydrocele may be distinguished from the ordinary idiopathic type either on anatomical or pathological grounds. Unfortunately some old and confusing terms are still used for the anatomical varieties. A *communicating hydrocele* is one in which the tunica vaginalis communicates with the peritoneal cavity through a patent processus vaginalis. It occurs almost only in infants, and its characteristic feature is its variability in size. Owing to the danger of peritonitis it should not be treated by tapping. This type is often called a *congenital hydrocele* by analogy with congenital hernia, but the term is misleading. *Infantile* is the term given to a hydrocele which extends up along the cord for a varying distance but does not communicate with the peritoneum. The term is unfortunate and the distinction of little importance so long as it is appreciated that a hydrocele may extend up into the groin. (Hydroceles of infancy and childhood are discussed in Chap. 14.)

*Secondary or symptomatic hydroceles* are those associated with disease of the testis or epididymis. In most cases the amount of fluid is small and the symptoms and signs of the underlying disease overshadow the hydrocele. An important but rare exception is syphilitic disease of the testis (*vide supra*). The common causes of secondary hydroceles are inflammatory disease of the testis and epididymis, and less often trauma. Tumours of the testis may be associated with a small hydrocele.

*Encysted hydrocele of the cord* is due to the collection of fluid in a small part of the processus vaginalis which has remained patent, the remainder above and below having been obliterated normally. It is usually encountered before middle age and it causes very few symptoms. Clinically it is recognised as a cystic swelling in the spermatic cord; it can be fixed by drawing the testis downwards. The examining fingers can be made to meet above the swelling which has no expansile impulse on coughing; but when the swelling is situated in the inguinal canal these features may

be difficult to determine, and it may then be mistaken for a small inguinal hernia. When a hydrocele of the cord occurs near the epididymis it may be difficult to distinguish from a cyst of the epididymis (e.g. spermatocele), but in practice the distinction is unimportant. If an encysted hydrocele of the cord is the cause of symptoms it should be removed.

**Cysts of the Epididymis.** These cysts are very common and are usually found in middle and old age. By far the commonest type is the *sperma-*



FIG. 244. *Cysts of Epididymis (Spermatocele).* Drawing made during operation for cysts of the right epididymis. This is the usual type in which the cysts are multiple.

*tocele* which is filled with an almost clear fluid, faintly opalescent because it contains spermatazoa and also fat globules and epithelial cells. Spermatoceles occur in the upper end (head) of the epididymis, and they are usually multiple, although one or two of the cysts are much larger than the rest (Fig. 244). They are often bilateral, but if so they are usually much larger on one side than the other. In their symptoms, or the lack of them, they resemble hydroceles. Their cause is unknown. The *physical signs* are those of a cystic, translucent swelling, often lobulated due to the multiplicity of the cysts, closely attached to the testis but distinguishable from it. The cysts are usually above and behind the testis, but this relationship is not constant, particularly when the cysts are large. When the cysts are small they are felt as a rubbery enlarge-

ment of the upper part of the epididymis, and may somewhat resemble chronic epididymitis. When the cysts are larger they have to be distinguished from a hydrocele, and this is best done by squeezing the upper part of the swelling; if the testis can still be distinctly felt, hydrocele is excluded. The need for treatment is determined by the symptoms and the condition of the patient. Tapping is a feasible method of treatment, but is more liable to be followed by troublesome complications than it is in hydrocele. This is partly because the needle or trocar is more liable to injure the veins of the pampiniform plexus and so cause a hæmatoma, and partly because the effects of infection, if it occurs, are more serious in the epididymis than in the tunica vaginalis. Operation is the more satisfactory treatment. The cysts may sometimes be shelled out, but often the upper half of the epididymis has to be removed if minute cysts are not to be left behind. Sometimes cysts of the epididymis do not contain spermatozoa, and of course they never do so in the rare cases occurring before puberty. Sometimes they contain so many spermatozoa or so much floating fat or other debris that the fluid is opaque, like milk, and the cyst may then be called a "galactoceles." It is traditional to ascribe the origin of some of the clear fluid cysts to vestigial embryonic remnants such as the hydatids of the testis and epididymis and the organ of Giraldes.

**Hæmatocele.** This is a collection of blood in the tunica vaginalis. It is usually due to trauma (occasionally operative); sometimes no cause can be found, and rarely it is caused by a tumour of the testis. The physical signs are similar to those of a hydrocele except that a hæmatocele cannot be transilluminated. It is usually harder than a hydrocele because of organised thrombus lining the tunica vaginalis, and in the early stages there may be bruising and oedema of the scrotum. In the traumatic cases the diagnosis is fairly obvious, but even in these the swelling may resemble epididymo-orchitis. When the onset is insidious it may be impossible on purely clinical grounds to distinguish a hæmatocele from a tumour of the testis, and even if aspiration of the tunica vaginalis draws blood the residual thickening may still make it impossible to exclude a tumour. A hæmatocele may be treated by aspiration of the blood, but often it is advisable to excise the thickened tunica vaginalis. In some cases orchidectomy may be justifiable in the middle aged or elderly. In every case a tumour must be excluded with certainty.

**Varicocele.** This term is applied to a varicose condition of the veins of the pampiniform plexus. It is quite common; it usually occurs on the left side, and in most cases it is symptomless. Its cause is not known, but there have been various speculative hypotheses. Rarely, a varicocele develops acutely due to obstruction of the testicular vein at its upper end by a renal tumour. The common idiopathic varicocele develops insidiously during adolescence. The great majority of varicoceles require no treatment. If there are symptoms (e.g. a dragging pain or a feeling

of weight) they may be relieved by a scrotal suspender. Operation is rarely justifiable on account of symptoms alone, but there is some evidence that varicocele may be a factor in impaired fertility by keeping the testis warm. The older operation consisted of the dissection and excision of most of the veins of the pampiniform plexus in the part of the cord running down from the external ring. Damage to the testicular artery in this situation is difficult to avoid with certainty, and it is likely to lead to atrophy of the testis. For this reason an operation is now advocated in which the testicular veins are tied in the iliac fossa, for in this position it is easier to avoid the artery, and even if the artery is ligatured here the testis may continue to be nourished by an anastomotic arterial supply through the cremasteric (external spermatic) artery.

**Tumours of the Testis.** Compared with the many common diseases affecting the scrotal contents, testicular tumours are rare. They are important because they are almost always malignant. Benign tumours are extremely rare. There is a form of benign teratoma consisting of a conglomeration of various well-formed tissues (adult in the embryological sense), including cartilage, muscle and glands. These tumours are symptomless and may be found during routine examination of an infant. Orchidectomy is usually necessary because without complete histology it is not possible to be sure that the tumour is not malignant. Another rare benign tumour is the interstitial cell tumour arising in the cells of Leydig. It may be associated with precocious puberty.

The majority of malignant tumours of the testis fall into one of two groups, the *seminomas* and the *teratomas*. The *seminoma* is the commoner tumour and consists of masses of cells of a uniform type giving the cut surface a homogeneous appearance macroscopically. Histologically the cells are large with a clear cytoplasm and resemble spermatocytes. The *teratomas* have a much more variegated histological appearance and may contain cells or groups of cells resembling any or all of the three germinal layers. There is every degree of differentiation and malignancy, but on the whole *teratomas* are more malignant than *seminomas*. Both types of tumours spread by the lymphatics to the iliac and para-aortic glands, and quite commonly to more remote glands such as the supraclavicular group. They also spread by the blood stream (particularly the *teratomas*), and pulmonary secondaries are common, but metastasis may be widespread; the sites include the liver, the brain and the bones. A rare and extremely malignant type of tumour, which may be classed with the *teratomas*, but is usually in a class by itself, is the *chorion-epithelioma*, which both histologically and biologically resembles chorionic tissue, and tends to cause hæmorrhage by eroding blood vessels.

Testicular tumours usually occur in young adults, the *teratomas* rather earlier (twenty to forty years) than the *seminomas* (thirty to fifty years). There is no doubt that imperfect descent of the testis is a predisposing factor, although there are differences of opinion as to the degree



of importance of this factor. The percentage of tumours occurring in imperfectly descended testes varies between 9 and 30 per cent. in different series, whereas the total incidence of imperfect descent is only 0.39 per cent. Nevertheless the great majority of testicular tumours are found in testes which have descended normally. One of the remarkable things about testicular tumours is that some of them (but rarely seminomas) are associated with a rise in the gonadotrophic hormone secretion of the pituitary so that the urine of such patients may yield a positive *Zondek-Asheim reaction*; the highest titre is given by the chorion-epitheliomas. This finding has, however, little practical importance except occasionally in the detection of impalpable and invisible secondaries when the primary tumour has been removed. A negative result is of no help. In rare cases the breasts become enlarged. The presenting symptom of the more malignant tumours may be due to metastatic deposits. The local symptom is usually insidious, painless enlargement of the testis. There is sometimes a history of trauma, but there is little evidence that trauma can be causal. In about 10 per cent. of cases the initial swelling is painful, and these early symptoms may appear to settle for a time. The explanation of this difficult and dangerous behaviour is that hæmorrhage occurs in the tumour and some of the blood is subsequently absorbed.

The diagnosis of testicular tumour is almost exclusively a clinical exercise. It may often be difficult, but the difficulties are greatly reduced and the risk of serious mistakes is lessened if every swelling of the testis itself is considered to be a tumour until proved otherwise. The great majority of swellings in the scrotum are cystic (hydrocele and spermatocele), or involve primarily the epididymis; the testis can be felt to be normal in most of these conditions although it may be necessary to aspirate a hydrocele to prove this point. A hydrocele does not commonly arise secondary to a tumour. A tumour usually produces an irregular enlargement of the testis. In most cases it is hard, but often its consistency varies in different parts of the tumour, and there may be soft or even cystic areas. Characteristically testicular sensation is lost where the tumour has destroyed or displaced the normal tissue, but this test is not reliable. In fact these details are not of great importance so long as it has been established that the testis itself is enlarged. A tumour entirely surrounded by normal testicular tissue produces a uniform enlargement of the testis with normal consistency. Although tumours are not common they are in fact one of the commonest causes of enlargement of the testis. Other causes are syphilis (which is nowadays rare in civilised countries and may be revealed by the Wassermann reaction) and chronic abscess. The most difficult cases are those in which it is impossible to decide whether or not the swelling is of the testis itself, and this problem is quite frequently presented by epididymo-orchitis at the stage in which the thickened and indurated fascial layers in the scrotum prevent accurate palpation of their contents. In most of these cases the symptoms and

signs are clearly those of an inflammatory condition, but it is important to remember that some testicular tumours closely mimic inflammatory conditions. In most cases of epididymo-orchitis the diagnosis declares itself soon, usually by the emergence of the characteristic epididymal swelling. Whenever doubt remains the testis should be exposed by opening the tunica vaginalis : if it is found to be enlarged and the epididymis normal, orchidectomy should be done.

The treatment of testicular tumours is a combination of surgery and radiotherapy. The testis is removed together with the cord up to the internal ring ; the gland areas, chiefly para-aortic and iliac, are subsequently treated by deep X-ray therapy. The seminomas in particular are very radio-sensitive and tumour deposits melt away ; unfortunately they frequently reappear. The teratomas are less radio-sensitive and for them the older operation of radical dissection of the lymph drainage area has been revived by some surgeons. In order to gain access to the glands lying beside the great vessels above the level of the renal pedicles an extensive thoracoabdominal exposure may be used. The prognosis is worst for chorion-epitheliomas (which are uniformly fatal) and best in the well-differentiated seminomas. The average five-year cure rate for all testicular tumours is about 40 per cent. and the ten-year cure rate about half that.

**Diseases of the Scrotum.** The rugosity of the scrotal skin makes it liable to collect and retain dirt so that faulty hygiene is often followed by *dermatitis*. The retention of foreign particles between the rugæ may have more serious effects if they contain a carcinogenic irritant. Chimney sweeps' and mule spinners' cancer were both due to the prolonged action on the scrotal skin of carcinogenic irritants. Happily both these occupational diseases are now rare. *Epithelioma* of the scrotum still occurs. It is treated by wide excision, and if the inguinal lymph glands are clinically involved they are treated by block dissection. The scrotal skin is rich in *sebaceous glands* and *sebaceous cysts* are therefore very common in this situation. The scrotum is a common site for *pruritus*.

*Idiopathic gangrene* of the scrotum is a remarkable though not a common condition. The cause is not certainly known although clinically it appears to be infective, and many different organisms have been isolated during the course of the disease. It begins suddenly with severe pain in the scrotum which becomes swollen and inflamed. The skin becomes gangrenous within a few days of the onset, and the patient's condition is extremely toxic. If the patient does not succumb the gangrenous skin separates, frequently leaving both testicles entirely exposed. The most remarkable feature of the disease is now witnessed, for the skin regenerates with astonishing rapidity and grafting is unnecessary. The treatment is to control the infection with antibiotics ; penicillin may be rapidly effective.

The laxity of the areolar tissue within the scrotum allows distension to

occur easily, and trauma, either accidental or operative, may be followed by the formation of a large *hæmatoma*. Similarly the scrotum may swell disproportionately when there is some general cause of *œdema* such as cardiac failure. Local or regional lymphatic obstruction may also affect the scrotum severely as in *filarial elephantiasis*.

**Male Infertility.** Fertility in the male depends on the delivery into the vagina of an adequate amount of semen containing a sufficient concentration of healthy, motile spermatozoa. Either partner in a marriage may be responsible for infertility; *in about half the cases the male is at fault*. In the investigation of male infertility it is necessary at the outset to exclude impotence or some gross technical error in sexual intercourse. A routine history and physical examination aims at discovering any organic disease of the genito-urinary system, particularly the testes, epididymes and vasa. The state of development of the secondary sex characteristics may indicate an endocrine disorder. One or two special tests are needed to complete the investigation in the majority of cases.

**Seminal Analysis.** The method of collecting the specimen that gives the most reliable results is by masturbation. The important data required are: (1) The volume of the ejaculate; (2) the numbers of sperms per cubic millilitre; (3) the proportion of motile sperms. Many other points may be investigated but their interpretation is controversial and doubtful. The volume matters only if it is unduly small, provided sperm density is adequate. The average is 3.5 ml., and less than 2 ml. may be inadequate for fertilisation. A sperm density of less than 20 million per ml. (i.e. a total ejaculate of about 60 million sperms) is probably the lower limit for fertility. More than half the sperms should be motile.

**Testicular biopsy** is a very minor operation, and can be done under local or general anaesthesia. In a minority of cases decisive information may be obtained, and it may take one of two forms: (1) Normal testicular histology with complete spermatogenesis. This finding if associated with azoospermia in the seminal analysis indicates obstruction somewhere between the testis and the ejaculatory duct, and may possibly be amenable to surgical treatment (e.g. epididymo-vasostomy). (2) Complete absence or destruction of the germinal cell layer. The prognosis is hopeless and treatment useless. The position should be explained to the patient who then has the opportunity to adjust himself to his misfortune. In the majority of cases the result of testicular biopsy is not decisive. Various degrees of spermatogenic arrest may be found or other histological abnormalities. In these cases there is some hope of improvement.

**Treatment.** Any defect in the technique or mechanics of sexual intercourse should be corrected if possible. This may entail, for example, operation for hypospadias. Obstruction in the epididymis or vas may be amenable to operative correction. Persistent oligospermia with a relatively slight defect of spermatogenesis as shown by testicular biopsy sometimes responds to "rebound treatment." This consists of still

further depressing spermatogenesis by administration of testosterone for about three months. During the course of the following six months the sperm count often rises above the original level. Unfortunately this laboratory improvement is seldom reflected in the achievement of pregnancy.

### OPERATIONS ON THE TESTIS

**Excision of Hydrocele.** The scrotum having been shaved and cleansed is firmly grasped so that the hydrocele is thrust forwards under the stretched skin. A vertical incision is made through the scrotal skin directly on to the hydrocele and deepened until the bluish translucent surface of the hydrocele is reached. This is the parietal layer of the tunica vaginalis; the fascial coats around it are stripped away and the bleeding points are carefully secured. When the hydrocele is completely mobilised, it is opened anteriorly, and then the tunica vaginalis is cut away on each side to within  $\frac{1}{4}$  inch of its attachment to the epididymis. Great care is taken not to injure the vas or the testicular vessels while this dissection is being done. The next step is hæmostasis. Every point, no matter how small, is secured. Diathermy is a great help. A running stitch in the cut edge of the tunica is a quick but slightly less certain method of gaining hæmostasis, and sometimes the two cut edges, one on each side of the epididymis, are sown together behind the epididymis. Before the testis is replaced the interior of the scrotum is carefully inspected for any bleeding. The incision in the scrotum is now sutured with drainage. Lastly the lower end of the scrotum is grasped in a tissue forceps and held up while the scrotum is firmly bound with a length of strapping displacing the testis into its upper, firmer part, thus further minimising the risk of post-operative hæmatoma. Catgut stitches are useful in the skin of the scrotum for unabsorbable stitches are difficult to remove. The strapping binding and the drain are removed on the third or fourth post-operative day.

**Epididymectomy.** The initial steps of this operation are the same as in excision of a hydrocele until the tunica vaginalis is incised. The head of the epididymis is then seized in fine tissue forceps and carefully dissected away from the testis. It should all the time be drawn laterally to avoid the line of the testicular vessels. When the head of the epididymis has been detached the rest of it is dissected away until the junction with the vas is reached. The vas is then separated from the other structures of the cord up to the level of the pubic tubercle, and here it is divided and the proximal cut end brought out on to the skin of the groin through a stab incision (infective material travelling along the vas will then be discharged harmlessly on the skin). The division of the vas completes the detachment of the epididymis and most of the tunica vaginalis is usually removed at the same time. Hæmostasis and closure are as for excision of a hydrocele.

**Vasotomy.** The testicle is drawn downwards and the vas is felt for through the skin of the scrotum. When it is identified it is steadied, lifted forward against the skin and cut down on to. When it is exposed in its sheath it is grasped with tissue forceps (e.g. Poirier's) and the sheath of the vas is then incised along the line of the vas until the naked vas is revealed. It is then lifted out of its sheath, clamped, divided and ligatured. The scrotal skin is closed with one catgut stitch.

## CHAPTER 32

### GENITO-URINARY TUBERCULOSIS

TUBERCULOSIS of the genito-urinary system arises by spread of infection from a focus in other parts of the body, chiefly the lungs, but in most patients presenting with symptoms of the genito-urinary disease, the primary focus is quiescent and not detectable clinically. The route of spread is the blood stream, and the kidneys are probably the first organs in the genito-urinary system to be involved. It is however extremely difficult to plot the course of the infection because healing may obliterate the tracks of the disease. Failure to find evidence of renal tuberculosis in some cases of tuberculous bacilluria has led to the suggestion that live organisms are "excreted" by the kidney, passing through it without causing disease. This unlikely suggestion may easily be discarded if the hypothesis is accepted that small tuberculous foci may arise in the kidney, infect the urine, but then succumb to the natural defence of the body leaving scars too small to be detected by any clinical investigation. This proposition is supported by the research of Medlar who examined histologically the kidneys of patients who had died of advanced pulmonary disease but without symptoms of renal infection. He found 367 tuberculous lesions of the kidneys but had to search through 100,000 sections to find them as they were so small and scanty. It is now generally accepted that small foci in the kidney may often heal; indeed it is probable that the initial lesions in patients who develop established disease are multiple and bilateral, but that most of them heal leaving one focus which progresses and causes clinical manifestations.

The rest of the genito-urinary system is probably involved by spread of infection from the kidneys in most cases, although the mechanism is not completely understood (Fig. 245). A likely sequence is that tubercle bacilli (*Mycobacterium tuberculosis*) from the urine infect the prostate or vesicles through their openings in the posterior urethra and thence by travelling along the lumen of the vas reach the epididymis. This does not



FIG. 245. *Genito-urinary Tuberculosis.* Scheme representing the spread of infection. (a) The broad arrow represents the hematogenous invasion which survives and develops: the slender branch arrows lead to small parenchymatous foci which do not survive. From the renal lesion, the infection spreads downwards to the ureter (b), the bladder (c), the prostate (d), and the vesicle (e). It spreads along the lumen of the vas (f) to the epididymis (g).

mean that in every case of tuberculosis of the prostate there must be manifest renal infection, nor must there necessarily be evidence of prostatic or vesicular infection in every case of tuberculous epididymitis. There are two main reasons for these apparent discrepancies. In the first place a tuberculous lesion which has passed infection on to another organ may regress or even heal while the lesion it has begotten develops and causes symptoms. In the second place a tuberculous lesion, even when it is active, may not be detectable clinically particularly in a deep-seated



FIG. 246. *Renal Tuberculosis.* Excretion pyelogram (I.V.P.) showing tuberculous of left kidney. The calices, pelvis and ureter are slightly dilated and there is a cavity communicating with one of the middle calices.

organ such as the kidney, and furthermore evidence of infection of the urine may be missed because it is intermittent. Even the relatively accessible prostate may easily hide a small lesion within its substance. The clinical picture reflects these underlying pathological events. There are two main types of cases, those presenting with urinary symptoms and those presenting with genital symptoms. In either of these two groups, detailed investigation often reveals evidence of infection of the other system. In patients presenting with epididymitis, evidence of renal infection is found in 70 per cent. As would be expected a rather smaller proportion of those presenting with urinary disease are found to have evidence of infection of the prostate, vesicle or epididymis; the figure is

53 per cent. In the female there is no direct relation between urinary and genital tuberculosis.

**Urinary Tuberculosis.** Small hematogenous tuberculous foci in the kidney usually heal, particularly those in the cortex which is the commonest site. Apart from general factors such as lowered resistance to the infection, the most likely cause of failure to heal is the occurrence of ulceration into the renal pelvis or calices. This communication with the urinary passages may occur quite early if the original lesion is submucous,



FIG. 247. *Renal Tuberculosis.* Excretion pyelogram (I.V.P.). The enlarged left kidney is extensively calcified. It is functionless. The calcified caseous material resembles putty, hence the term "putty kidney." The right pyelogram is normal, but rather lacks definition. The many other scattered shadows in the abdomen are calcified mesenteric glands. (Courtesy of Dr. J. M. Stewart.)

or later when a deeper lesion breaks down and discharges into the urine. Cavitation occurs and healing is much delayed or, more often, altogether prevented. The granulation tissue in the abscess cavity is the source of a continuous discharge of pus into the urine, and from time to time tubercle bacilli also escape. The tuberculous process may spread by direct continuity into the adjacent renal or pelvic tissue or downwards in the wall of the ureter. Groups of adjacent lesions may coalesce and form caseous masses which in turn may break down into abscess cavities (Fig. 246). Calcification quite commonly occurs (Fig. 247). The whole of one kidney may be involved by the destructive process especially if there is obstruction at the renal outlet by inflammatory swelling, and ■



tuberculous pyonephrosis may be produced. Obstruction of a part or the whole of a kidney may seal off the tuberculous process from the urinary flow causing a "closed" infection. Sometimes tuberculous activity dwindles in such a lesion and of course function of the affected renal tissue ceases; the term "autonephrectomy" has been used to describe the condition when the whole of the kidney is affected, but in fact living tubercle bacilli are immured in these kidneys from which dangerous fresh infection may arise and spread. In all cases of "open" renal tuberculosis, distant foci of infection may be set up along the banks of the urinary stream in the ureter and bladder. These distant lesions are primarily mucosal and consist of tubercles, ulcers and diffuse inflammation, but usually the underlying submucosa and muscle are involved by œdema, inflammatory infiltration and later fibrosis.

The pathological anatomy of urinary tuberculosis is determined partly by the tuberculous process itself and partly by the effects of this process on the dynamics of the urinary tract. The early effects of the infection on the smooth muscle is to hinder its contraction and so to cause dilatation. More severe effects are due to obstruction produced either by inflammatory swelling or, more permanently, by fibrosis. The effect of obstruction is twofold; it damages renal function and at the same time it weakens the power of the tissues to resist the infection so that the disease develops and spreads more rapidly. Tuberculosis has important effects on the bladder. The early mucosal infection may cause typical symptoms of cystitis, but the later inflammatory infiltration or fibrosis of the muscle may have more serious effects, particularly in and around the lower end of the ureter. Here inflammatory swelling or fibrosis may simply cause obstruction, but quite often the infiltration stiffens the walls of the intramural ureter without narrowing its lumen, and the result is to abolish its valvular action and so to allow reflux of urine up to the kidney when the bladder contracts. This has much the same deleterious effect on the kidney as ureteric obstruction. The effects of ureteric obstruction are aggravated when the bladder is small and spastic, a condition characteristic of the later stages of vesical tuberculosis. The shortening of the ureter caused by the longitudinal contraction of the fibrous tissue in its wall is sometimes very conspicuous and distorts the bladder by drawing up the ureteric orifice.

The commonest early symptom of urinary tuberculosis is *frequency of micturition*, chiefly by day but usually also by night. Most patients complain of some pain on micturition and quite commonly hæmaturia as well. These symptoms begin insidiously and last for weeks or months. They are due to cystitis, and at times are severe, with incontinence due to urgency, and strangury. There may be renal pain. Sometimes hæmaturia occurs in isolation. Tuberculosis of the urinary system seldom produces physical signs. On the other hand secondary involvement of the genital tract is common, and examination frequently reveals the charac-

teristic features in the epididymis, prostate or seminal vesicles. A large pyonephrosis may be palpable, but it is surprising what gross lesions can remain hidden in the "abdominal attic." It is common not to find any clinical signs of disease outside the genito-urinary system, although there is frequently radiological evidence of previous pulmonary tuberculous lesions, and patients with genito-urinary tuberculosis usually appear in good general health. The maximum incidence is between the ages of twenty and forty years.

*Investigation and Diagnosis.* The characteristic finding in the urine is a deposit (obtained by centrifuge) which contains pus and is sterile when cultured on ordinary media. In most cases tubercle bacilli are not quickly found, but when they are plentiful they are shown by Ziehl-Neelsen staining of an ordinary centrifuged deposit. As the bacilli are usually few a large amount of deposit is needed, and the two alternatives are an early morning or a twenty-four-hour specimen. The deposit may be stained directly, or cultured on special media, or inoculated into a guinea-pig. The latter two methods are much more likely than the first to be positive, but they take six weeks to give the result, a delay which is an unavoidable inconvenience. *Radiological* evidence may be extremely suggestive although by itself it never amounts to proof. The plain X-ray may show the rather woolly shadow of calcification in the tuberculous foci or it may show an enlarged renal outline. The *excretion pyelogram* is the most informative of all the means of investigation, particularly in showing the extent and severity of the renal disease. The earliest change is often a slight dilatation of the affected part, perhaps a solitary calix, or it may be most of the kidney together with part of the ureter (Fig. 246). Another characteristic change is a "fuzziness" of outline due to ulceration of the mucosa, and a tuberculous cavity shows as a pool of "dye" outside the normal caliceal system. A stricture of the neck of a calix or of the ureter is quite common. There is usually some diminution of concentration of the contrast medium in the affected part, and sometimes the affected kidney appears entirely functionless. Doubtful findings in the excretion pyelogram call for an *instrumental pyelogram*; however, *cystoscopy* is always needed as it often provides valuable, sometimes conclusive, evidence. There may be a cluster of tubercles around the orifice of the affected ureter or there may be ulceration. In many cases there is a diffuse and intense cystitis without specific features. In advanced disease the ureteric orifice may be drawn upwards and laterally and held rigidly open (the so-called "golf hole" ureter), but this finding, although classical, is uncommon. The bladder is often irritable and of small capacity so that a general anaesthetic may be necessary for the cystoscopy. Although the excretion pyelogram often gives very definite information about the distribution of the infection in the urinary tract, further decisive evidence is obtained by examination of the urine drained from each kidney separately by means of ureteric catheterisation. As

only a small amount of urine is obtained by this means, tubercle bacilli may not be found, and therefore greater significance attaches to the finding of pus cells. A kidney cannot be considered free of infection if the urine from it contains pus cells, even though the presence of tubercle bacilli cannot be demonstrated.

The treatment of urinary tuberculosis consists of a judicious combination of a constitutional regime, chemotherapy and surgery over a period of time lasting at least one year and often longer. The constitutional measures may include residence in a sanatorium, but they must in any case ensure adequate rest and nutrition. Chemotherapy is of the greatest importance, but opinions differ about the details of its administration. The three main drugs are streptomycin, para-amino-salicylic acid (P.A.S.) and Isoniazid, and they are used in combination, often in alternating groupings, for two important reasons. In the first place, a combination of drugs lessens the risk of the development of resistant strains of tubercle bacilli. Secondly, streptomycin, although very effective, has at least one serious drawback: it tends to form an avascular fibrous barrier around the lesions which insulates them from further attack by blood-borne therapeutic agents. Isoniazid, on the other hand, breaks down this barrier by revascularising it. A representative scheme of administration is as follows:

Streptomycin (intramuscular), 1 gm. daily	} for six weeks.
P.A.S. (oral), 5 gm. four times a day	
Isoniazid (oral), 50 mg. four times a day	} for six weeks.
P.A.S. (oral), 5 gm. four times a day	

These two courses alternate for six months. After this time smaller doses are given less frequently. The usual watch is kept for toxic effects of streptomycin.

Operative treatment is best withheld for two or three months. The state of the lesions is then reassessed by pyelography, cystoscopy and examination of the urine. Evidence of persistent infection of one kidney is usually an indication for surgery provided the other kidney is sound. The usual operation is nephrectomy, and the ureter is also removed if it is affected and if the patient's general condition warrants the extended operation. In some cases partial nephrectomy may be done particularly if a small lesion is confined to one pole. Sometimes severe renal destruction, particularly when it is associated with obstruction (pyonephrosis), necessitates the removal of the kidney at an early stage in treatment, for until it is removed there is no hope of general measures gaining the upper hand. In such cases also it is necessary and justifiable to remove the severely affected, perhaps functionless, kidney even when the other kidney also is known to be infected. Complications following operations on tuberculous kidneys have been greatly reduced by modern chemotherapy. The commonest of these complications used to be wound

infection with sinus formation and the most serious complication was generalised haematogenous ("miliary") tuberculosis.

One of the greatest problems is the treatment of residual effects in the bladder whether or not the renal condition has been satisfactorily controlled. The symptoms are painful frequency of micturition often with urgency incontinence which may make life almost intolerable. In addition there may be reflux up the remaining (sound) ureter so that the function of the kidney is threatened. Chemotherapy is usually ineffective in this condition because it may not be due to active infection; indeed in some cases the fibrosing effect of streptomycin may cause or at least aggravate bladder contraction. Surgical treatment may be the only means of relief. One of the most commonly performed operations is transplantation of the ureter into the colon, or into an isolated loop of ileum or even on to the skin (cutaneous ureterostomy). Another line of attack is to enlarge the capacity of the bladder by grafting on to it a pouch made from an isolated loop of ileum (ileocystoplasty).

**Genital Tuberculosis (Male).** The primary lesion is probably a prostatitis or vesiculitis. From these sites infection may spread along the lymphatics or in the lumen of the vas. The lymphatics happen to accompany the vas in the pelvis but part company from it at the brim of the pelvis running upwards and backwards to the lymph glands of the iliac groups. This spread is therefore entirely intra-abdominal and it produces no clinical manifestations. If infection spreads along the lumen of the vas, it is led straight to the epididymis and finds no lodgement till it gets there. A tuberculous process is set up in the lower pole of the epididymis and from here it spreads by direct extension onwards along the epididymis and backwards in the wall of the vas. The infection is slow to spread into the testis although it does so in some cases. More often tuberculous pus forms in the scrotum, behind and below the lower pole of the epididymis, where it discharges on the skin and produces a sinus.

The symptoms of genital tuberculosis are usually insignificant. Prostatovesiculitis may cause some pain on sexual intercourse but is often symptomless. Epididymitis sometimes has an acute onset, and then there may be severe pain and tenderness both in the epididymis and in the groin. More often the symptoms are very mild even when a sinus is forming. In contrast with the symptoms, the *physical signs* are usually obvious and characteristic. On rectal examination the affected part of the prostate feels hard and irregular and the seminal vesicle may be enlarged and indurated. In the differential diagnosis of the findings *per rectum*, several fairly common possibilities have to be distinguished. In non-specific prostatitis the induration is less localised and not so hard, but prostatic calculi may feel very like a tuberculous focus; an X-ray usually settles the point. Carcinoma of the prostate occurs mainly in an older age group, and in many cases shows the characteristic lack of definition of the prostate due to lateral and upward infiltration of the tumour.

only a small amount of urine is obtained by this means, tubercle bacilli may not be found, and therefore greater significance attaches to the finding of pus cells. A kidney cannot be considered free of infection if the urine from it contains pus cells, even though the presence of tubercle bacilli cannot be demonstrated.

The treatment of urinary tuberculosis consists of a judicious combination of a constitutional regime, chemotherapy and surgery over a period of time lasting at least one year and often longer. The constitutional measures may include residence in a sanatorium, but they must in any case ensure adequate rest and nutrition. Chemotherapy is of the greatest importance, but opinions differ about the details of its administration. The three main drugs are streptomycin, para-amino-salicylic acid (P.A.S.) and Isoniazid, and they are used in combination, often in alternating groupings, for two important reasons. In the first place, a combination of drugs lessens the risk of the development of resistant strains of tubercle bacilli. Secondly, streptomycin, although very effective, has at least one serious drawback: it tends to form an avascular fibrous barrier around the lesions which insulates them from further attack by blood-borne therapeutic agents. Isoniazid, on the other hand, breaks down this barrier by revascularising it. A representative scheme of administration is as follows:

Streptomycin (intramuscular), 1 gm. daily	}	for six weeks.
P.A.S. (oral), 5 gm. four times a day		
Isoniazid (oral), 50 mg. four times a day	}	for six weeks.
P.A.S. (oral), 5 gm. four times a day		

These two courses alternate for six months. After this time smaller doses are given less frequently. The usual watch is kept for toxic effects of streptomycin.

Operative treatment is best withheld for two or three months. The state of the lesions is then reassessed by pyelography, cystoscopy and examination of the urine. Evidence of persistent infection of one kidney is usually an indication for surgery provided the other kidney is sound. The usual operation is nephrectomy, and the ureter is also removed if it is affected and if the patient's general condition warrants the extended operation. In some cases partial nephrectomy may be done particularly if a small lesion is confined to one pole. Sometimes severe renal destruction, particularly when it is associated with obstruction (pyonephrosis), necessitates the removal of the kidney at an early stage in treatment, for until it is removed there is no hope of general measures gaining the upper hand. In such cases also it is necessary and justifiable to remove the severely affected, perhaps functionless, kidney even when the other kidney also is known to be infected. Complications following operations on tuberculous kidneys have been greatly reduced by modern chemotherapy. The commonest of these complications used to be wound

**SECTION IV**  
**THORACIC SURGERY**  
by  
**GEOFFREY FLAVELL**

**CHAPTER 33**  
**BRONCHOSCOPY**

THE chest surgeon must approach his patient bronchoscope in hand ; it is to him as the cystoscope is to the urologist, and there is almost no pulmonary disease about which it does not yield valuable information, for it is the only direct means of viewing the interior of the bronchial tree. Except in infancy, bronchoscopy can nearly always be carried out under local anæsthesia, and, when necessary, on a patient in bed or in the consulting room. There is no age limit from the suckling to the senile, and even in the very ill it remains practicable and very often life saving. The only contraindication to its use is the recent ingestion of a meal, and this is not absolute.

**Technique.** Preferably some four hours should have elapsed since the last meal, but it is well to remember that in small children food often remains undigested in the stomach for much longer periods if the child is frightened and ill, and may be inconveniently regurgitated when least expected. When a patient is anæsthetised, either generally, or locally to the pharynx, such vomit may be aspirated by a sudden gasp into the bronchial tree and cause death by drowning, or by impaction of solid fragments. Such an emergency calls in itself for bronchoscopy and vigorous suction to clear the airways ; solids sometimes have to be removed with forceps. Accidents of this kind are common enough so it is all the more important not to allow them to occur during bronchoscopy, when instrumentation of the pharynx may promote gagging and retching, and an anæsthetised glottis render aspiration easy. For this reason then, the stomach should be empty ; anæsthesia efficient ; instrumentation gentle and practised ; suction powerful, and sucker tubes wide and quickly interchangeable ; and facilities exist for rapidly lowering the patient's head should it be necessary. If these conditions are observed the dangers against which they guard will not occur. A cocaine sensitivity test should be carried out at some prior time. It consists of an intradermal injection of  $\frac{1}{12}$  gr. of cocaine hydrochloride with subsequent pulse readings at ten-minute intervals over an hour. If no significant rise takes place, no

Tuberculous epididymitis often takes a very typical form. It affects the epididymis mainly at its lower pole which becomes moderately enlarged, hard, irregular and angular. The traditional term craggy is very apt. When the skin of the scrotum is involved or when there is actually a sinus, the diagnosis is hardly in doubt. The sinus leads to the epididymis, usually the lower pole, and for this reason its opening is often on the posterior aspect of the scrotum ; but the enlarged lower pole of the epididymis juts forwards under the testis so that a sinus may run from it to the front of the scrotum. The important thing is that the epididymis is the source of the sinus, whereas when *acute epididymitis* suppurates and discharges, the sinus is likely to lead to the disorganised body of the testis. In tuberculous epididymitis the lower end of the vas, where it runs up from the epididymis, may be thickened. Very rarely there may be isolated thickenings along the course of the vas. Sometimes chronic non-specific epididymitis may give findings very similar to those of tuberculosis, and in these cases the findings *per rectum* may be very helpful in settling the diagnosis because tuberculous prostatitis and vesiculitis (the precursors of the epididymitis) usually feel very characteristic, and the absence of any such findings points away from a diagnosis of tuberculous epididymitis. Once tuberculosis of the *genital* system has been diagnosed, a full investigation must be made to exclude infection of the *urinary* system.

*Treatment.* The general treatment and the chemotherapy of genital and urinary tuberculosis are similar. The operations that may be done are *epididymectomy*, *orchidectomy* and *vasotomy*. Operations on the prostate or vesicles are extremely seldom done for tuberculosis. Epididymectomy is the usual operation and the results are very satisfactory. It may easily be combined with excision of a scrotal sinus. Rarely, the testis itself is involved and orchidectomy must be done. Neither of these operations removes the immediate source from which infection reached the scrotal organs through the vas deferens ; further infection may therefore occur at the point where the vas is divided, or in the other epididymitis if the operation has been confined to one side. The effects of infection at the cut end of the vas may be minimised by exteriorising the stump (vasostomy). Infection of the other side may be prevented by vasotomy but this will, of course, be followed by permanent sterility. The place of operation in genital tuberculosis has not yet been redefined since effective chemotherapy has been available. There is no doubt that even advanced local disease with sinus formation may be arrested by chemotherapy alone. Operation is however still the standard treatment, but it is probable that the indications for it will become more restricted.

placed over the upper incisors or gum, and the middle and ring fingers of the surgeon's left hand rest gently upon it. The bronchoscope rests in turn upon the fingers and thus its weight is never allowed to fall upon the teeth or gums of the patient. The bronchoscopist's left index and thumb help to control the movements of the instrument. At this stage the "beak" of the instrument is placed anteriorly, at "twelve o'clock," and the back of the tongue is lifted forward with it. At "six o'clock" the uvula is visible. As the tongue is lifted, the epiglottis comes into view, usually lying in contact with the posterior pharyngeal wall, and must always be identified. It is in turn gently lifted forward by the beak of the instrument, which should not at this stage be too far advanced. The glottis is then seen lying anteriorly, with the two arytenoid cartilages behind it. The vocal cords are inspected and their movement checked by asking the patient to say "ee." Lag, or paresis, indicate a lesion of the recurrent laryngeal nerve. The left nerve, because of its special anatomical relations, is commonly involved in tumours in the thorax and may also be paralysed by aneurysms of the aortic arch.

The bronchoscope is now rotated through 90 degrees so that the beak is in line with the glottic fissure, and while the patient breathes quietly and steadily it is then passed down between the vocal cords into the trachea. This, like the rest of the bronchial tree, lengthens on inspiration and shortens on expiration; it also expands and contracts with each breath. In addition, there is some mobility of the trachea within the mediastinal tissues (limited upon the left by the aortic arch) and it can be appreciated during bronchoscopy. Absence or reduction of any of these normal movements is of significance and may be due to an extra mural mass or some other lesion. Similarly, indentation or deformity of the wall of the trachea is looked for, and, lastly, the mucosa is examined for abnormalities, e.g. the hyperæmia, ulceration, scarring, or actual tubercles of endotracheal tuberculosis, or the presence of neoplastic infiltration, ulcers or tumours.

The bronchoscope advances down the trachea to the bifurcation at the *carina*, the keel-like edge dividing the right stem bronchus from the left (Figs. 249 and 250a). As the former more nearly continues the axis of the trachea its lumen is almost in full view, but the latter, departing from the trachea at a more acute angle, is only half seen (Fig. 250a), and to enter it requires flexion of the patient's head to the opposite side and some elevation of the head-rest. Should the *carina* be widened at its base, and shortened, the deformity is probably due to enlargement of the lymph glands that lie at the bifurcation. Hence in cases of bronchial carcinoma, this sign indicates involvement of these glands and consequent inoperability.

The right main bronchus is short, and after about 1 inch its first branch, the bronchus to the upper lobe, is seen opening from it at "three o'clock." To obtain a further view, therefore, into this orifice a right-angled tele-



abnormal sensitivity exists. In adults, premedication may be varied with the temperament of the patient and is not essential; nervous subjects should be given Omnopon  $\frac{1}{3}$  gr. and hyosine  $\frac{1}{60}$  gr. an hour before operation. Half an hour before, a 50-mg. Decicain (amethocaine, B.P.) lozenge is sucked on the back of the tongue. On arrival in the theatre the patient is asked to sit up and extend the neck. After the overlying skin has been sterilised the cricothyroid membrane is felt, and a fine intramuscular needle passed through it into the tracheal lumen. The patient's body is then inclined to the side of particular interest, if this is known, and 1 ml. of 10 per cent. cocaine injected into the trachea; the needle is withdrawn, and the patient asked to cough in order to distribute the cocaine. A pledget of ribbon gauze, grasped between the teeth of a Krauss' forceps, is now dipped in 10 per cent. cocaine, the surplus squeezed out, and the pledget passed over the back of the tongue down into the pyriform fossa where it is held for some thirty seconds. One such application only is used on each side.

All the cocaine used (it need never exceed 2 ml.) is carefully measured from a graduated glass. The back of the throat should never be sprayed with cocaine for accurate measurement is then impossible.

Some bronchoscopists prefer to use one or other of the proprietary cocaine derivatives such as 2 per cent. Butyn or 4 per cent. Xylocaine, on the grounds that their toxicity is less than that of cocaine. Whereas this is true to some extent, most patients who have experienced these alternative drugs as well as cocaine prefer the latter. Cocaine shares with pentothal the position of being the most dangerous drugs in common use. Cocaine sensitivity is exceedingly rare, but cocaine poisoning by overdose is appallingly frequent. Cocaine derivatives are equally lethal, but they tend to give anaesthetists a feeling of false security. Meticulous measurement and accurate dosage of all these drugs are absolutely essential if accidents are to be avoided.

The patient, now ready for bronchoscopy, is placed upon the table with the occiput resting in the supporting cup of the head-rest and the hair and eyes covered by a towel. The neck is somewhat extended on the trunk and the head upon the neck, so that the chin is thrown back towards the operator who is seated behind.

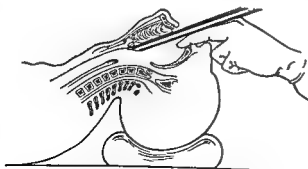


FIG. 248. The chin is thrown upwards and backwards to bring mouth, glottis, and trachea into line. The back of the bronchoscope lifts the epiglottis forward and the fingers of the left hand protect the patient's teeth and lips.

Thus is assured the posture (likened to that of a barking dog) which best brings mouth, fauces, glottis and trachea into line (Fig. 248). A swab is

## BRONCHOSCOPY

lobe is usually visible at "nine o'clock" (Fig. 250c). If of the three basal segments, anterior, lateral and posterior, although not simultaneously (Fig. 250c). With excursion each comes into view in turn.

All of these orifices must be identified individually and confirmed. As with the trachea, so with these, distort

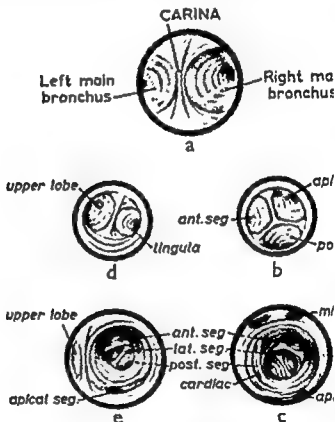


FIG. 250. Progressive bronchoscopic views of the primary division of the bronchial tree.

occlusion of the lumen, or changes in the mucosa are not blood, or pus, exuding from any branches are inspected for further examination. By employing the bronchoscope as an extension of one's finger some idea of the wall may be obtained, and a more direct view into the lumen. If abnormal tissue is encountered, forward-looking or retrograde are used for close visual examination and biopsy is performed.

On the left side a similar course is followed. The left main bronchus is much longer than the right, being some 1 1/2 inches from the trachea. The upper lobe branch at "nine o'clock" (Figs. 249 and 250)

scope is needed. With its aid the lumen of the bronchus can be inspected, and the three branches (anterior, posterior, and apical) identified (Fig. 250b). Because of the short length of the right main bronchus, it follows that a malignant growth in the right upper lobe bronchus rapidly becomes inoperable, for enough room must exist to amputate the involved lung through a healthy part of the main bronchus. It is not uncommon to

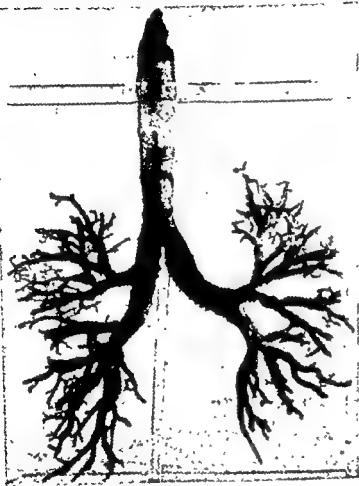


FIG. 249. A cast of the bronchial tree viewed from in front. The cast is supported at the carina and the differences between right and left main bronchi are clearly shown. Note also the angle at which the upper lobe bronchi leave the main stems.

find the apical part of the upper lobe communicating with the side of the trachea just above the carina by a separate small bronchus.

Continuing the inspection of the right bronchial tree, the next orifice seen is that to the middle lobe lying anteriorly at "twelve o'clock" (Fig. 250c), and separated from the stem bronchus to the lower lobe by a nearly horizontal carina. As an almost direct view into it can be obtained, it is usually possible also to see its division into medial and lateral branches. Just below it, a little further down the stem and arising at "five o'clock," is the first segmental branch of the lower lobe, that to the apex, or "dorsal" segment (Fig. 250c). It lies in the posterior bronchial wall like a golf hole in a putting green; an angulated telescope is needed to view it directly. Beyond this point the branch to the cardiac segment of the lower

## BRONCHOSCOPY

lobe is usually visible at "nine o'clock" (Fig. 250c). First of the three basal segments, anterior, lateral and posterior directly, although not simultaneously (Fig. 250e). With excursion each comes into view in turn.

All of these orifices must be identified individually and confirmed. As with the trachea, so with these, direct

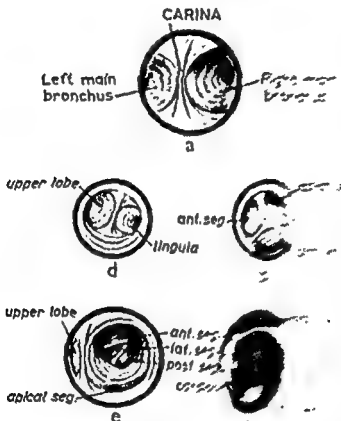


FIG. 250. Progressive bronchoscopy.

short distance into the lumen of this branch (Fig. 250d), and its lingula segmental opening can be identified. Thereafter a right-angled telescope is again required. From this point on, the bronchus to the apical segment of the lower lobe appears on the posterior wall of the stem, at "six o'clock" (Fig. 250e), and finally the three basal segments—*anterior, lateral, and posterior*—of the lower lobe come into view (Fig. 250e).

When a complete examination has thus been made of both right and left bronchial trees, and all the primary divisions have been identified and inspected to the limit of vision, the bronchoscope is withdrawn to the trachea; observations are checked on the way, and finally the instrument is removed altogether in an upwards and forwards sweep. As the glottis remains anæsthetic for some time, the patient must not eat or drink for another hour and a half until the effect of the cocaine has worn off and water can be sipped without spluttering.

The bronchoscope is used diagnostically and therapeutically. In diagnosis it provides the only direct access to lesions of the bronchial tree, whereby their character can be estimated, secretions from them viewed, aspirated and examined, and the location and extent of lesions accurately decided and delimited. Impairment of mobility, function and aeration of a bronchial segment becomes evident. Sources of bleeding or of suppuration, changes in the state of the mucosa (extramural, mural or intramural) may be observed. No investigation of chest disease is complete without bronchoscopy; its omission in the examination of a patient suspected of bronchial carcinoma amounts to culpable negligence. Therapeutically, the value of bronchoscopy is also great, though somewhat more restricted.

**Foreign Bodies.** These are commonly aspirated into the bronchial tree, especially by children and idiots, who are apt to insert small objects such as peas, marbles or studs into their nostrils and so inhale them (Fig. 251). Coins, pins and loose teeth may find their way through the glottis from the mouth. The practice of holding small screws, tacks, or bolts in the mouth while working is dangerous. The symptoms produced by inhalation vary with the size and character of the object. When large, it may become impacted in the glottis or in the trachea. Complete respiratory arrest and rapid death may ensue if it is not promptly removed by bronchoscopy. In such circumstances the patient should never be turned upside down in the hope of the foreign body being coughed up, for if it is lying in the trachea or one of the main bronchi, it will then be dislodged and become impacted against the under surface of the glottis, converting a partial obstruction into a complete one with fatal consequences. A coin in the trachea usually lies in the sagittal plane, for it has to adopt this position to pass the glottis. Whereas if it is in the *œsophagus* it lies transversely. This difference is useful in the interpretation of radiographs.

If foreign bodies pass further they cause partial or complete obstruction of the bronchus in which they are arrested, and frequently carry with them

sepsis, the effects of which vary with the virulence of the organisms. Extracted teeth or dental roots and fragments of tonsil or adenoid tissue are likely to be very septic and the consequent infection is acute and serious; if not swiftly removed they will rapidly produce, first, segmental collapse of the lung distal to their impaction, and, secondly, an acute

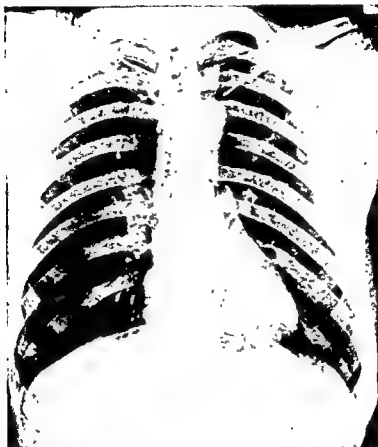


FIG. 251. Two buttons, a tin tack, and a small screw all inhaled from the nostril into an idiot's lung.

putrid lung abscess in the collapsed segment (Fig. 252). If the infection is more indolent and the obstruction less complete, the presence of the foreign body may remain unsuspected for a long time. The fact that an object has been inhaled is not always realised, even by a conscious adult. A fit of choking over a hurriedly gulped meal, or coughing while working with tacks held in the mouth, is sometimes followed by no immediate symptoms, until months or even years later when purulent expectoration leads to a diagnosis of bronchiectasis. In such circumstances, X-ray examination does not always reveal the foreign body, for chicken or rabbit bones may not be visible among the lung markings, and even

short distance into the lumen of this branch (Fig. 250d), and its lingula segmental opening can be identified. Thereafter a right-angled telescope is again required. From this point on, the bronchus to the apical segment of the lower lobe appears on the posterior wall of the stem, at "six o'clock" (Fig. 250e), and finally the three basal segments—*anterior, lateral, and posterior*—of the lower lobe come into view (Fig. 250e).

When a complete examination has thus been made of both right and left bronchial trees, and all the primary divisions have been identified and inspected to the limit of vision, the bronchoscope is withdrawn to the trachea; observations are checked on the way, and finally the instrument is removed altogether in an upwards and forwards sweep. As the glottis remains anaesthetic for some time, the patient must not eat or drink for another hour and a half until the effect of the cocaine has worn off and water can be sipped without spluttering.

The bronchoscope is used diagnostically and therapeutically. In diagnosis it provides the only direct access to lesions of the bronchial tree, whereby their character can be estimated, secretions from them viewed, aspirated and examined, and the location and extent of lesions accurately decided and delimited. Impairment of mobility, function and aeration of a bronchial segment becomes evident. Sources of bleeding or of suppuration, changes in the state of the mucosa (*extramural, mural or intramural*) may be observed. No investigation of chest disease is complete without bronchoscopy; its omission in the examination of a patient suspected of bronchial carcinoma amounts to culpable negligence. Therapeutically, the value of bronchoscopy is also great, though somewhat more restricted.

**Foreign Bodies.** These are commonly aspirated into the bronchial tree, especially by children and idiots, who are apt to insert small objects such as peas, marbles or studs into their nostrils and so inhale them (Fig. 251). Coins, pins and loose teeth may find their way through the glottis from the mouth. The practice of holding small screws, tacks, or bolts in the mouth while working is dangerous. The symptoms produced by inhalation vary with the size and character of the object. When large, it may become impacted in the glottis or in the trachea. Complete respiratory arrest and rapid death may ensue if it is not promptly removed by bronchoscopy. In such circumstances the patient should never be turned upside down in the hope of the foreign body being coughed up, for if it is lying in the trachea or one of the main bronchi, it will then be dislodged and become impacted against the under surface of the glottis, converting a partial obstruction into a complete one with fatal consequences. A coin in the trachea usually lies in the sagittal plane, for it has to adopt this position to pass the glottis. Whereas if it is in the oesophagus it lies transversely. This difference is useful in the interpretation of radiographs. If foreign bodies pass further they cause partial or complete obstruction of the bronchus in which they are arrested, and frequently carry with them

postero-anterior and lateral X-rays, and if possible its character, shape, and position within the bronchus determined. The most suitable instrument for its removal is selected, and bronchoscopy is carried out under the best possible conditions, for extraction at the first attempt is likely to be much easier than at a second. In the clinic of Chevalier Jackson it is customary to reduplicate the object and its position within the hose-pipe bronchus of a dummy man so that a complete plan of campaign may be worked out, and if necessary a suitable instrument made or adapted.

Conical bodies such as teeth are easily driven further down a bronchus



FIG. 253. (a) Chest during inspiration. (b) Chest during expiration. The diaphragm on the right side does not rise. There is also displacement of mediastinum to left.

if insecurely grasped at their presenting ends with forceps, and it is better to pass an instrument beyond them and hook them back from their point of impaction. If an object has been in position a long time, oedema of the mucosa or granulations proximal to it may not only constrict the lumen but almost conceal it, and attempts at removal are doomed to failure unless the swelling is first passed and pressed back by expanding forceps. Pins, especially open safety pins, must be removed with especial care lest they pierce the bronchial wall during the process and lead to pneumothorax and possibly an empyema. If the point is directed upwards, it must be seized and drawn into the lumen of the bronchoscope; if downwards, the whole pin is extracted with the point trailing; and if in an intermediate position it must first be manoeuvred into one of the other two positions before removal is begun. Many foreign bodies are too



relatively dense objects like teeth may be remarkably hard to see. Peanuts are often inhaled by young children (to whom they ought never to be given) and may produce no radiological changes. X-rays in inspiration and expiration must always be taken, for if partial obstruction is present, air may enter the lung as the bronchi distend on inspiration, but be unable to escape as they contract down again during expiration. This will result



FIG. 252. Tooth impacted in posterior basal segmental bronchus causing collapse.

in *obstructive emphysema*. Radiologically, as the air is expelled, the mediastinum is displaced to the unaffected side and the diaphragm on the side of the blocked bronchus is unable to rise (Fig. 253).

Mothers are frequently mistaken in their belief that their child has "breathed something in," but even in the absence of physical or radiological signs, if inhalation of a peanut is suspected, it is safer to proceed with bronchoscopy rather than run the risk of later impaction and bronchial obstruction, and consequent pulmonary abscess or bronchiectasis, which in turn would eventually lead to resection of a lobe.

When radio-opaque, a foreign body's exact location is first plotted by

fourth post-operative day, and, in fact, at this time collapse of the residual lobe or segments is most common. In such post-operative patients, and in those suffering from pulmonary oedema or congestive heart failure, the passage of a bronchoscope while the patient is in bed is often convenient, provided that really efficient suction is always available. The patient simply reclines backward on the bed-rest or pillows, the head is extended and supported by an assistant, while the bronchoscopist stands behind the head of the bed on a stool. The bronchoscope drops directly down the trachea, rather like a sword down the gullet of a professional swallower. The plug is located and extracted—if necessary with forceps—and vigorous suction applied to the dammed-up secretions beyond. It is a good plan, while the sucker is in the affected lobe, to ask the patient to cough and so ensure complete clearance. A finer sucker-end, soft-tipped, can also be passed on into the smaller bronchi. The result of the treatment is the prompt re-expansion of the collapsed lung, and the patient is saved from possible lung abscess, empyema, or later bronchiectasis. The diagnosis of "*post-operative pneumonia*" is unsatisfactory, and the fact that, thanks to antibiotics, complications seldom occur must not excuse the withholding of bronchoscopy and aspiration.

**Other Indications for Bronchoscopy.** Patients who are becoming inundated with their bronchial secretions, due to pulmonary oedema, as in cardiac failure, can often be dramatically relieved by bronchoscopy. Such subjects are never made worse by bronchoscopy, and it is indefensible, by inactivity, to condemn them to slow death by drowning.

Aspiration by bronchoscopy of pus discharged from a lung abscess or from a bronchiectatic area is sometimes carried out as a therapeutic measure. But in both conditions the value of bronchoscopy is chiefly diagnostic, although it is also of importance in eliminating any obstruction to free drainage. Once this is established both diseases are treated by other means (excision of the affected areas, or conservative measures such as postural drainage).

The practice of insufflating antibiotic powders into an infected part of the lung by means of a bronchoscope is both ineffectual and unnecessary, and, furthermore, it is dangerous, for the powder itself may act as a bronchial embolus.

Strictures of the bronchus are seldom satisfactorily treated by bronchoscopic means. Compression of a child's bronchus causing collapse, or, less often, obstructive emphysema, is most commonly caused by enlarged tuberculous glands. Therefore attempts to dilate such a stricture are futile, for as soon as the dilators are removed the pliable bronchial wall collapses again. Occasionally, however, some respite may be obtained. Intrinsic strictures caused by endobronchial tuberculosis in older patients, are indications for resection since they are always associated with permanent pulmonary damage distal to the point of stricture. As a purely

large, or of an unsuitable shape, to be drawn up the bronchoscope, so they must be firmly secured with forceps and the whole entity, bronchoscope, forceps and foreign body, removed in one clean sweep; the object is held in close apposition to the bronchoscope beak and follows it through the glottis in an "after-coming" position.

When impaction of a small foreign body occurs in a distal bronchus it may be quite beyond the reach of a normal bronchoscope (see Fig. 251). Chevalier Jackson has designed special slender instruments for such occasions, but much experience and skill are needed for their use; they are not usually available when required. Since such impactions necessarily affect a much smaller segment of the lung their consequences are not as a rule so serious. After accurate localisation they can be extracted at thoractomy by incision of a narrow segment of pulmonary tissue, for their position is always peripheral.

**Atelectasis.** When this condition is caused by accumulation of natural secretions it can be relieved by means of bronchoscopy. Following any operations, but especially those involving laparotomy or thoractomy, the patient finds it painful to cough; in addition, during the period of anaesthesia, the cough reflex itself has been abolished, and may remain so for varying periods post-operatively. Hence mucus is not expelled in the normal fashion, but accumulates in the bronchial tree until it causes bronchial obstruction with consequent collapse of the lung distal to the block. The bad old practice of keeping herniotomy patients lying flat on their backs for weeks on end notoriously promoted chest complications, most of which were labelled "hypostatic pneumonia" but were in fact one degree or another of pulmonary collapse; the same can be said of chest complications following upper abdominal operations, especially partial gastrectomy. When dehydration is added to the other factors the mucus becomes tenacious and so more difficult to expel. Indeed it is common to find the actual mucoid plug in the blocked bronchus so tough and dry that it is no longer possible to suck it out with the widest sucker tube; it therefore has to be extracted with biopsy or grasping forceps. Such plugs are rubbery and gelatinous, and can be rolled between the fingers. Their expulsion by coughing is inconceivable, and since the segment or lobe of lung beyond them has already become airless and collapsed, no "tussive blast" exists to dislodge them. On the contrary, a negative pressure drag is established which tends only to suck them further and further distally in the bronchus and make their removal hourly more difficult. It is thus evident that bronchoscopy is not only the sole efficient treatment in such cases, but its use is a matter of urgency as soon as the diagnosis has been made. In chest surgery, such emergencies are common after lobectomy or segmental resection of the lung, for all the above-mentioned causal factors are likely to exist, and added to them is the trauma to the bronchus at the point of amputation and the accompanying oedema. The latter is at its worst on about the third or

## CHAPTER 34

### LUNG ABSCESS

**The "Malignant" Abscess.** By far the commonest variety of lung abscess is the "malignant" one, and every abscess or empyema seen in a man of forty-five or more should be assumed to be due to an underlying carcinoma until its existence is disproved. When an abscess of apparently pyogenic origin fails to respond promptly to adequate treatment, malignancy should be suspected. Every adult patient suffering from a lung abscess, of whatever variety, must be bronchoscoped, and both bronchial trees carefully inspected for a possible growth. An unflinching observance of these rules will save many lives.

Bronchial carcinomas produce lung abscesses in three ways: firstly the growth itself may become necrotic and break down at its centre to form an abscess. When this occurs, and it is by far the commonest type of malignant abscess, the growth involved is always of the squamous-celled type. The abscess itself has a characteristically thick wall which is irregular and bosselated, and the cavity is often eccentrically placed within the surrounding X-ray opacity. This is due to projections of the neoplasm into the area of necrosis, and contrasts sharply with the rather regular wall of an ordinary acute putrid abscess and with the second type of malignant abscess due to obstruction (not to neoplastic necrosis) (Fig. 254).

Squamous carcinoma cells are almost always to be found in the sputum of such patients, for an abscess recognizable radiologically is usually in communication with a bronchus. Bronchoscopy often confirms the existence of a growth.

The second type of abscess associated with carcinoma is much less characteristic; it is due to bronchial obstruction by growth and consequent pyogenic infection in the distal segment of lung. The abscess itself therefore differs in no way from those following other forms of obstruction (e.g. by foreign bodies, or innocent neoplasms such as adenomas), and its walls therefore show none of the irregularity seen in the first type (Fig. 255). It may be possible, however, to distinguish an opacity produced by the growth proximal to the cavity. The causal neoplasm in this type of abscess may be of any histological type. Such a shadow is not always to be seen, and only bronchoscopy will ensure correct diagnosis.

The third type is the least common of the three, and its malignant source most often missed, for the abscess may occur in lobes unrelated to the growth or even in the opposite lung. It results from bronchial emboli, composed of detached particles of growth itself, fragments of

palliative measure, when surgery is for some other reason contraindicated, dilatation may be justified, but should never be attempted if active disease of the mucosa is still present.

Cauterisation of tuberculous ulcers gives poor results, for the ulcers indicate widespread infection of the submucosal layers, and cautery applications are more likely to be followed by strictures than by healing. Strictures due to bronchial adenomas were at one time usually treated by this method, often with much relief, but if the condition of the patient permits, pulmonary resection is now always to be preferred. Obstruction by inoperable bronchial carcinomas was likewise treated by implantation into them of radon seeds at bronchoscopy, or insertion of a Tudor Edwards radon box. The results were disappointing and the practice has been abandoned.

In the administration of general anaesthesia for chest operations bronchoscopy plays an important part. For many resections, either lobectomy or pneumonectomy, it is desirable to block off the portion of the bronchial tree to be amputated, by the accurate placing of an endo-bronchial balloon or blocker. The anaesthetist puts a blocker in the exact position required by means of bronchoscopy and subsequently inflates it so that it firmly keeps its place; the bronchoscope is then withdrawn: A sucker passes through the blocker itself so that the bronchus distal to it can be kept aspirated. When the operation is concluded, the bronchoscope is again passed, to inspect the whole of the remaining bronchial tree and clear it of any blood or secretions that may have spilled over into it during and after the operation.

suppuration. In men of forty-five or more the presence of a carcinoma is assumed until disproved. When it is known that an abscess or an empyema is caused by a growth, surgical drainage is if possible avoided, for neither of these conditions implies inoperability of the neoplasm for which complete extirpation is the goal, and furthermore, if the cancer is inoperable, every effort must be made to spare the patient the further



FIG. 255. An abscess distal to a small growth obstructing the left lower lobe bronchus. Note the fluid level indicating communication with the bronchus.

suffering and discomfort caused by the presence of a drainage tube—inevitably permanent and productive of a constant purulent discharge requiring daily dressings.

**The Aspiration, or Acute Putrid Lung Abscess.** It is under one of these titles that the syndrome of the classical "lung abscess" is usually described; but it must be borne in mind that any suppurating lung cavity whatever its origin may become foetid if infected by the anaerobes from which the offensive sputum associated with these abscesses originates. An acute abscess is occasionally free from these organisms and consequently does not stink.

blood clot, or pus from an area of suppuration related to the neoplasm. Such emboli, instead of being coughed up, are sometimes aspirated into another part of the lung, producing an abscess in the same way as aspirated infected matter from the mouth. The resulting abscess is therefore most likely to appear in one of the segments favoured by posture. It is clear that unless bronchoscopic examination is systematic, and includes the



FIG. 254. A squamous-celled carcinoma breaking down to form a typically irregular cavity. This is easily the commonest type of lung abscess.

whole lung, not just the lobe affected by the abscess, the cancer, if not seen radiologically, may be missed, especially as treatment with antibiotics will probably result in prompt clearance of the suppuration. The patient is then sent home "cured." Only by routine conscientious bronchoscopy will such tragedies be averted.

In all three kinds of malignant abscess, the manifestations of suppuration may quite obscure those due to the growth and the patient may present with fever and purulent sputum. Clearly, the possibility of a carcinoma must *always* be kept in mind in dealing with intrathoracic

suppuration. In men of forty-five or more the presence of a carcinoma is assumed until disproved. When it is known that an abscess or an empyema is caused by a growth, surgical drainage is if possible avoided, for neither of these conditions implies inoperability of the neoplasm for which complete extirpation is the goal, and furthermore, if the cancer is inoperable, every effort must be made to spare the patient the further



FIG. 255. An abscess distal to a small growth obstructing the left lower lobe bronchus. Note the fluid level indicating communication with the bronchus.

suffering and discomfort caused by the presence of a drainage tube—inevitably permanent and productive of a constant purulent discharge requiring daily dressings.

**The Aspiration, or Acute Putrid Lung Abscess.** It is under one of these titles that the syndrome of the classical "lung abscess" is usually described; but it must be borne in mind that any suppurating lung cavity whatever its origin may become foetid if infected by the anaerobes from which the offensive sputum associated with these abscesses originates. *An acute abscess is occasionally free from these organisms and consequently does not stink.*



blood clot, or pus from an area of suppuration related to the neoplasm. Such emboli, instead of being coughed up, are sometimes aspirated into another part of the lung, producing an abscess in the same way as aspirated infected matter from the mouth. The resulting abscess is therefore most likely to appear in one of the segments favoured by posture. It is clear that unless bronchoscopic examination is systematic, and includes the



FIG. 254. A squamous-celled carcinoma breaking down to form a typically irregular cavity. This is easily the commonest type of lung abscess.

whole lung, not just the lobe affected by the abscess, the cancer, if not seen radiologically, may be missed, especially as treatment with antibiotics will probably result in prompt clearance of the suppuration. The patient is then sent home "cured." Only by routine conscientious bronchoscopy will such tragedies be averted.

In all three kinds of malignant abscess, the manifestations of suppuration may quite obscure those due to the growth and the patient may present with fever and purulent sputum. Clearly, the possibility of a carcinoma must *always* be kept in mind in dealing with intrathoracic

The amount of lung involved depends both on the size of the embolus and the bronchial division blocked; but is most often a segment or sub-segment. Consequently the subsequent atelectasis is wedge-shaped with the base of the wedge on the pleural surface of the segment. It is through this surface that surgical access to an abscess can always be obtained. The collapsed segment is invaded by the organisms accompanying the



FIG. 236. An aspiration abscess in the apical segment of the right lower lobe. It has not yet ruptured into a bronchus and therefore shows no fluid level.

embolus, and as these often originate in the mouth, they are likely to include fusiform bacilli, spirochaetes and anaerobic streptococci. Coliform bacilli are commonly present, but this by no means implies that the chest infection is derived from the bowel or extension of a subphrenic abscess. The resulting area of intense pneumonitis soon undergoes suppuration, necrosis and sloughing to form an abscess cavity filled with foul pus.

The onset of an acute putrid lung abscess is generally marked by a rigor and afterwards by pleural pain and a rise of temperature. As we have seen, it often follows an operation, or loss of consciousness for some

Blood, pus or septic material inhaled into the bronchial tree is normally expelled from it by coughing and by ciliary action. When, however, the cough reflex is abolished or seriously hampered, the inhaled matter becomes lodged somewhere in a bronchus, blocks it, and causes collapse of the corresponding segment. Conditions favouring aspiration of septic material exist during operations on the upper respiratory tract under general anaesthesia. Thus during tonsillectomy, adenoidectomy and tooth extractions there is an abundant supply of very septic material. Teeth extractions, particularly when multiple, and performed with patients sitting in a dental chair, are especially dangerous; and it is in this type of operation that the standard of anaesthesia is apt to be unsatisfactory. During surgical anaesthesia the cough reflex is absent. Thus if nasal or oral infection is present, septic material may find its way into the trachea or be carried by intratracheal tubes passed through the nose or mouth. In 318 abscesses not due to malignancy, Brock found sixty-eight (21.4 per cent.) to be post-operative, and twenty-two of them followed dental extractions. Stern, in 115 cases, attributes 16 per cent. to tonsillectomy, 11.3 per cent. to dentistry, but notes that 84 per cent. of the whole group had bad teeth and infected gums. A similar relationship exists between dental sepsis and chronic suppurative pneumonitis. Other circumstances which may lead to aspiration of septic matter and interference with the cough mechanism are acute alcoholism, epilepsy and partial drowning. The importance of the first is difficult to assess; but when a history of recent operation is lacking alcoholism should always be thought of, whatever the social position or sex of the patient, for impaired consciousness may lead to aspiration of vomit. Neither anaesthesia nor unconsciousness from other causes are invariable prerequisites, for any foreign body occluding a bronchus produces distal collapse and hence renders coughing ineffective. General debility and low resistance to infection favour the development of lung abscess. A primary cause for the abscess probably always exists, although in about a third of the cases it is not possible to identify it with certainty.

Foreign material entering the bronchial tree gravitates according to the posture of the patient at the moment of inhalation, and also according to the anatomy (Fig. 249). Thus the right stem bronchus, being more in line with the trachea than the left, receives aspirated material more often than the left. If the patient is lying on his side, the upper lobe orifice is the first dependent one encountered, if on his back the orifice of the apical segment of the lower lobe; if a foreign body enters the upper lobe it is likely to lodge in its posterior segment, or in one of the axillary branches. Thus the distribution of lung abscesses is explained and their embolic origin confirmed. The posterior segment of the right upper lobe, the apical segment of one or other lower lobes, and the left upper lobe, in that order, are the commonest situations for a pyogenic abscess (Fig. 256); and it is noteworthy that these are also frequent sites of tuberculous cavitation.

complete drainage, the situation is that of any badly drained abscess. Therefore the rapid shrinkage and resolution which accompanies good drainage cannot occur. Furthermore, cavities in the elastic mesh of the pulmonary parenchyma are often subject to effects not encountered elsewhere. One of the commonest of these is the increasing tension in the cavity and communicating bronchus produced by the ball-valve action of a slough, permitting air to enter during inspiration but preventing it being expelled during expiration; similarly a small oblique fistula between cavity and bronchus may admit air when the bronchus expands but may not allow it to escape. In either of these two ways, therefore, although the foul contents of the abscess may be coughed up, the cavity itself, instead of diminishing, may progressively increase in size. Furthermore, when drainage is unsatisfactory, or is not promptly established, an abscess is likely to extend by direct invasion of the surrounding lung; new daughter cavities thus open out of the parent cavity, often by narrow communications, so that the parenchyma becomes honeycombed with septic diverticula. This is the state of affairs in chronic suppurative pneumonitis (Fig. 257). Yet another mode of extension of an abscess is by the same mechanism giving rise to the initial abscess—septic bronchial embolism. It is not surprising that pus or fragments of slough expelled from the cavity are apt to spill into and occlude the bronchus of another lobe or segment and thus produce another area of acute pneumonitis from which a new abscess develops. Thus it is not uncommon, when an abscess already exists in the right upper lobe, to find a few days after bronchial drainage begins, a further abscess in the middle lobe, and sometimes spread from there to the apex of the lower lobe, or indeed to the opposite lung. It has been said (*vide supra*) that a fringe of pulmonary tissue is generally preserved intact immediately deep to the visceral pleura of the affected segment because of the generous blood supply provided here by the subpleural vascular plexus. This integrity, however, is by no means inviolate in the face of a virulent infection or incomplete drainage. The subpleural layer may be invaded, its vessels thrombosed, and its tissue necrosed so that the abscess eventually reaches and ruptures through the pleura discharging pus into the pleural cavity and thus producing an empyema. A pleural effusion not infrequently accompanies lung abscesses, and must always be regarded as the precursor of an empyema.

Finally, as with any suppurative lesion of the lung, the threat of a metastatic blood-borne cerebral abscess is ever present, and is greatly increased by delayed or inadequate drainage. Even if none of these calamities befall, and the original abscess drains itself substantially by way of the bronchial tree, the abscess cavity may diminish considerably in size, but may not quite resolve, leaving a small chronic cavity in the affected segment of the lung communicating with a bronchus. Epithelium soon extends from the bronchus to line the cavity with squamous or cuboidal cells, thus rendering it permanent. Such a cavity will never

other reason, and is commonly associated with dental sepsis. A careful history, and persistent search for a possible cause, are of great importance. If a causal incident is identified, it usually precedes the onset of marked symptoms by a week or ten days, although the interval varies according to the virulence of the infection and the degree of obstruction. The fever, rigor and pain may be dismissed simply as "pleurisy" or "post-operative pneumonia," and since the widespread and indiscriminate use of potent antibiotics, there is no doubt that many abscesses are aborted, or their manifestations so masked that they may pass on to a phase of chronic cavitation without at first being recognised.

In the early stages the patient may have little or no sputum, and any that is expectorated need not be foul, for no drainage is taking place into a bronchus. There is, however, fever, malaise and toxæmia. The tongue is brown and coated. Physical signs of diminished movement of the affected part of the chest, dullness, lessened air entry, and sometimes pleural friction, are present. The white cell count rises to 20,000 or more; X-rays reveal a rounded opacity with ill-defined margins, usually situated in the posterior or axillary segments of the upper lobe, or in the apical segment of the lower. Both postero-anterior and lateral films are essential for accurate localisation.

As the abscess increases in size, occupying more and more of the affected segment of lung, it approaches the pleural surface, but the subpleural vascular plexus tends to preserve it from the advancing gangrene. Thus a rind of pulmonary tissue intervenes, for a time at least, between the abscess cavity and the visceral pleura. The patient's cough, hitherto irritative and only scantily productive, may now change. A small hæmoptysis often occurs and a foul taste is noticed by the patient. A wave or two of sepsis precedes the rupture of the abscess into the adjacent bronchus, and consequent production of copious filthy pus of nauseating odour, so characteristic of anaerobic suppuration, and particularly notable in the lung (hence the term "acute putrid abscess"). Occasionally the patient is unaware of the bad smell, which is, however, distressingly apparent to others. Several ounces of pus continue to be coughed up for a number of days, but the quantity may be as great as half a pint or more if the abscess is large and drainage free. Such abrupt and profuse bronchial drainage is not uncommon and may result in progressive diminution in the size of the cavity and ultimate spontaneous healing, although some residual effects, such as scarring and bronchiectasis or the persistence of a small residual cavity, are usual. As soon as drainage begins, the X-ray opacity reveals itself beyond doubt as an abscess, for a fluid level now appears separating the air-filled upper part from the pus below (Fig. 255).

Unfortunately this simple course cannot be relied on, for it depends on early establishment of adequate drainage through a bronchus. If the spontaneous fistula between abscess and bronchus is inadequate for

ing a lung abscess. As operations on the teeth and upper respiratory tract are known to be dangerous from this point of view, certain prophylactic measures should be respected. General anaesthetics must always be given under good conditions by well-trained, skilled anaesthetists. Large tooth extractions leaving wide areas of bleeding and often purulent gums should be avoided, and the practice of extracting teeth or performing operations on the nose or throat with patients sitting upright is to be deplored. When the cough reflex is abolished, safety is secured by keeping the patient's head at a lower level than the thorax. Following all general anaesthetics an early return to consciousness is desirable, and over-dosage with such drugs as *morphine* and *pentothal* should be avoided. Until consciousness returns, and particularly when the cough reflex is absent, great care must be taken to maintain an unobstructed airway. The nose and mouth are kept clear of blood and secretions by means of suction, and the patient is placed semi-prone, with the trunk and head slightly raised, and the head turned to one side to minimise the possibility of aspirating pharyngeal contents. When consciousness returns (as in modern anaesthesia this should take place in the theatre, even after large operations) the patient must at once be encouraged to cough. Before operations it is wise to clear up dental and gingival sepsis by all available means, especially if an intratracheal tube is likely to be passed.

**Treatment.** The introduction of antibiotics has transformed the management of lung abscess. Thus 70 to 80 per cent. of acute abscesses heal with adequate chemotherapy and postural drainage, whereas previously the majority were subjected to rib resection and external drainage, a method of treatment giving unsatisfactory results and carrying a substantial mortality. If pus is present it must be drained ideally by spontaneous and complete evacuation from the bronchial tree, but if this does not occur, or is inadequate, by surgical intervention. It is an error to regard conservative treatment as "medical," for abscesses of the lung, like those of the abdomen, must be under the constant observation of a surgeon, whose attention must be directed, above all, towards preventing an acute abscess becoming chronic, for once chronic suppuration in the lung is established, permanent damage follows and may prove fatal unless the affected pulmonary tissue is removed.

As soon as a lung abscess is diagnosed and accurately localised by postero-anterior and lateral X-rays, penicillin is given intramuscularly in doses of 500,000 units six-hourly. If sensitivity tests show penicillin to be unsuitable, it is discontinued and the appropriate antibiotics substituted. In addition, once rupture occurs, postural drainage of the affected lung segment is employed daily for periods as long as the patient will tolerate, sometimes aided by slapping and percussion from a physiotherapist. The posture is determined by the anatomy of the bronchus involved, but if more pus is produced in some other position than the apparently correct one, it should obviously be employed instead. It is almost always

close, but persists as does a chronically infected cyst, causing a productive cough like that seen in bronchiectasis, and even when the fire of acute infection burns out it is usual to discover in the damaged segment of lung an area of residual chronic bronchiectasis. It is evident that all types of lung abscess are a potential source of danger to life, formidable complications, or chronic ill-health if not promptly and efficiently treated.



FIG. 257. Chronic suppurative pneumonitis established in the upper lobes and apical segment of the lower lobe of the right lung.

**Prophylaxis.** Since the introduction of antibiotics in the treatment of most kinds of inflammatory disease in the chest, the incidence of acute putrid lung abscesses has decreased. Nevertheless, they still occur, although they are now more likely than before to reach the surgeon in a subacute or even chronic phase, for antibiotics not only save the lives of patients who would previously have died, but also diminish the severity of the symptoms of all abscesses. There is, however, no room for complacency, for published series still show a mortality averaging more than 10 per cent. and a very much higher rate of subsequent morbidity. It is, therefore, indefensible to expose patients to unnecessary risks of develop-

of choice, and resection should then be substituted unless steady progress continues with conservative treatment.

**External Drainage.** The position of the abscess is exactly localised by postero-anterior and lateral X-rays. As all lung abscesses soon become close to the visceral pleura, and the great majority are readily accessible on its costal aspect, surgical approach is by rib resection. If, however, an abscess points towards the mediastinal surface of the lung or in a fissure, it is probably wiser to resect it rather than to submit the patient to the two-stage drainage otherwise necessary (*vide infra*).

Ribs are carefully counted and distances measured from the midline so that a point on the chest wall is selected exactly corresponding to the underlying abscess. Local anaesthesia is always employed to avoid the risk of pus flooding over into the patient's opposite lung. It is also an advantage, if access is not thereby interfered with, to have the patient sitting up with the head on the arms which are in turn supported by the operating table (as for the drainage of an empyema). This position is not, however, always possible. The rib to be resected is exposed by a short vertical incision; the periosteum is incised and stripped off the rib and about 3 inches of the latter are resected subperiosteally with the aid of a guillotine costotome. The subcostal bundle of artery and vein is ligated at each end of the gap and the intervening part of the vessels is excised. The posterior periosteum (i.e. bed of the rib) is now incised and the parietal pleura exposed. If localisation is accurate the parietal pleura is adherent to the visceral pleura and is thickened and opaque. If, however, it appears unaffected and the pleural cavity is free, the approach may be inaccurately placed and must be carefully reviewed. It may be possible to correct it by resecting a little more of the rib or by extending the vertical incision and removing some of the rib above or below until the adherent area is found. Occasionally, however, obliteration of the pleural space has not occurred. If this is so, the usual method is to insert an iodine gauze pack into the wound for about two weeks and then to drain through the adherent area so formed, but if external drainage is indicated at all it is indicated urgently, so that a fortnight's delay is too long; therefore it is better to stitch the visceral and parietal pleural layers together and drain the abscess at once. There is little fear of infecting the pleural cavity if the patient is having appropriate antibiotics.

An aspirating needle is passed through the obliterated area and the position of the abscess confirmed. The intervening tissue, including a layer of lung parenchyma, is then cut away with a small diathermy loop and good access to the abscess established. Should the pleural cavity be inadvertently opened at any time it must be securely closed; the subsequent effusion is aspirated. After the pus has been sucked out of the abscess cavity a malleable Nelson light can be passed inside and all sloughs and debris removed. The abscess wall is inspected for any abscess extensions which are also evacuated. The cavity is dusted with penicillin,



desirable to bronchoscope the patient soon so that any solid material in the bronchus can be cleared, the anatomy can be confirmed, and the presence of a neoplasm or of a tooth fragment or some other foreign body can be excluded (*tartar flakes from teeth are much more often responsible for abscess formation than teeth themselves or foreign bodies*). Otherwise, bronchoscopy does not play any part in the treatment, either as a means of aspirating pus or insufflating antibiotics. Chest X-rays are taken every two or three days to follow the progress of the abscess. There should be regular diminution in its size. A persistent fluid level, or cessation or sudden diminution of sputum implies unsatisfactory drainage.

If steady reduction in the size of the abscess and improvement in the patient's general condition do not occur, or, still more, if the cavity shows signs of extending or of ballooning, serious thought must be given to surgical intervention. This may take one of two forms, external drainage or primary resection. For many years external drainage was used routinely for lung abscess at any stage of its development. During the acute phase results were good and resolution was usual, although the operation not infrequently led to such complications as secondary hæmorrhage from intercostal or pulmonary vessels, chest-wall infection, secondary bronchial fistulæ, or empyema, and the mortality following this method of treatment varied between 3 and 28 per cent. As soon as an abscess becomes subacute or chronic, i.e. after a month of conservative treatment, external drainage becomes less satisfactory, for closure of the cavity is slower or may never be achieved. This is easy to understand for the cavity walls have become fibrous, satellite abscesses may have formed, or a bronchial fistula may have become established. Sometimes secondary infection occurs from old broken-down tuberculous lesions. Furthermore, permanent bronchiectatic changes may take place in the affected lobe and a residual abscess cavity—soon to become epithelialised—is likely to remain. Both of these conditions continue to give rise to trouble and eventually need further operative treatment. Therefore, rib resection and external drainage should never be used after the acute phase has passed; primary resection of the involved lobe or segment is the correct treatment, for this not only cuts short an otherwise long and dangerous illness by removing the whole of the affected area at once, but it would probably ultimately be needed if external drainage was established originally. Since fewer and fewer acute lung abscesses are submitted to the surgeon, external drainage is falling into desuetude and being replaced by resection. If, however, an acute abscess is seen to contain a large slough and therefore cannot drain, or fails to drain adequately for some other reason (maintaining or increasing its size as observed by X-rays), external drainage still remains the correct treatment, and it is also indicated for subacute and chronic abscesses in patients too ill or frail for pulmonary resection. After a month or six weeks, external drainage ceases (except in the latter group of patients) to be the treatment

such infections the virulence of the organisms is low or general resistance is high. Alternatively, the acute manifestations may have been suppressed, but not extinguished, by chemotherapy, and after its withdrawal the embers once more light up.

According then to the severity of the initial infection, patients may give a history of acute onset resembling that associated with an acute abscess, i.e. fever, pleural pain, and sputum which may or may not have been fetid, depending on whether or not anaerobes were initially present. In fact, a carefully taken history usually does reveal a period when the taste and smell of the sputum were foul. Sometimes the onset is mild and signs of consolidation lead to the diagnosis of "bronchopneumonia." Frequently the onset follows soon after an operation or dental extractions. Oral or nasal sepsis may be present. There may have been exposure, immersion, drunkenness, or excessive intake of drugs—in short, exactly the soil from which, as we have already seen, springs the acute putrid abscess.

Many months, or occasionally years, may have gone by before the patient presents with a story of repeated "attacks of pneumonia." Between these attacks the patient expectorates several ounces and sometimes much larger quantities of pus which by the time he is seen is not offensive. A reduction in the amount of pus heralds the onset of febrile bouts and is due to drainage becoming obstructed. Between bouts the patient often feels deceptively well. Hæmoptysis may occur and there is often clubbing. X-rays show a consolidated area in the lung, often initially involving the upper lobe or apex of the lower, and in the midst of the consolidation it is possible to discern one or more abscess cavities. The process may involve extensive areas of the lung or even both lungs. It is of great importance to distinguish such chronic suppuration from pulmonary tuberculosis or a cavitating neoplasm. In chronic suppuration alone, neither acid-fast bacilli nor malignant cells are present in the sputum, although a suppurative lesion is sometimes secondarily infected from an adjacent focus of tuberculosis. Bronchoscopy must always be carried out to localise the lesion and also in search of a possible growth or foreign body. The sputum yields, as would be expected, a mixed bag of organisms amongst which will be found *Streptococcus viridans*, pneumococci, staphylococci and hæmolytic streptococci. Bronchography helps little as it merely tends to obscure the radiological appearances, and the opaque medium seldom enters the affected bronchi, many of which are already obstructed. It may have value, however, in demonstrating absence of disease in normal areas.

The longer the disease is present the more it is prone to spread : (a) By direct extension and the burrowing out of new cavities until the affected lobe or lung is honeycombed with them ; (b) by spill-over (embolic spread), fragments of pus or debris lodging elsewhere in the bronchial tree and setting up a fresh suppurative process. This is precisely what happens when an acute abscess is left undrained.

and a loose gauze-roll pack, impregnated with zinc oxide emulsion, is inserted into it. After three or four days the pack is removed and replaced by a large-bore rubber tube, draining directly into the dressings. The tube should not impinge on the abscess wall, but simply maintain the drainage route from the abscess to the chest wall, otherwise it may cause ulceration with the risk of secondary hæmorrhage or fistula formation. Chemotherapy and breathing exercises are continued post-operatively. The cavity should shrink rapidly. Its progress can be watched by running lipiodol into it from time to time and comparing the X-rays taken. The drainage tube is shortened and later replaced by one of smaller bore. Ultimately when the abscess cavity is obliterated the tube is removed from the remaining track in the chest wall.

**Complications of External Drainage.** The most troublesome is secondary hæmorrhage. The severe type arises from an intercostal artery, but bleeding may also be caused by ulceration of a pulmonary vessel, or simply by oozing granulations. The patient should always be taken to the theatre where the bleeding can be inspected in a good light, and dealt with by ligation or electrocoagulation. A direct attack must always be made on the source of bleeding. Blood transfusions may be required. Packing the track and cavity is ineffectual.

If the pleural obliteration has not been secure, or if it has inadvertently been opened at operation, an effusion and subsequent empyema may follow drainage. Any such effusion is completely and promptly aspirated, and penicillin is left in the pleural space before withdrawing the needle. If this does not suffice to control the infection it must be dealt with as described in the chapter on empyema.

A bronchial fistula may persist in a lung abscess if the original communication was large, or one may form as the result of ulceration produced by a drainage tube. Persistence of a bronchial fistula suggests the possibility of secondary infection by an adjacent tuberculous focus which has broken down, or the presence of a hitherto unsuspected neoplasm. The sputum and pus are searched for acid-fast organisms or malignant cells. Bronchoscopy is repeated, and biopsy of both the affected bronchus and the drainage track is carried out. Persistent fistulæ and unresolved abscess cavities are treated by resection of the involved lobe or segment.

**Chronic Lung Abscesses.** Unless resolution, both clinical and radiological, continues steadily, an acute abscess, whether infected with anaerobes ("putrid" abscess) or not, is regarded as having entered a chronic phase after some six weeks. Chronic pulmonary suppuration once established seldom recovers, and may lead within a few years to a fatal termination unless adequate operative treatment is carried out. Chronic lung abscesses arise from infection of the lung by aspiration of septic material which does not cause an acute abscess (or the acute abscess is formed but not detected by reason of its milder symptoms) but enters first a subacute and then chronic phase before declaring itself. In

such infections the virulence of the organisms is low or general resistance is high. Alternatively, the acute manifestations may have been suppressed, but not extinguished, by chemotherapy, and after its withdrawal the embers once more light up.

According then to the severity of the initial infection, patients may give a history of acute onset resembling that associated with an acute abscess, i.e. fever, pleural pain, and sputum which may or may not have been foetid, depending on whether or not anaerobes were initially present. In fact, a carefully taken history usually does reveal a period when the taste and smell of the sputum were foul. Sometimes the onset is mild and signs of consolidation lead to the diagnosis of "bronchopneumonia." Frequently the onset follows soon after an operation or dental extractions. Oral or nasal sepsis may be present. There may have been exposure, immersion, drunkenness, or excessive intake of drugs—in short, exactly the soil from which, as we have already seen, springs the acute putrid abscess.

Many months, or occasionally years, may have gone by before the patient presents with a story of repeated "attacks of pneumonia." Between these attacks the patient expectorates several ounces and sometimes much larger quantities of pus which by the time he is seen is not offensive. A reduction in the amount of pus heralds the onset of febrile bouts and is due to drainage becoming obstructed. Between bouts the patient often feels deceptively well. Hæmoptysis may occur and there is often clubbing. X-rays show a consolidated area in the lung, often initially involving the upper lobe or apex of the lower, and in the midst of the consolidation it is possible to discern one or more abscess cavities. The process may involve extensive areas of the lung or even both lungs. It is of great importance to distinguish such chronic suppuration from pulmonary tuberculosis or a cavitating neoplasm. In chronic suppuration alone, neither acid-fast bacilli nor malignant cells are present in the sputum, although a suppurative lesion is sometimes secondarily infected from an adjacent focus of tuberculosis. Bronchoscopy must always be carried out to localise the lesion and also in search of a possible growth or foreign body. The sputum yields, as would be expected, a mixed bag of organisms amongst which will be found *Streptococcus viridans*, pneumococci, staphylococci and hæmolytic streptococci. Bronchography helps little as it merely tends to obscure the radiological appearances, and the opaque medium seldom enters the affected bronchi, many of which are already obstructed. It may have value, however, in demonstrating absence of disease in normal areas.

The longer the disease is present the more it is prone to spread : (a) By direct extension and the burrowing out of new cavities until the affected lobe or lung is honeycombed with them ; (b) by spill-over (embolic spread), fragments of pus or debris lodging elsewhere in the bronchial tree and setting up a fresh suppurative process. This is precisely what happens when an acute abscess is left undrained.

Either spontaneously, or under the influence of antibiotic therapy, initial areas may resolve, leaving behind only the radiological appearance of fibrosis; but such a "burning out" is deceptive, for there is often subsequent recrudescence. If these lesions are untreated, extension in the lung continues indolently, with fresh febrile attacks, fresh abscesses, and new spill-overs, until death occurs from bronchopneumonia. Empyema may supervene, but when it does it is usually related to some new extension of the suppuration, since in the older parts the pleural cavity is long since obliterated. Cerebral abscess may develop at any stage, and amyloid disease is probable if life lasts. Without treatment, few patients survive longer than three years.

In the affected areas there is much damage to and distortion of bronchi. The abscess cavities are often epithelialised from the bronchi, but the mucosa undergoes squamous metaplasia. The cavities become permanent. Cavities of various sizes intercommunicate through a pulmonary honeycomb, and surrounding areas are chronically inflamed and fibrosed.

**Treatment.** It has been demonstrated that when penicillin levels in the blood rise sufficiently high some of it reaches abscess cavities. Patients with chronic pulmonary abscesses, therefore, need large doses—at least 2 mega units in twenty-four hours, continued for two to three weeks or more according to response. Other antibiotics are used when penicillin-resistant organisms are identified. A well-balanced diet and adequate fluids must be given. As with acute abscesses, postural drainage is valuable and must be carried out regularly, although owing to the complexity of the abscess system it is not fully effective. By these means, but chiefly by the use of antibiotics, the disease is brought under control and regression may occur. In particular, the quantity of sputum, previously at least several ounces and sometimes a pint or more in twenty-four hours, is greatly reduced. If the patient is too old, or otherwise unfit for pulmonary resection, or the disease is already bilateral, no more can be achieved. Postural drainage should, therefore, be continued and antibiotics reserved for reinfection of the epithelialised abscess cavities as it occurs, but in all fit patients the further treatment of choice is prompt pulmonary resection, either lobectomy or pneumonectomy, removing the whole of the suppurative area. External drainage of chronic abscess cavities is absolutely contraindicated.

**Staphylococcal Lung Abscess.** The abscesses so far considered follow aspiration of septic material from the nose or mouth, and are often associated with severe dental sepsis, an operation, or recent loss of consciousness, and characteristically affect certain lung segments more than others. Their walls are clearly defined on X-ray examination, and the pus expectorated from them is copious and more commonly foetid than not. The infecting flora is mixed. They may call for surgical relief.

The staphylococcal lung abscess contrasts with them in almost all these respects. Although the infection may sometimes reach the lung by

aspiration, it usually does so by the blood stream, and follows a septicæmia arising from a staphylococcal focus elsewhere in the body. There is no relation to diseased gums and teeth, or to suppression of the cough reflex, but general debility and wasting diseases are sometimes associated in adult patients. Particular lung segments are not selected. The abscesses are typically thin walled, cyst-like, and empty. The pus from them is seldom fætid and is often scanty, yielding a pure culture of *Staphylococcus pyogenes*. Surgery is very seldom indicated.

Staphylococcal pneumonia, first recognised during the influenza epidemic of 1918, is well seen during such outbreaks when it assumes a virulent and overwhelming form and is the usual cause of death. The patient is prostrated, with a high remittent fever and a relatively slow pulse, although weak and poor in volume. There may be sweating, or at least a clammy skin, but usually no rigor, and a peculiar pinkish cyanosis is a feature of the illness. Much frothy purulent sputum, which is sometimes blood streaked, is produced and grows *Staphylococcus pyogenes* in profusion. Consciousness may be long retained, until the victim dies from respiratory insufficiency and profound toxæmia. This severe infection, seen during the influenza epidemics, carried a very high mortality. Its behaviour and prognosis have been entirely altered by antibiotics, and staphylococcal pneumonias of a much milder type are now common, and many of them lead to—and indeed are recognised by—the peculiar abscesses which characterise them.

Staphylococcal infections of the lung with abscess formation are most often seen in infancy and childhood, but also occur quite frequently during adult life when they may be associated with long illnesses or debility. If X-rayed in the early invasive stage, the lungs often display scattered areas of mottling due to patchy bronchopneumonia, although only one area is sometimes affected. The typical distribution of aspiration abscesses is not seen. After a few days the sputum, at first scanty, becomes more profuse and purulent, but seldom offensive. Renewed X-rays show the area of consolidation to have cavitated into small multiple abscesses the walls of which rapidly distend until they assume the appearances not of abscesses but rather of tension cysts of the "alveolar" type. Sometimes they are single, but they are much more commonly multiple, forming a ballooning group of delicate "soap bubbles," quite empty as a rule, but occasionally with a meagre and transient fluid level at their base due to staphylococcal pus (Fig. 258). These small thin-walled abscesses rapidly become expanded by the partially obstructed air flow, until they cease to resemble abscesses but are much more like the bullæ of severe emphysema.

In infancy such ballooning cysts frequently rupture very quickly into the pleural cavity, infecting it and giving rise to the type of staphylococcal empyema so common in childhood, and so often assumed to be primary, since the initial lesion and the subsequent abscess in the lung pass un-

recognised (Fig. 259). Many, if not most, of the "congenital lung cysts" and the "ballooning cysts" seen in the infant lung are in fact staphylococcal abscesses, and most cases of spontaneous pneumothorax in early life owe their origin to the same cause. Sometimes early rupture does not occur, and one of the cyst-abscesses may distend to such a degree as to cause mediastinal displacement and respiratory embarrassment. When this occurs it may become necessary to aspirate it, or even leave a tension pneumothorax needle in place as a safety valve, and brave the risk of



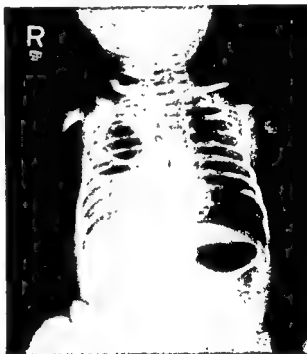
FIG. 258. Ballooning staphylococcal abscesses occurring in the lung of a man who had recently had a hemicolectomy.

subsequent empyema for the sake of immediate relief. On no account should the surgeon allow his anxious medical colleagues to persuade him to operate on such cases by lobectomy or pneumonectomy, for they will recover with patience and penicillin. No less striking than their spectacular apparition is the sudden melting away of these cysts when infection is overcome and the obstruction to the alveolar areas relieved. Occasionally, however, some may linger for weeks or even months, and possibly the groups of bullae described in young adults as "localised emphysema" may represent past staphylococcal infection the monuments of which persist long after the disease. A group of such abscess-cysts related to the diaphragm or inferior mediastinum is not uncommonly mistaken for

herniation of loops of bowel into the chest because of the distended wall occasional fluid levels, and evidence in consecutive X-rays of rapid alteration in size and shape. The clinical history and course of the illness serve to differentiate the two conditions, but if any doubt lingers a barium follow-through settles the matter. Complete resolution of the cysts is the rule, but evidence of scarring and some fibrosis at their site can be detected radiologically, and residual bronchiectasis sometimes remains.

Certain other specific pneumonias, much rarer than that caused by the

FIG. 259. Typical appearance of staphylococcal pneumonia in infancy with an accompanying pyopneumothorax.



staphylococcus, are known to produce lung abscesses. *Friedlander bacillus* is the infecting agent in one and *actinomycosis* in another. The former takes either an acute and rapidly fatal form or a much more chronic one resembling chronic pulmonary tuberculosis radiologically, but in which the sputum yields a pure culture of Friedlander's bacillus and is innocent of tubercle bacilli. The infection begins as a bronchopneumonia which soon becomes confluent, involving the whole lobe. If the infection is not soon arrested, severe destruction of pulmonary tissue occurs with abscess formation in the midst of the consolidated areas. Such abscesses are formed by, and may contain, substantial lung sloughs which can be seen radiologically like icebergs lying amongst the pus and surrounded by a ragged cavity wall (Fig. 260). Friedlander's bacillus is insensitive to penicillin, but usually responds to chloromycetin or aureomycin. When a lung slough is present primary extirpation of the lobe or lung is indicated.



*Actinomyces* have been found living saprophytically in a large proportion of healthy lungs and it is not clear upon what factors their pathogenicity depends. Lung infection with actinomycosis however, does occur, and once it is established, abscess formation develops. Indolent excavations of the pulmonary tissue follow with extension to the pleura and resultant empyema. This is often localised, since the intense pleural reaction secures obliteration of much of the cavity, and the empyema may



FIG. 260. A huge abscess of the right lung from which a pure culture of Friedlander's bacillus was obtained.

be coughed up through a bronchial fistula, or sinuses may develop through intercostal spaces to discharge upon the surface. Diagnosis depends on the recognition of actinomyces with their yellow granules, and treatment consists of large dosage of penicillin (2 mega units a day) which should be maintained for some time after all trace of the infection has been extinguished.

*Hydatid cysts* rupturing into a bronchus inevitably become secondarily infected and may be mistaken for simple pyogenic abscesses if scolices or daughter cysts are not found in the sputum, or if the possibility of an

echinococcal infection is not envisaged. A history of residence in areas where the disease is endemic may prove helpful, and the presence of other cysts with a positive Casoni reaction is conclusive. Once a hydatid cyst does establish a fistula and becomes infected its removal is best accomplished by lobectomy.

*Congenital bronchial cysts* communicating with a bronchus are likewise prone to secondary infection, and their subsequent behaviour and treatment are that of a chronic lung abscess.

## CHAPTER 35

### NON-TUBERCULOUS EMPYEMA

WHEREAS it is true that empyema, like most pyogenic infections, has become less common since the general use of antibiotics, it still remains a frequent complication of a wide variety of chest diseases and is, undoubtedly, one of the worst treated of common disorders. Indeed, the promiscuous use of antibiotics in febrile disorders may mask the development of pleural infection, so that it enters a chronic phase before recognition; and then the pus may be sterile and the infection therefore is mistakenly thought to be tuberculous. Furthermore, whereas some empyemas result from infection of the pleural sac during acute and transient inflammation of the lung, others accompany pathological changes of far graver import; it is therefore never sufficient to diagnose simply "empyema"; but the question "secondary to what?" must always be answered.

**Aetiology.** Infection of part, or the whole, of a pleural cavity most commonly occurs as a complication of pneumonia, but also may follow rupture of a lung abscess through the visceral pleura. A good example of the two combined is seen in the staphylococcal empyemas so often encountered in infants and sometimes mistaken for primary conditions. They are in fact always secondary to staphylococcal bronchopneumonia with its characteristic thin-walled cyst-abscesses, one of which ruptures producing a pyopneumothorax.

Chronic bronchiectasis is characterized by periodic acute exacerbations, often with pleurisy, and during one of these an empyema may form. Empyema may be one manifestation of blood-borne infection, as in pyæmia. Infected hydatid or bronchial cysts sometimes rupture into the pleura. Bronchial obstruction from any cause, e.g. an inhaled foreign body or an innocent neoplasm, may result in distal infection which can cross the visceral pleural barrier. An empyema in a man of cancer age should be regarded as due to bronchial obstruction by a carcinoma, or to the breaking down of a neoplasm, until bronchoscopy has been performed and a growth excluded. Pyogenic organisms may reach the pleura through a penetrating injury of the chest wall (rarely through the clean track of a high-velocity bullet, but commonly through the ragged wound of a high-explosive fragment which, because of its irregular shape and relatively low velocity, is likely to carry with it fragments of clothing).

About a quarter of all subphrenic abscesses are accompanied by infected effusions or empyemas above the diaphragm (Fig. 261); but spread of infection in the reverse direction, from an empyema to the

subphrenic space, is exceedingly rare. Clearly, the longer a subphrenic abscess is left undrained the more likely is it to cause an empyema, and ill-directed attempts to aspirate a subphrenic abscess may result in puncture of the pleura in its costophrenic sinus and consequent spread of the infection, with possibly disastrous effects.

On its mediastinal aspect, the pleural sac may become infected by



FIG. 261. A right basal empyema secondary to a subphrenic abscess. Note the displacement of the heart to the opposite side.

perforation of the œsophagus. This is usually due to laceration of its wall by a foreign body; and of such foreign bodies the œsophagoscope is the commonest. Oesophageal carcinoma does not often result in empyema because inflammatory adhesions have time to form round a sloughing growth, but rupture of the œsophagus is more likely during instrumentation, especially when attempts are being made to dilate malignant strictures. Occasionally empyema may arise from perforation of a peptic ulcer of the lower end of the œsophagus.

Broncho-pleural fistulae were once the commonest complication of pulmonary resections, but since the advent of antibiotics they have become comparatively rare. When however a post-operative fistula does occur, the communication between the respiratory tract and the pleural sac, through the leaking bronchial stump, leads almost inevitably to an empyema.

**Diagnosis.** If empyema supervenes in lobar pneumonia the patient becomes more ill and more toxic. Fever, hitherto sustained, becomes hectic and remittent and increasing dyspnoea occurs as the pleural fluid accumulates and the heart and mediastinum are pushed towards the opposite side. The chest movements, percussion note and air entry are further impaired, and the bronchial breath sounds of the consolidated lung are diminished. Vocal fremitus, which is increased in pneumonia, decreases and finally disappears. A leucocytosis occurs and rises above 20,000 cells per cmm. Radiography of the chest shows the increased

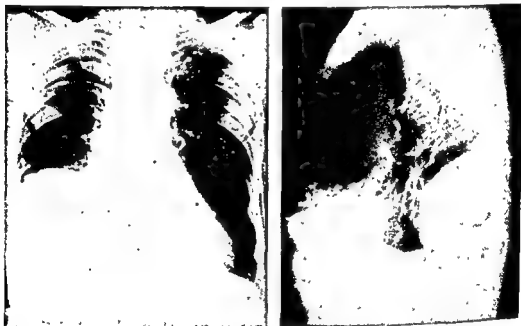


FIG. 262. An encysted posterior basal empyema of long standing. It was symptomless, being discovered on routine radiography, and was thought to be a tumour.

opacity of an accumulation of fluid rising high in the axilla. When the pleural layers are already considerably adherent however, the infected effusion may be localised to a small area directly related to the diseased part of the lung and, depending on the size of the empyema and the effects of antibiotics, physical signs and systemic effects are modified, or even so much diminished that the empyema escapes diagnosis and passes untreated into a chronic state before revealing itself (Fig. 262). Occasionally pus may be confined in one or other of the lung fissures or between the lung and the mediastinum or the diaphragm; thus it is not directly related to the chest wall at all, and aspiration is rendered very difficult. This type of empyema is however uncommon. When an empyema is suspected, and confirmation gained by physical examination of the chest, accurate postero-anterior and lateral X-rays are taken to determine its precise situation. If the empyema is a total one, i.e. involving the

whole pleura, very little difficulty is met, but if the collection is small and localised it is imperative to count the ribs accurately and to confirm the radiological appearances by percussion and auscultation until the true position of the empyema is ascertained.

**Principles of Treatment.** Errors in treatment are less likely to be made if a clear conception of its aim exists. The true object of the surgeon is to restore the normal function of the chest as quickly as possible. The aims are therefore : (1) to secure prompt re-expansion of the underlying lung, and (2) to maintain the mobility of the chest wall (for physiological purposes the "chest wall" includes the diaphragm). It is to these ends that treatment is directed, and all other considerations are secondary to them. If either of the above physiological principles is neglected treatment fails.

**Aspiration.** The diagnosis of acute empyema should at once be confirmed by aspiration. The apparatus needed for aspiration of the chest consists of a syringe of 20-ml. capacity or more, a two-way tap and needles of bores varying with the thickness of the pus. One of the most useful and efficient types of aspirator is the Martin syringe with its two-way connection (Fig. 263). The syringe, tap and needles have bayonet or screw fittings, otherwise they may separate during the aspiration, allowing air to enter the chest. If this happens the layers of the pleura, not yet firmly adherent, may separate and a localised empyema be converted into a total one. Furthermore, to allow air to enter the chest on any pretext is, at best, to delay re-expansion of the lung. It is thus indefensible and amounts to malpractice.

A point is chosen at the lower border of the intercostal space (avoiding the intercostal vessels and nerve which run along the inferior border of the rib) corresponding with the maximum dullness and maximum opacity on the X-ray films. No attempt should be made to find the most dependent part of the empyema for aspiration, for it is here that all the debris, clots and fibrin collect and aspiration is therefore difficult if not impossible. Most failures to aspirate empyemas are due to needling too low. After preparation of the skin and the arrangement of sterile towels, a skin bleb is raised over the chosen spot with  $\frac{1}{2}$  per cent. procaine and more of the local anæsthetic is infiltrated down to the parietal pleura. A liberal quantity should be employed as it is essential for the whole procedure to be quite painless. The patient is placed in a comfortable and relaxed position which should afford good light and easy access to the area to be needled. If the latter is posterior, as is often the case, the patient may rest his arms and head upon pillows placed before him on a bed table. It is essential, too, that the operator be seated comfortably, as both he and the patient may have to remain in one position for a considerable time.

The aspirating needle is now advanced through the chest wall until pus is located. As soon as some is withdrawn, a pair of rubber-tipped Spencer-Wells forceps is lightly clipped on to the needle flush with the

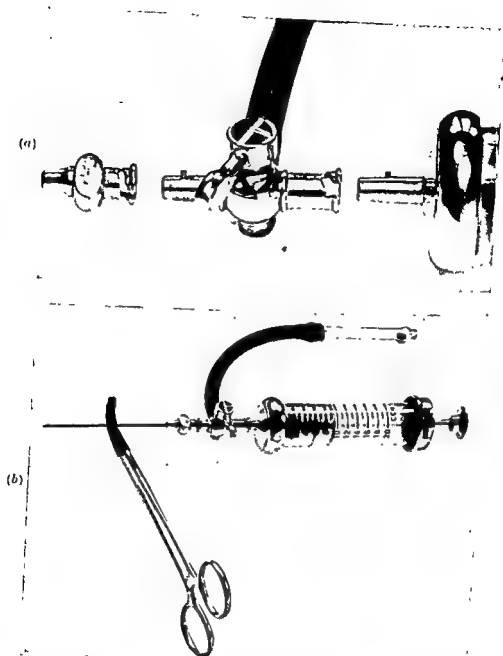


FIG. 263. (a) The two-way tap and bayonet fittings of a Martin's syringe.  
(b) The syringe assembled.

skin (Fig. 263b). This prevents the needle see-sawing, either backward into the muscles or forward into the lung ; damage to the lung may cause a pneumothorax or an air embolism. "Pleural shock" is a coroners' myth. Deaths attributed to it are almost always due to air embolism.

The first sample of pus obtained is delivered into a sterile test tube and sent at once for culture and an estimation of antibiotic sensitivity. A further tube full is kept in a rack in the ward for comparison with pus obtained at successive aspirations. Finally, all the purulent fluid in the

chest is removed. None should be allowed to remain, for with the passage of time more and more fibrin is deposited on both the visceral and parietal layers of the pleura and thereby chest wall mobility and lung expansion are impaired. As the object of treatment is the speediest possible restoration of both of these, it follows that all available pus must be evacuated as soon as possible. There are no exceptions to this rule provided the aspiration is carried out gently, painlessly and efficiently. Before the needle is finally withdrawn, the opportunity is taken to inject 1 mega unit of penicillin into the infected cavity (if bacteriological examination of the pus shows penicillin to be unsuitable, a more appropriate antibiotic is



FIG. 264. On the left, a left-sided empyema before treatment. On the right, after treatment by aspiration alone.

substituted). Penicillin given intramuscularly, irrespective of the dose, does not pass into an empyema cavity. Therefore injections into the cavity must be repeated.

The procedure detailed above is repeated as often as fluid re-accumulates—perhaps daily, possibly every second day—with the object of securing, as rapidly as possible re-expansion of the lung and so complete obliteration of the space. If the empyema is diagnosed early, and promptly treated in this manner, there is an excellent chance of complete and early cure with minimal damage to the machinery of respiration (Fig. 264).

**The Use of Enzymes.** Aspiration often fails to cure an empyema, for it may not be recognised until much thickening of the pleura has already occurred, or the pus may contain so much fibrin and slough that aspiration, even through a wide-bore needle, proves impossible. Pneumococci in particular tend to produce much clot, whereas streptococci secrete



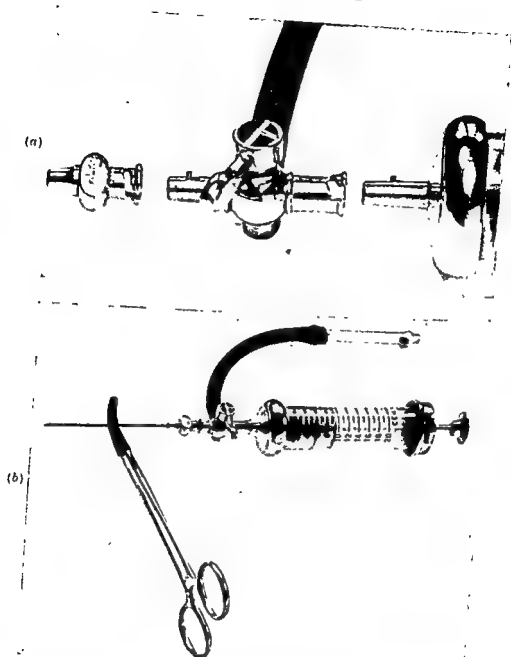


FIG. 263. (a) The two-way tap and bayonet fittings of a Martin's syringe.  
(b) The syringe assembled.

skin (Fig. 263b). This prevents the needle see-sawing, either backward into the muscles or forward into the lung; damage to the lung may cause a pneumothorax or an air embolism. "Pleural shock" is a coroners' myth. Deaths attributed to it are almost always due to air embolism.

The first sample of pus obtained is delivered into a sterile test tube and sent at once for culture and an estimation of antibiotic sensitivity. A further tube full is kept in a rack in the ward for comparison with pus obtained at successive aspirations. Finally, all the purulent fluid in the

occluded and the pin is secured flat against the chest wall by two strips of strapping.

Usually for the first ten or twelve days after the insertion of such a drainage tube there is still considerable mobility of the affected lung. Therefore it is of advantage to connect the tube to a water-sealed drain. The tubing is passed through the lattice of the patient's back rest and is delivered under a measured quantity of saline or antiseptic in a Winchester bottle beside the bed (Fig. 265). A shorter glass tube connects the air space above the liquid to the external air or, if gentle continuous suction is desired, a pump is attached to the air vent. The fluid in the bottle rises and falls in the longer tube with each respiration and the height of the meniscus provides a measure of the negative pressure in the pleural cavity (Fig. 265). So long as much mobility of the lung remains, this negative drag facilitates re-expansion of the lung. This passive effect is supplemented by active measures in the form of breathing exercises vigorously carried out. Special attention is paid by the physiotherapist to movements of the diaphragm and the part of the chest wall most impaired. As active exercises are always more valuable than passive ones, it is usually not long before the advantages of closed drainage are outweighed by the disadvantages of keeping the patient tethered to the bed by the bottle and its tubing. This point is generally reached after about ten days, when the re-expansion of the lung will be found to have slowed down. The tube should then be cut short just external to its retaining pin and allowed to drain directly into a dressing (Figs. 266 and 267). The patient gets up and the exercises are intensified. The treatment is now that of a chronic empyema cavity.

**Intercostal Drains.** On rare occasions this method of drainage is used as an interim measure. When, for example, an elderly patient is not thought well enough to be moved from bed it is justifiable to use an intercostal drain. A self-retaining catheter of the Malecot or de Pezzer type is inserted by means of a large trocar and cannula, which is passed through an intercostal space under infiltration anaesthesia. Afterwards the catheter is connected to an underwater seal as already described. The method has two serious disadvantages: (1) the lumen of the tube (which is limited in size by that of the cannula) is too small and is apt to be blocked by clots; (2) as the tube is confined between two ribs it very soon causes pain by pressing on the intercostal nerve immediately above it.

**Management.** Once an empyema is drained the tube must in no circumstances be removed until the related lung has completely re-expanded. When the cavity is wholly obliterated the track merely extends through the thickness of the chest wall. The tube must not be removed because discharge has ceased or because it is sterile, or for any other reason whatsoever. Untimely removal of the tube is the commonest mistake in the treatment of empyema and may cause disastrous consequences. A residual cavity of this kind left within the chest constitutes

lysins which dissolve the fibrin and so keep the pus fluid. Such lysins can be artificially prepared and used therapeutically. The empyema cavity is injected with 40,000 (Christensen) units of streptokinase and 10,000 units of streptodornase on three alternate days, and aspiration attempted on each of the intervening days. As the fibrinolysins do their work the clots are broken down, the pus becomes more fluid, and aspiration is again effective. Lysins do not however dissolve fibrin that has already been laid down on the pleura. The only reaction caused by them is a rise in temperature. The resulting discomfort can be controlled by antipyretics.

**Surgical Drainage.** If in spite of these measures or because the empyema is already chronic when diagnosed satisfactory clearance is not achieved, surgical drainage is necessary. By the time such a stage is reached, the pleural layers are completely adherent and there is no risk of their separation when the chest is opened. Ten millilitres of lipiodol are injected into the cavity and postero-anterior and lateral X-rays taken. The most dependent point of the empyema is thus clearly marked. The operation is best performed under local anaesthesia with the patient sitting upright on a stool and leaning forward with the arms resting on the operating table, which is adjusted to a comfortable height. Thus if a bronchial fistula is exposed when slough is removed there is no risk of pus flooding into the lungs. A vertical incision is made over the area marked by the lipiodol and the presence of pus confirmed by exploration with a needle. About four inches of the most dependent rib are now resected subperiosteally. Its bed is incised and the empyema cavity exposed. Not only is all the pus it contains sucked out, but a retractor and a malleable light are inserted so that a good view of the interior is obtained and all the mantle of soft fibrin and slough coating the walls stripped off with sponge-holding forceps. At the same time the cavity is inspected for neoplasm or fistulae and the mobility of the lung observed. When a complete toilet has been completed and the interior is smooth and clean, a large-bore rubber drainage tube is inserted and the layers of the chest wall stitched loosely about it. The tube is held to the skin by a nylon suture until the incision is healed. A safety-pin is then passed through the wall of the tube so that the lumen is not

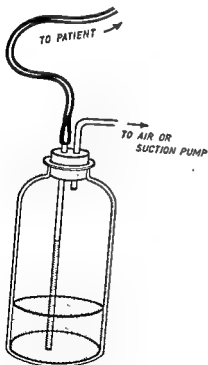


FIG. 265. A water-sealed drain.

a time-bomb which sooner or later, occasionally within weeks, but often not for months or even years, "explodes." As soon as a drainage tube is prematurely removed the chest wall rapidly contracts and may close completely, or from time to time a bead of pus may be extruded from a narrow sinus. The infected pocket within may track further in the pleural space, causing periodic febrile illness, or perhaps present somewhere else upon the chest as an *empyema necessitatis*. The infected pocket may eventually rupture into the lung, flooding it with pus, or it may give rise to a cerebral abscess or to amyloid disease.

It is therefore essential in the management of chronic empyema to follow the gradual contraction of the cavity by periodic pleurograms. At intervals of a few weeks the tube is removed. Lipiodal is run into the track through a soft rubber catheter and a glass funnel. A radio-opaque marker is placed on the skin sinus and the orifice lightly plugged with ribbon gauze. The patient is then X-rayed in a variety of positions so that the whole of the internal shape and dimensions of the cavity can be clearly seen. The plug is then removed, the lipiodal is run out, and the drain is re-inserted.

A pleurogram may reveal the position of the tube to be such that drainage is not dependent and several ounces of pus are being allowed to stagnate below it (Fig. 267b). The tube may not be sufficiently long so that the track is forming an isthmus beyond it, perhaps pinching off a substantial distal pocket (Fig. 267c). If attempts are made to follow the progress of such a track merely by the use of probes or bougies, these are arrested at the stenosis and the cavity beyond remains undetected. These conditions must be corrected. A lower rib may have to be resected to secure truly dependent drainage. A stenosis may have to be dilated and the tube passed beyond it so that the extremity of the cavity is obliterated first. Then the tube is withdrawn an inch or two in advance of the expanding lung.

Occasionally an unsatisfactory pocket is drained afresh by the insertion of a separate tube. If dilatation of a stenosed track is required, it must never be attempted with a solid dilator or bougie which would act as a piston in the tube and thus create a high positive pressure before it, and probably give rise to a fatal air embolism. A hollow sound with a perforated head is employed, or a laminaria tent is inserted and left in place for twenty-four hours. Some of the difficulties arising in drainage are illustrated in Fig. 267.

**Failure to Heal.** Aided by vigorous breathing exercises, the lung should advance steadily, diminishing the cavity regularly as it does so until it reaches the chest wall and virtually extrudes the drainage tube. Failure of expansion of the lung must lead to a search for the following impediments: (1) non-dependent drainage, (2) bottle-necking of the track, (3) secondary infection of the area by tuberculosis or actinomycosis, (4) a persistent bronchial fistula, or (5) the presence of a foreign body within the cavity. Of these the first two have already been discussed.

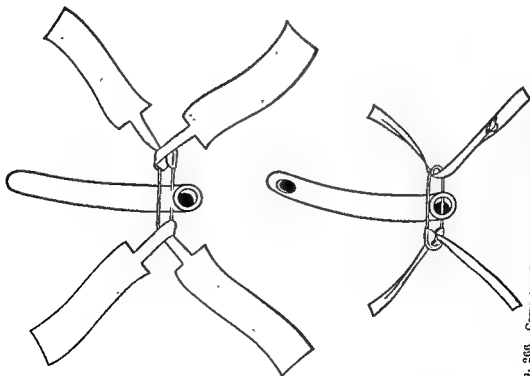


FIG. 266. Correct and incorrect methods of fixing an open empyema drainage tube. In the lower drawing the tapes are insufficient to hold the tube, and are not crossed to keep the pin flat against the chest. The pin passes through the lumen of the tube and is therefore likely to obstruct it. Side holes should not be cut in drainage tubes, for granulation tissue grows into such holes.

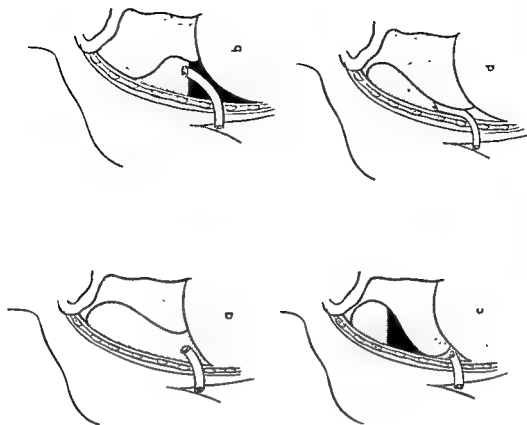


FIG. 267. (a) An empyema with tube in good position. (b) The drainage tube is too long. Pus is collecting below it. (c) The lung has expanded, pushing the tube up. If a sound is passed up the tube the bottle-necking will pass unnoticed. (d) The situation shown in (c) is corrected by lengthening the tube so that the superior part of the empyema can drain.

dilate it with a laminaria tent, insert an adequate drainage tube and carry out pleurograms; or the pus is aspirated, some lipiodal injected and the appropriate rib resected for dependent drainage. In either case the organism must be determined and tuberculosis or actinomycosis excluded as causative infections. At the time of drainage the interior of the cavity is inspected for possible growth, fistulae, rib sequestra, or foreign bodies such as pieces of rubber tubing. The remaining mobility of the lung is carefully observed and the extent and shape of the cavity examined. Upon these observations depend to some extent the future treatment. Equally it is essential to discover the state of the underlying lung. Does it perhaps contain a carcinoma or an adenoma or some other organic obstruction responsible for the pleural infection? Or is it bronchiectatic, or the seat of chronic suppurative pneumonitis, or is a broncho-pleural fistula present? If the latter exists the patient expectorates the contents of the empyema and a fluid level is visible in the pleural space. In all cases of chronic empyema bronchoscopy is indicated, and perhaps bronchography. An accurate diagnosis having thus been made, treatment can be carried out.

When the lung is apparently healthy and retains some mobility on the side of the empyema adequate drainage in good position, together with systematic and intensive breathing and postural remedial exercises may be all that is necessary for cure, although the duration of treatment will be proportionate to the duration of the disease, for the longer pus has existed in the pleural cavity the thicker will be the fibrin deposited and the more rigid the consequent corsetting of lung and chest wall.

It is not sufficient for the patient to carry out the exercises once or twice a day in the presence of the physiotherapist whose visits are merely the periods of instruction. The patient must devote some part of every waking hour to this vital part of treatment. The progress of lung expansion is followed by occasional pleurograms, at other times the length of the track can be measured by a fine gum-elastic bougie; the tube is adjusted as required. Thus the tube is shortened if it protrudes too far into the cavity, so acting as a bung, or lengthened when the diaphragm rises, or a portion of lung advances, rendering the track longer and tending, if not checked, to cut off the cavity from it (Fig. 267c). Side holes must not be cut in empyema drainage tubes for granulations grow into them, obstructing the lumen and bleeding freely when tubes are changed. It is unwise to employ sharp and otherwise traumatic probes or sounds for measuring purposes when a malleable gum-elastic bougie is available. The size of the latter must never be large enough to act as a piston in the sinus, producing a positive pressure and so, perhaps, a fatal air embolism. If discharge from the tube ceases it is a sign not of recovery but of danger; the obstruction to drainage must therefore be located. First remove the tube; if it is too long pus pours after it. If it is too short the track must be measured with a bougie and comparison made between the most

Any disease in the chest may be complicated by tuberculosis, for quiescent foci are common and may be re-illuminated by nearby pathological changes. Tubercle bacilli will not usually be found in the pus, but inoculation of guinea pigs or biopsy of the track wall may reveal tuberculosis. A persistent bronchial fistula may be caused by tuberculosis. Whatever may be its cause, resection of the lobe of the lung involved together with the empyema will probably be necessary.

Actinomycosis may be a primary cause of empyema. The characteristic fungi with their "sulphur granules" may be demonstrated. The condition is best treated by aspiration and administration of penicillin, which is injected into the cavity and intramuscularly, in daily doses of 2 mega units for about six weeks. It is now recognised that actinomyces are not always pathogenic in the lung and appear sometimes to exist as saprophytes and casual contaminants.

The commonest foreign body to find its way into the cavity is, of course, the drainage tube itself, especially during the out-patient stage of treatment. Pins may be removed at home or may cut through the tubing, and then any sharp intake of breath or sudden pressure on the chest results in the tube's disappearance. If the patient is unaware of its fate the doctor or nurse is likely to replace it with another tube, and only when delayed healing and continued suppuration arouse suspicions is the presence of the lost tube in the chest suspected. Radiographs do not show tubes unless they are barium lined. The insertion of a thoracoscope (or even a bronchoscope) along the track reveals the lost tube and sometimes it can be extracted without further surgery. Clearly the use of barium tubing is desirable, but it has the disadvantage of being expensive.

#### CHRONIC EMPYEMA

A chronic empyema is usually the monument of neglect or of surgical incompetence. Either the acute empyema has not been diagnosed at all or it has been untreated or ineffectively treated. The longer the empyema has been present the greater is the deformity and the greater the resulting impairment of function. The ribs fall together and become more and more oblique and triangular in section; the diaphragm rises and is immobilised; the heart and mediastinum are dragged to the side of the lesion, and the intervening lung lies shrunken and functionless; scoliosis develops, diminishing still further the size and mobility of the hemithorax. The accessory muscles of respiration waste, and as they are also the muscles of the shoulder, this joint becomes limited in range, stiff and painful. Clubbing of the fingers and pulmonary osteoarthropathy may exist, and sometimes the albuminuria, oedema and hepatic enlargement of amyloid disease occur. If a rib resection has been previously performed it is likely that, either at the site of operation or elsewhere upon the chest, an indolent discharging sinus will be found. The first step in reparation is to drain the empyema efficiently. If a sinus exists it is probably best initially to

## CHAPTER 36

### BRONCHIECTASIS

CLINICAL bronchiectasis is caused by two factors : (1) Atelectasis and (2) infection. When a bronchus becomes obstructed from any cause, e.g. an inhaled foreign body, a thick plug of tenacious sputum which cannot be dislodged by coughing, or, most commonly, the extrinsic pressure of enlarged peribronchial and hilar lymphatic glands, the pulmonary lobe or segment distal to the block becomes airless, and the normal mucous secretions are dammed within the corresponding part of the bronchial tree so that the lumen becomes distended by them. After early relief of the obstruction the bronchi resume their normal shape, but if the pent-up secretions become infected, as is commonly the case, permanent damage is done to the mucosa and to the bronchial wall. The mucous membrane becomes ulcerated, and after a time its ciliated columnar cells are replaced by squamous ones, the submucosal tissues become inflamed, with resulting lymphocytic infiltration and fibrosis, and are thus eroded, weakened and further distended. Clinical bronchiectasis is now established.

In infancy three common conditions—measles, whooping cough, and, above all, the primary tuberculous infection—cause enlargement of the lymphatic glands related to the bronchi, which, at this age have soft, very pliable cartilages, and therefore are most susceptible to pressure from without. Such lobar or lobular collapse is usually transitory but may persist. Clinical manifestations may be slight or absent, but, with increasing use of radiography, the condition is now recognised more often during childhood. At this age, too, septic adenoids or tonsils are a potential source of infection in the respiratory tract. It is therefore not surprising to find that adults with bronchiectasis have had a cough, intermittently productive of purulent sputum “from childhood” or for as long as they can recall. Natural resistance, or administration of antibiotics may extinguish the infection for considerable periods until it is reilluminated by a cold, tonsillitis, or pus from infected nasal sinuses being aspirated into the bronchi. This latter process becomes a vicious circle, for pus coughed up from the bronchiectatic bed may in turn infect the sinuses. The combination of bronchiectasis and chronic sinusitis is exceedingly common and it is futile to treat one without the other.

In the intervals between acute exacerbations, little sputum may be produced, but the excoriated bronchial wall may bleed, with resulting hæmoptysis. This is the basis for the artificial division of the disease into “wet” and “dry” varieties. The alternation of purulent sputum with hæmoptysis merely marks exacerbations and remissions of infection.



recent pleurogram and the length of the tube ; appropriate adjustments are then made. Possibly the retaining safety pin has been passed through the lumen of the tube instead of through its wall and the tube is thus blocked. With correct treatment the lung expands at last to fill the cavity completely and the tube track measures only the thickness of the chest wall. When this point is reached, and not until then, the tube may be removed.

**Decortication.** When the empyema is large and the lung immobile, although free from underlying disease, decortication provides a speedy and satisfactory cure. The selection of suitable patients must be stringent however, and they must be able to withstand a major surgical procedure. The operation of decortication is described under tuberculous empyema.

**Unroofing Operations.** When the residual empyema is small and the lung immobile, either Shédé's or Roberts' operation is of value. The former consists essentially of unroofing the empyema, saucerising it and allowing it to heal by granulation. The overlying ribs are resected and all the external tissues cut away, usually with a diathermy loop, so that finally no overhang persists at any point. The cavity is then lightly packed with wide ribbon gauze soaked in zinc-emulsion. When the granulating surface is clean healing may be expedited by skin grafting. For rather larger residual empyemas which are slow to heal, the Roberts' operation, employing a similar principle but in less mutilating fashion, is indicated. The drainage track is excised down to the empyema cavity and the latter is laid open by cutting along its anterior boundary. The skin and muscles overlying the cavity are then retracted and the ribs over the cavity are resected subperiosteally. The parietal wall of thickened pleura is cut away so that the soft flap of muscles and periosteum may fall in against the visceral surface (liberally dusted with penicillin). A pack is placed subcutaneously and retained there for a week to ensure adhesion between the muscle flap and the inner wall of the empyema cavity. Both Shédé's and Roberts' operation cause considerable blood loss. Therefore ample blood should be available for transfusion. A serious disadvantage of both operations is the probability that several intercostal nerves may be damaged with resulting paresis and anaesthesia of a part of the abdominal wall.

**Resection.** Finally, in cases where there is serious disease in the lung or lobe underlying an empyema, or when a bronchopleural fistula is present, pneumonectomy or lobectomy should be performed. Pleurectomy is also performed in order to remove the whole of the empyema with the affected lung. The technique for this operation differs in no way from any other pulmonary resection except that technical difficulties are inevitably greater. Dissection is carried out external to the parietal pleura from the start and fortunately as the hilum is approached it is generally found free of gross involvement by the empyema.

to send the patient to the doctor. Constant expectoration of pus can render life a burden. Clinical examination of the chest may reveal moist sounds at the left base (the most commonly affected part) ; they are more evident after coughing. There may be signs of an area of collapse, although examination may be quite negative. Similarly, plain X-rays sometimes show collapse, increased mottling or even "cysts," but may be altogether inconclusive. Bronchography is indicated when the patient's history suggests bronchiectasis, irrespective of the physical signs, or radiological findings. It should be meticulous in technique ; each lung is filled separately to avoid confusion in the interpretation of lateral films, and the diagnosis must not be regarded as complete while any bronchial



FIG. 268. Clubbing of the fingers in a patient with severe bronchiectasis.

segment remains unfilled (Fig. 269). The flora of the sputum should be examined, and the sensitivity of the organisms to the various antibiotics determined. A rigorous search should be made for tubercle bacilli. When the presence of a foreign body is suspected, bronchoscopy is carried out.

If the disease is suitably localised, surgery is the treatment of choice, and indeed is the only definitive treatment (Fig. 270). The continued existence of the purulent bronchiectatic bed—apart from the economic and physical disabilities to which it gives rise—is a menace to life, for there is the risk of metastatic brain abscess, amyloid disease, or spill-over and aspiration of pus into other parts of the lung. Furthermore, if any collapse persists, venous blood passing to the affected region in the pulmonary arteries is returned unoxygenated. It follows that ligation of these vessels during resection directs the flow to areas of the lung which are functioning properly, thus lobectomy may result in the patient being less breathless than before.

Similarly, whether bronchiectasis is "tubular" or "saccular" depends simply on its degree; thus the former represents a fairly homogeneous bronchial dilatation of moderate severity whereas the latter represents more extensive distension.

Although the sequence already described is perhaps the commonest and most characteristic, bronchiectasis will follow any chronic bronchial obstruction coupled with infection. Hence the urgency for the removal of an aspirated foreign body, combining as it is likely to do both causal factors, and the necessity to recognise and relieve post-operative atelectasis by prompt bronchoscopy. Pulmonary suppuration or chronic tuberculosis similarly give rise to bronchiectasis if, as is commonly the case, the bronchi of the inflamed or cavitated areas are occluded, and since the advent of antibiotic therapy, the surgical treatment of acute putrid lung abscess and suppurative pneumonitis is often confined to dealing with the bronchiectasis they leave behind.

The lobes of the lung most commonly affected with bronchiectasis are the left lower lobe, the lingular process, the middle lobe, and the right lower in that order or in progressive combination. The order indeed appears capricious, for aspiration is commoner into the right lower lobe than into the left, but the explanation of the sites of incidence of bronchiectasis lies in the anatomical distribution of peribronchial lymph nodes which, by their enlargement, more readily obstruct the lobes in the order named. Bronchi are squeezed between glands disposed anteriorly and posteriorly to them. If, however, the damage to the bronchi is due to primary suppuration, it is evident that it may affect any lobe, and since the posterolateral segments of the upper lobes, and the apical parts of the lower lobes are most often the seat of abscesses, it is here that localised post-inflammatory areas of bronchiectasis are found. Indeed, if the previous infection passes unnoticed hæmoptysis may occur, and its origin remain obscure until bronchograms are done.

Although bronchiectasis is recognised clinically by the history, it is only by bronchography that the diagnosis is confirmed and accurate delineation of the lobes and lobules involved and the state of all the remaining bronchi of both lungs demonstrated.

The patient is generally either a child or a young adult with a long-standing loose rattling cough productive of yellow sputum and worse on rising in the morning. There is commonly evidence of nasal obstruction, septal deviation or chronic sinusitis. If the disease is extensive, the fingers and toes may be clubbed and the patient sallow and toxæmic (Fig. 268). Children are often apathetic and stunted and are frequently mouth breathers.

The history probably includes acute febrile attacks of varying frequency, recurrent pneumonia, pleurisy, or even empyema. Remissions are usual although not invariable. In older patients hæmoptysis often appears during remissions; indeed blood in the sputum may be the first symptom

to send the patient to the doctor. Constant expectoration of pus can render life a burden. Clinical examination of the chest may reveal moist sounds at the left base (the most commonly affected part) ; they are more evident after coughing. There may be signs of an area of collapse, although examination may be quite negative. Similarly, plain X-rays sometimes show collapse, increased mottling or even "cysts," but may be altogether inconclusive. Bronchography is indicated when the patient's history suggests bronchiectasis, irrespective of the physical signs, or radiological findings. It should be meticulous in technique ; each lung is filled separately to avoid confusion in the interpretation of lateral films, and the diagnosis must not be regarded as complete while any bronchial



FIG. 268. Clubbing of the fingers in a patient with severe bronchiectasis.

segment remains unfilled (Fig. 269). The flora of the sputum should be examined, and the sensitivity of the organisms to the various antibiotics determined. A rigorous search should be made for tubercle bacilli. When the presence of a foreign body is suspected, bronchoscopy is carried out.

If the disease is suitably localised, surgery is the treatment of choice, and indeed is the only definitive treatment (Fig. 270). The continued existence of the purulent bronchiectatic bed—apart from the economic and physical disabilities to which it gives rise—is a menace to life, for there is the risk of metastatic brain abscess, amyloid disease, or spill-over and aspiration of pus into other parts of the lung. Furthermore, if any collapse persists, venous blood passing to the affected region in the pulmonary arteries is returned unoxygenated. It follows that ligation of these vessels during resection directs the flow to areas of the lung which are functioning properly, thus lobectomy may result in the patient being less breathless than before.

It remains, therefore, to assess operability. In an otherwise healthy patient, the maximum lung resection that does not eventually impose a fatal curtailment of respiratory function is pneumonectomy on one side when the opposite lung is healthy, removal of both lower lobes together with the lingula segment on the left, or removal of the equivalent of this amount of tissue in some other distribution. The question, however, that must occupy the surgeon is not "How much can I take away?" but "How much can I leave behind?" If, for example, bronchograms show the three basal segments of a lower lobe to be bronchiectatic, with a normal apical segment, it is unjustifiable to perform lobectomy. The basal



FIG. 269. Bronchogram of a normal right lung. Note that every anatomical segment is well filled, but there is no alveolar filling, or overfilling; no lipiodol has been allowed to spill over into the opposite lung. Such bronchograms provide a good opportunity for studying the anatomy of the bronchial segments.

segments alone should be resected and the apical one spared. When disease is bilateral, as it very commonly is, the side which appears worse should be resected first, and the patient warned that although sputum will be diminished it will not be abolished, and that a second operation will be required in two or three months time.

Instances often occur when bronchography shows some apparently mild bronchial distortion with good aeration of the parenchyma. Treatment in such cases must be decided by the disability produced. How much sputum has the patient? How frequently does hæmoptysis occur? How many febrile attacks are there a year? How many days are lost from work? Upon the answer to such questions must depend the decision to operate, and they must be weighed with particular care in patients over the age of forty, who with a little emphysema, a little

chronic bronchitis, may tolerate badly loss of any pulmonary tissue. On the whole, surgery is better avoided in patients much over the age of forty years unless there is actual collapse of a lobe or a whole lung is destroyed, for patients over this age who have had bronchiectasis for many years are likely to have so much associated bronchitis and emphysema that, apart from the added risks, surgery gives them little relief.



FIG. 270. Cystic bronchiectasis affecting chiefly the lower lobe basal segments (lateral view).

Once operation has been decided on and its exact extent defined, preparation of the patient begins. Upper respiratory tract diseases must first be corrected as far as possible. Thus infected tonsils or adenoids are dealt with, and infected nasal sinuses are drained. Throughout the patient's stay in hospital daily physiotherapy secures maximum chest and diaphragmatic mobility, and before operation the patient is taught the technique of dissociated chest movements. At the same time the affected segments of the lung are drained posturally at least twice daily for periods of about twenty minutes at a time. The position is suited to the lesion by the use of a Nelson's bed. For example, if either of the lower

lobes are involved the patient lies prone with the head and shoulders well below the level of the hips ; if the lingula or middle lobes are involved the patient lies supine. Forced coughing aids drainage. Aerosol inhalations of penicillin help to subdue infection and diminish sputum, but if penicillin-resistant organisms predominate, other appropriate antibiotics are indicated. When the sputum has become minimal, operation may be performed. The above regime may be continued as treatment for those



Fig. 271. Extensive bilateral bronchiectasis quite unsuitable for surgery. In such cases the palliative measures described are used.

patients in whom the disease is too extensive to permit excision, or who are unfit for operation (Fig. 271).

Post-operatively, the commonest complication is undoubtedly atelectasis of the remaining lobe or segments on the side of operation—the upper lobe when lower and lingular segments have been removed, and, very frequently, an apical segment when it has been spared and the remainder of the lower lobe excised. Such collapses do not usually occur at once, but most often on about the third or fourth post-operative days, when there is œdema near the healing bronchial stump, closely related to the remaining airway. Collapse is signalled by a sensation of tightness across the chest, dyspnoea, and a rise of pulse rate and temperature. The trachea or apex beat will be displaced to the same side as the airless lobe ; the percussion note is impaired over the affected lobe, and breath sounds are absent

or bronchial in character. The diagnosis is confirmed by X-rays. It is important that the patient should never be thirsty or dehydrated, for this makes the sputum more tenacious and difficult to expel, and every care should be taken to ensure freedom from wound pain so that there is no hindrance to coughing. The patient should be encouraged to sit out of bed on the second day, and soon to take a few steps. This minimises muscle stiffness and consequent wound pain, encourages chest movement, and usually pleases and encourages the patient. Should atelectasis nevertheless occur, it must instantly be relieved by bronchoscopic aspiration. Each hour that elapses renders relief more difficult. Often the mucous plug responsible cannot be aspirated at all, but must be plucked from its lodgement with forceps. If the attempt proves unsuccessful, the collapsed lung must be protected from the danger of secondary infection, and hence of further bronchiectasis, by systemic penicillin in full dosage and postural drainage. Bronchoscopy is repeated until re-expansion occurs. Thereafter nothing remains but to aspirate any effusion, and to promote full expansion of the remaining lung.

Persistence of sputum following resection for bronchiectasis usually means: (1) Disease in other segments of the lung; (2) secondary bronchiectasis in a residual segment of lung collapsed post-operatively; or (3) the formation of a puddle in the bronchial stump if this has not been divided flush with the remaining stem bronchus. Bronchograms should be carried out. Further resections of the areas involved may be necessary, or in the case of a defective stump, re-amputation is required.



## CHAPTER 37

### BRONCHIAL CARCINOMA

BRONCHIAL carcinoma is now much the commonest cancer in the male. It accounts for more than 7 per cent. of all male deaths and some 40 per cent. of all male deaths from cancer. It also accounts for many more deaths than does pulmonary tuberculosis in both sexes. The incidence is increasing rapidly, in almost epidemic proportions, in all civilised countries. The World Health Organisation figures per 100,000 for the four years ended 1952 (the last available date) are as follows: England and Wales, 61.4 (up 31 per cent.); Denmark, 24.8 (up 49 per cent.); Scotland, 56.3 (up 36 per cent.); France, 28.2 (up 30 per cent.); Italy, 16.4 (up 45 per cent.); Norway, 11.9 (up 32 per cent.); Switzerland, 33.5 (up 28 per cent.); Japan, 4.9 (up 68 per cent.); New Zealand, 31.5 (up 46 per cent.); and the U.S.A., 26.1 (up 21 per cent.). Over the past twenty-five years the number of cases has increased at least fifteen-fold in

England. In New York, where the records are particularly good, it has increased over 500 per cent. in men and 50 per cent. in women in the last twenty years. Some of this increase is undoubtedly accounted for by better diagnosis, but the figures quoted above cover a period in which this factor can have played no significant part. The increase is not due to an ageing population, as statistics for all other forms of cancer show level or slightly declining curves (Fig. 272). Unsuccessful attempts have been made to lay the blame on industrial smoke, fumes from tarred roads and the exhaust gas of petrol engines, but no evidence has yet been adduced in support of this. Women, for example, breathe the same

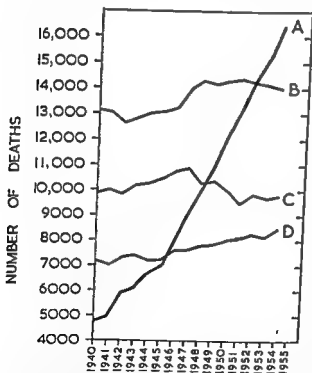


FIG. 272. Graph (after Horace Jowles) showing the increase in the number of deaths from cancer of the bronchus (A), in England and Wales between 1940 and 1954, compared with deaths from cancer of the stomach (B) and intestine (C), and from heart disease (D).

air as men and should therefore suffer equally from lung cancers were those caused by atmospheric pollution, but men outnumber them 12 : 1. A few industrial occupations such as nickel-cobalt mining in Schneeberg and processes involving arsenic or asbestos have been demonstrated to entail added risk, but these clearly do not affect the whole population.

In recent years a number of researchers have implicated increased cigarette smoking which has closely paralleled the carcinoma increase. In 1950 Wynder and Graham compared 600 men dying of bronchial carcinoma with controls : only 1.3 per cent. were non-smokers as opposed to 14.6 per cent. of the controls, and 51 per cent. smoked heavily compared to 19 per cent. in the control group. Doll and Hill's controlled and statistically sound survey for the Medical Research Council of more than 700 bronchial cancer patients in twenty London Hospitals demonstrated that above the age of forty-five the risk of developing the disease increased in simple proportion with the amount smoked, and that a man who has smoked twenty cigarettes a day for twenty years is about fifty times more likely to develop bronchial carcinoma than a non-smoker. Since 1950 thirteen similar investigations in six countries have yielded similar results. The American Cancer Society in 1952 recorded the life and smoking histories of no less than 188,000 men ; since then 8,000 have died, 285 from bronchial cancer ; and the death rate proved twenty-nine times as great among tobacco addicts as among non-smokers. Ewatts Graham, who in 1937 performed the first successful pneumonectomy for cancer, and E. L. Wynder of the Sloan Kettering Institute for Cancer Research, have consistently produced cancers on the backs of mice by painting with tobacco tar, and a specific carcinogen, 8 : 4 benzopyrene, has been isolated from cigarette smoke.

The annual consumption of cigarette tobacco in this country in the decade 1891 to 1900 per adult man was 0.4 lb. ; in 1911 to 1920 it had risen to 3.8 lb. ; and in 1941 to 1950 it was 8.3 lb. Women in 1911 to 1920 consumed only 0.2 lb. ; and even by the Second World War, 1941 to 1950, had reached only to 2.4 lb., that is, less than men smoked in 1911 to 1920, thirty years ago. This lag is reflected in the present sex incidence of bronchial cancer which is twelve or more men to every woman affected. The somewhat lower incidence in rural areas, adduced by some workers in support of the industrial smoke hypothesis, is similarly explained, for it has been shown that " the smaller a man's home town, the less likely he is to smoke cigarettes heavily " ; but it is none the less true that benzo-pyrene is present in smoke and can be recovered from urban air.

As carcinoma of the bronchus does occur among non-smokers, it is evident that, whatever the role of tobacco may be, other factors certainly play a part. The nature of these is speculative, but the frequency with which inflammatory lesions precede growths suggests that possibly squamous metaplasia, known to occur in some of them, may go further than a conversion of columnar ciliated cells to squamous cells, and

predispose, if it does not directly cause, malignant changes in the bronchial mucosa. Squamous or anaplastic carcinomas may arise in the midst of areas of chronic bronchiectasis.

**Pathology.** With the exception of a very few dubious varieties, all primary carcinomas of the lung arise in the bronchial mucosa and reproduce in a greater or lesser degree of differentiation its epithelial elements. It is not always easy, or possible, to decide precisely the histological type of pleomorphic bronchial carcinomas, but, in general, they can be divided into three groups the relative proportions of which vary in different series according to the views of the recording pathologist. The most common, and in general the least malignant (because best differentiated), are the squamous-celled carcinomas in which the cells vary from polygonal to flattened squames, and may show keratinisation and cells nests. Next come the "oat-celled" tumours, the undifferentiated, highly malignant type. The cells are small and oval with scanty cytoplasm and big darkly-staining nuclei. Finally there are the adenocarcinomas, which arise in mucosal glands, and are composed of characteristic columnar cells arranged around aveoli in glandular style. About 60 per cent. of bronchial carcinomas are squamous, 30 per cent. oat-celled, and 10 per cent. adenocarcinomas. It is mostly the latter that are found in non-smokers. Some American pathologists regard most "oat-celled" growths as undifferentiated squamous growths.

Metastasis occurs first to the hilar and to the many mediastinal lymph glands disposed about the trachea at its bifurcation and in the pulmonary ligament. Thence metastasis occurs below the diaphragm and up to the neck and supraclavicular fossæ; the axilla is not a common site. Blood-borne secondary deposits may lodge in the brain or in the liver, and later in the bones, the skin, the suprarenals, and other organs. Or they may travel along the air passages and appear in bronchi distal to the growth (Fig. 273), or spill into more dependent lobes, or even into the opposite lung.

Cancer of the bronchus may occur at any age from childhood to advanced old age, but is commonest in men between forty and sixty years of age. Growths are not infrequent in patients aged thirty to forty, and are occasionally seen during the twenties. In the two latter groups they are almost invariably anaplastic and should be treated by deep X-rays as well as by surgery.

**Diagnosis.** Irrespective of its histological character, cancer of the bronchus is a curable disease if diagnosed betimes; early diagnosis, as in all other cancers, is incomparably the most important single contribution to cure. On an average, eight to nine months elapse between the first symptoms of the bronchial cancer and the patient's arrival in a thoracic surgical bed, and as the expectation of life after diagnosis is about the same time it is not surprising that 50 per cent. of the patients already have mediastinal metastasis at operation. Only earlier recognition and prompt surgery afford hope of cure.

The earliest common symptom of cancer of the bronchus is cough, but because many men over fifty have a "winter" or "smoker's" cough this symptom is difficult to assess and is often disregarded. But if, in a man over forty-five, a winter cough persists into the spring, or a non-productive cough becomes productive, a chest X-ray should be taken. The next commonest early symptom is pain, which is not as a rule due to direct chest wall involvement, but to bronchial obstruction by a neoplasm causing collapse of the lobe or segment distal to it, with consequent pleural reaction. Sixty per cent. of patients on admission to hospital have X-ray evidence of atelectasis. Sometimes "pain" means only

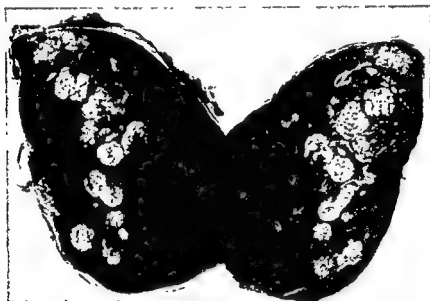


FIG. 273. Air borne secondary deposits all distal to a primary growth at the hilum of an upper lobe. Each secondary deposit was related to a small bronchus and no secondaries were present elsewhere.

discomfort, for the solid tumour, growing in the elastic web of the lung, prevents full inspiration. When infection occurs distal to the obstructed bronchus, as is so common, pyrexia, malaise and sometimes purulent sputum result in a diagnosis of "a touch of flu" or "broncho-pneumonia." If antibiotics are then given, the symptoms may be suppressed and a growth ignored. Should an opacity persist on X-ray, the term "unresolved pneumonia" should never be used. Pneumonia resolves, slowly sometimes, but progressively, and if the lesion does not resolve it is not "pneumonia." Such facile labels serve only to deceive, and may cost the patient his life. Dyspnoea is the next symptom in frequency, for sudden collapse of a lobe or even a segment in a man already emphysematous (as so many are in this age group) is likely to cause breathlessness. Haemoptysis is by no means the earliest of the common symptoms: this is

unfortunate for it is sufficiently dramatic for both patient and doctor to take seriously. It occurs characteristically in the form of a bright streak mixed with the sputum and is repeated each morning for about a week. This is the one symptom which is in itself almost diagnostic of bronchial carcinoma.

Loss of weight implies cachexia, and to wait for it is to wait too long, although its presence should not deprive the patient of the chance of surgery. Anorexia is a sign of sinister significance usually presaging hepatic metastasis. Later still may come hoarseness due to infiltration of the left recurrent laryngeal nerve, dysphagia from œsophageal involvement, distension of neck veins by mediastinal obstruction, or the intractable brachial plexus pain and Horner's syndrome caused by a tumour in the thoracic outlet (Pancoast's syndrome). All these manifestations spell inoperability.

*Hypertrophic pulmonary osteoarthropathy*, with or without finger and toe clubbing, is sometimes the first sign of carcinoma of the bronchus and helps to differentiate a doubtful lesion from tuberculosis in which disease it is extremely rare. "Rheumatic" pain of sudden onset in wrists, elbows, ankles or knees, always calls for a chest X-ray, and radiographs of the long bones may show new subperiosteal deposition. The pain is often nagging and is the patient's main complaint, for it keeps him awake at night. It can be abolished at once, and permanently, by resection of the pulmonary growth, or by cutting the vagus nerve to the affected lung in inoperable cases. More rarely, unexplained symmetrical neurological manifestations resembling peripheral polyneuritis or atypical myasthenia, are seen in the absence of cerebral or spinal metastasis, and these may precede, accompany, or follow a bronchial carcinoma.

When the doctor's suspicions have been roused he must follow them to a conclusion. Physical examination may elicit signs in the chest of diminished movement, displacement of the trachea or apex beat (perhaps pushed away from the lesion by its mass, but more often dragged to the same side by pulmonary collapse), dullness, reduced air entry, bronchial breathing or absent breath sounds. A persistent rhonchus not cleared by coughing suggests an organic bronchial stricture and even localises its position. Enlarged glands must be sought in the supraclavicular fossæ. The liver is palpated, nerve lesions are sought, a rectal examination is done. Pain in the limbs, backache, or "sciatica" must be fully investigated lest secondary deposits are the cause.

**Investigations.** Physical examination may be wholly negative, and it is possible for the most skilled clinician to be entirely deceived and to overlook advanced lesions of the lung. Postero-anterior and lateral X-ray films of the chest are indispensable. These may show a wide variety of abnormal shadows. The outline of a growth itself is typically irregular, with ill-defined edges; it is often situated near the hilum, flaring out into normal tissue (Fig. 274), but it may be compact and discrete with smooth

margins, closely resembling an innocent tumour or a tuberculoma, and situated peripherally (Fig. 275). Often the picture is one of collapse—a triangular segment of lung with a concave edge, drawing the mediastinum towards it, and causing increased translucency in the rest of the lung by resultant emphysema. Perhaps an abscess is visible, with a fluid level, and if the abscess is peripheral to the growth it may exactly resemble an acute lung abscess. Most often a malignant abscess is due to necrosis in the mass itself, when its walls will be seen to be thick, irregular and bossed



FIG. 274. A massive carcinoma growing rapidly at the hilum of the right lung.

(see under Lung Abscess and Figs. 254 and 276). Rarely, an abscess may be seen in a lobe remote from the growth, and is then caused by "spill-over" of pus or a necrotic fragment. Together these three varieties of malignant abscess constitute the commonest variety of all lung abscesses. Thus any abscess seen in the chest X-ray of a man over forty-five must be assumed to be due to cancer until proved of other origin. Symptomless "small round lesions" discovered on mass radiography must always be regarded with the gravest suspicion, and in middle-aged men they are more likely to be carcinomas than tuberculomas, especially if they are situated anteriorly in the lung. Peripheral growths of this type often turn out to be adenocarcinomas and tend to metastasise early. Out of a series of fifty consecutive lesions in this group, covering all ages, referred

unfortunate for it is sufficiently dramatic for both patient and doctor to take seriously. It occurs characteristically in the form of a bright streak mixed with the sputum and is repeated each morning for about a week. This is the one symptom which is in itself almost diagnostic of bronchial carcinoma.

Loss of weight implies cachexia, and to wait for it is to wait too long, although its presence should not deprive the patient of the chance of surgery. Anorexia is a sign of sinister significance usually presaging hepatic metastasis. Later still may come hoarseness due to infiltration of the left recurrent laryngeal nerve, dysphagia from œsophageal involvement, distension of neck veins by mediastinal obstruction, or the intractable brachial plexus pain and Horner's syndrome caused by a tumour in the thoracic outlet (Pancoast's syndrome). All these manifestations spell inoperability.

*Hypertrophic pulmonary osteoarthropathy*, with or without finger and toe clubbing, is sometimes the first sign of carcinoma of the bronchus and helps to differentiate a doubtful lesion from tuberculosis in which disease it is extremely rare. "Rheumatic" pain of sudden onset in wrists, elbows, ankles or knees, always calls for a chest X-ray, and radiographs of the long bones may show new subperiosteal deposition. The pain is often nagging and is the patient's main complaint, for it keeps him awake at night. It can be abolished at once, and permanently, by resection of the pulmonary growth, or by cutting the vagus nerve to the affected lung in inoperable cases. More rarely, unexplained symmetrical neurological manifestations resembling peripheral polyneuritis or atypical myasthenia, are seen in the absence of cerebral or spinal metastasis, and these may precede, accompany, or follow a bronchial carcinoma.

When the doctor's suspicions have been roused he must follow them to a conclusion. Physical examination may elicit signs in the chest of diminished movement, displacement of the trachea or apex beat (perhaps pushed away from the lesion by its mass, but more often dragged to the same side by pulmonary collapse), dullness, reduced air entry, bronchial breathing or absent breath sounds. A persistent rhonchus not cleared by coughing suggests an organic bronchial stricture and even localises its position. Enlarged glands must be sought in the supraclavicular fossæ. The liver is palpated, nerve lesions are sought, a rectal examination is done. Pain in the limbs, backache, or "sciatica" must be fully investigated lest secondary deposits are the cause.

**Investigations.** Physical examination may be wholly negative, and it is possible for the most skilled clinician to be entirely deceived and to overlook advanced lesions of the lung. Postero-anterior and lateral X-ray films of the chest are indispensable. These may show a wide variety of abnormal shadows. The outline of a growth itself is typically irregular, with ill-defined edges; it is often situated near the hilum, flaring out into normal tissue (Fig. 274), but it may be compact and discrete with smooth

margins, closely resembling an innocent tumour or a tuberculoma, and situated peripherally (Fig. 275). Often the picture is one of collapse—a triangular segment of lung with a concave edge, drawing the mediastinum towards it, and causing increased translucency in the rest of the lung by resultant emphysema. Perhaps an abscess is visible, with a fluid level, and if the abscess is peripheral to the growth it may exactly resemble an acute lung abscess. Most often a malignant abscess is due to necrosis in the mass itself, when its walls will be seen to be thick, irregular and bossed



FIG. 274. A massive carcinoma growing rapidly at the hilum of the right lung.

(see under Lung Abscess and Figs. 254 and 276). Rarely, an abscess may be seen in a lobe remote from the growth, and is then caused by "spill-over" of pus or a necrotic fragment. Together these three varieties of malignant abscess constitute the commonest variety of all lung abscesses. Thus any abscess seen in the chest X-ray of a man over forty-five must be assumed to be due to cancer until proved of other origin. Symptomless "small round lesions" discovered on mass radiography must always be regarded with the gravest suspicion, and in middle-aged men they are more likely to be carcinomas than tuberculomas, especially if they are situated anteriorly in the lung. Peripheral growths of this type often turn out to be adenocarcinomas and tend to metastasise early. Out of a series of fifty consecutive lesions in this group, covering all ages, referred



from mass X-ray units, 40 per cent. proved at operation to be malignant. When doubt as to the nature of these shadows exists, it is indefensible not to remove them.

Fluoroscopy helps to determine the mobility of shadows, their relation to the heart and mediastinum, and whether they move with deglutition. Paradoxical movement of one side of the diaphragm is proof of phrenic paralysis, and virtually removes any hope of removal of the tumour, as



FIG. 275. An adenocarcinoma in the lower lobe of the right lung. This growth was associated with hypertrophic pulmonary osteoarthropathy.

does also oesophageal distortion seen during a barium swallow. The use of lipiodol in the bronchial tree is best avoided for it obscures doubtful shadows.

The sputum is now searched for predominant organisms, and their sensitivity to antibiotics determined. Search is made for tubercle bacilli and malignant cells. This last investigation is technically simple but results depend on the skill and experience of the pathologist.

Bronchoscopy must never be omitted when cancer of the lung is suspected, even when the chest X-ray is negative. It is a simple procedure which is carried out under local anaesthesia. It provides the only means of clinching the diagnosis by biopsy. If, however, the growth is peripheral,

it may be beyond bronchoscopic vision, or its presence may be deduced only by the rigidity of a bronchus, by a bulging wall, or by a stricture. Bronchoscopy is negative in 30 per cent. of cases of carcinoma of the bronchus. This investigation is also vital in assessing operability. If a vocal cord is paralysed, the trachea invaded, the carina widened by enlarged mediastinal glands, or an insufficient length of stem bronchus is left between the trachea and the growth removal is impossible.

Further contraindications to major surgery are paralysis of the dia-

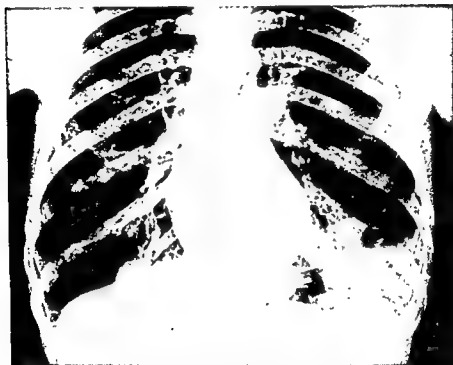


FIG. 276. This breaking-down squamous growth (note fluid level) arose in the midst of an area of chronic bronchiectasis in the patient's left lower lobe which had been X-rayed at intervals for twelve years.

phragm, Pancoast's syndrome, involvement of mediastinal structures such as the oesophagus, and a blood-stained pleural effusion (Fig. 277), but not necessarily a clear effusion (though such fluid should be searched for malignant cells), or even empyema. In addition, cardiac or respiratory insufficiency may render pulmonary resection impossible, and it is in borderline cases of this nature (a not inconsiderable proportion of men in the sixties) that the possibility of removing the neoplasm by lobectomy rather than pneumonectomy must be considered.

Invasion of the chest wall does not always preclude surgery and sections of affected diaphragm or pericardium are easily removed. Paralysis of the phrenic or recurrent laryngeal nerves is usually due to

from mass X-ray units, 40 per cent. proved at operation to be malignant. When doubt as to the nature of these shadows exists, it is indefensible not to remove them.

Fluoroscopy helps to determine the mobility of shadows, their relation to the heart and mediastinum, and whether they move with deglutition. Paradoxical movement of one side of the diaphragm is proof of phrenic paralysis, and virtually removes any hope of removal of the tumour, as



FIG. 275. An adenocarcinoma in the lower lobe of the right lung. This growth was associated with hypertrophic pulmonary osteoarthropathy.

does also cesophageal distortion seen during a barium swallow. The use of lipiodol in the bronchial tree is best avoided for it obscures doubtful shadows.

The sputum is now searched for predominant organisms, and their sensitivity to antibiotics determined. Search is made for tubercle bacilli and malignant cells. This last investigation is technically simple but results depend on the skill and experience of the pathologist.

Bronchoscopy must never be omitted when cancer of the lung is suspected, even when the chest X-ray is negative. It is a simple procedure which is carried out under local anaesthesia. It provides the only means of clinching the diagnosis by biopsy. If, however, the growth is peripheral,

cure in about 40 per cent. of the patients who are technically operable. *The extent of the operation must be decided strictly upon the facts of the case, including the age and general health of the patient, and the character and situation of the growth.* It is wrong to remove the whole lung, particularly on the right side, which is the larger of the two, if a lobectomy will yield just as good a result.

No patient should be rejected for operation unless one of the signs of inoperability already mentioned is present, or unless he clearly will not tolerate removal of the affected lung. This can sometimes be decided on clinical grounds, seldom on bronchospirometric tests, and never on estimation of the vital capacity, which is in no way a measure of function. The patient should always be given the benefit of the doubt, and it is often of the greatest value to see whether his colour is maintained on 20 per cent. oxygen when an intrabronchial balloon is made to occlude the bronchus of the affected lung. It must also be remembered that a patient with a functionless or collapsed lung is likely to be less breathless after ligation of the corresponding pulmonary vessels has diverted the blood flow to aerating pulmonary tissue. Patients over the age of seventy must be in particularly good condition before being accepted for surgery, but below this age lung resection is usually practicable with a low mortality. A past history of coronary disease is a serious drawback, but some degree of hypertension does not militate against success. Emphysema is common, therefore every effort must be made to increase costal mobility and to secure the maximum diaphragmatic movement before operation. The efficacy of the breathing exercises given can be measured by the increase of vital capacity produced by them ; this is the chief value of estimating the vital capacity.

Antibiotics are used for at least a week preoperatively to suppress associated infection and they are continued for ten to twelve days afterwards. Postural drainage is sometimes useful if the bronchial obstruction is not complete. The hæmoglobin percentage is raised to at least 80 by blood transfusion, given not less than four or five days before operation, and a further 2 or 3 pints of blood will be required during the operation itself. It must not be forgotten that part, if not all, of the dyspnoea may be due to anæmia. There must be a limit to the time spent in preoperative investigation and treatment, for a cancer continues to grow and may at any time disseminate. Its removal is urgent and other considerations are therefore subordinated to this.

In the first post-operative week the management of the patient is similar to that following any pulmonary resection : blood replacement, maintenance of the blood pressure, and adequate oxygenation are the chief concern in the first twenty-four hours. The patient returns from the theatre wearing a light well-fitting oxygen mask and with the intravenous drip still running. Both are maintained for as long as necessary—usually not more than twelve hours and often less. After pneumonectomy,

infiltration and not merely to pressure, but in very rare instances excision may still be possible. A long history suggesting growth is not always a hopeless omen, for it implies low malignancy and encourages determination. In general, it is impossible to assess operability on X-ray evidence. If all investigations prove inconclusive, and therefore doubt still



FIG. 277. The patient presented with a recurrent blood-stained pleural effusion. After aspiration no definite growth was visible in X-rays of the lung, but thoracotomy showed neoplasm spread over the whole pleura.

remains as to the nature of an abnormal X-ray shadow in a man of cancer age, it must be regarded as malignant until proved otherwise. In a few exceptional circumstances it may be justifiable to watch it for a short period only, under constant supervision, to see if its outline changes. It is never justifiable to stick needles through the chest wall into a suspected growth in an attempt to aspirate it or its contents. The next logical step in diagnosis when reasonable doubt exists is thoracotomy.

**Treatment.** Prompt pulmonary resection offers an excellent chance of

age should be encouraged to return to work as soon as they are fit to do so ; the great majority find this possible within three months of operation.

Deep X-ray therapy is not given routinely, but is reserved for selected cases. Well-differentiated squamous carcinomas respond poorly to it, and adenocarcinomas are completely radioresistant. Its chief value, therefore, is in treating poorly differentiated or oat-celled tumours. Even then it is contraindicated when dissemination has already taken place, when tumours are associated with sepsis, or when they are so situated (e.g. in a lower lobe) that radiation causes widespread damage to the lung, making an already breathless patient worse. Radiotherapy is prescribed for tumours involving bone (e.g. superior sulcus growths which affect the ribs, and tumours impinging on the vertebræ), or when there is mediastinal obstruction, for slow strangulation in this fashion is an unpleasant way of dying. Radiotherapy is also indicated for dysphagia due to œsophageal involvement. Sometimes irradiation will so reduce the size of an anaplastic (poorly differentiated) tumour that resection becomes possible, but the prognosis nevertheless remains bad.

Out of 222 patients undergoing thoracotomy at one hospital 145 proved operable. Of these 145 in whom resection was possible (109 pneumonectomies and 36 lobectomies) the survival rate for three years or more was 40 per cent. The hospital mortality rate was 4.5 per cent. ; the most frequent cause of death was heart failure. The age of patients submitted to surgery varied from nineteen to seventy-four, the very great majority were between fifty-five and sixty-eight.

In considering these results it must be remembered that no patient was rejected unless one of the specific contraindications already detailed was present, and that many purely palliative operations were deliberately undertaken. Palliation by surgery in patients who have no chance of permanent cure is often indicated. When bronchial obstruction has caused distal lung infection the patient is ill, pyrexial and toxic, and will be much benefited by the removal of a lung which may virtually be reduced to a bag of pus. The fact that a high temperature exists before the operation is no contraindication in itself to surgery, for it will fall to normal after its source is excised. Growths threatening to invade the chest wall or the thoracic outlet and brachial plexus are likely to cause severe and intractable pain, and demand, if necessary, heroic surgery and resection of the affected areas *en bloc* with the lung itself, but vertebral involvement calls for deep X-ray therapy, not surgery.

it is important to keep the mediastinum central. The position is adjusted before the patient leaves the theatre by insuring that the pressure in the empty pleural cavity is made atmospheric, but as air is absorbed, or fluid accumulates in the space, displacement may recur and is the commonest source of respiratory embarrassment. The intrapleural pressures are checked from time to time and air either added or withdrawn to maintain atmospheric pressure. If fluid accumulates too rapidly aspiration may be required. Any sign of intrapleural hemorrhage is an indication to return the patient to the theatre, reopen the chest, and secure the bleeding point. The latter is much more likely to be a systemic vessel than a pulmonary one. The state of the remaining lung is of vital importance and every effort must be made by the patient to expel secretions by coughing. He must be encouraged and aided in coughing at regular intervals, and the area of the wound manually supported. Should the patient's efforts prove insufficient, prompt bronchoscopy and aspiration are indicated as he lies in bed with his back supported by pillows. If much bronchospasm is present, relaxant drugs may be given by mouth, by injection, or by inhalation. Their effect has been shown to be summated if more than one route is employed.

In the absence of complications the patient may often be able, and indeed should be encouraged, to get up on the second post-operative day unless old age or cardiac disease makes a longer period of bed-rest desirable. Some elderly patients, and a larger proportion of patients in whom intrapericardial resection has been performed, develop post-operative auricular fibrillation which calls for medical treatment and longer bed-rest. For most patients, however, early ambulation has great advantages: it encourages pulmonary ventilation and expansion; it prevents muscle wasting and therefore vascular stagnation and thrombosis, and so lessens the hazard of embolus; it diminishes post-operative pain, which is largely due to stiffness and bruising; the joints and chest are kept mobile; patients are freed from the tyranny of the bedpan; and anxieties related to the operation are allayed and convalescence shortened. Patients are usually ready for discharge from hospital in less than three weeks.

Further treatment depends on whether or not the empty pleural space fills up sufficiently with serum to maintain the mediastinum in a central position. In most cases it does so, but sometimes it is necessary to readjust the intrapleural pressures by adding air once a month with an artificial pneumothorax apparatus. About 80 per cent. of patients who develop a recurrence or metastatic lesions do so within a year after resection and about 98 per cent. within two years. Patients whose general health is maintained and who show no evidence of recurrence two years from operation are unlikely to die from later recurrence, although there are exceptions to this rule. Therefore, patients still needing air-refills at the end of two years to keep the mediastinum central, should be offered the alternative of lateral thoracoplasty. All patients under pensionable

thus does not obstruct the airway. At the same time the blood is aspirated through a wide bronchoscope sucker, and as soon as a view can be obtained of the bleeding site an adrenalin swab is held against it until the flow ceases.

When the presence of an adenoma has been confirmed surgical removal is indicated. As the tumour is innocent, removal should be as conservative as possible, but depends on whether or not damage has been caused to the lung distal to the tumour. In very early cases, it is sometimes feasible to perform merely a bronchotomy. The posterior wall of the affected bronchus is incised and the adenoma is shelled out with a cuff of mucosa around its base, but more often a lobectomy is the best procedure. As a main bronchus is usually involved and patients have a long history, so much damage has sometimes been done to the lung that pneumonectomy is necessary, although it should be avoided if possible. The old practice of fulgurating adenomas at bronchoscopy to clear the bronchial lumen and thus allow the lung beyond to re-expand has no place in modern treatment, for it may cause malignant change. The results of conservative operations are excellent.

**Hamartomas.\*** In the lungs this term is now applied to two not uncommon but quite separate types of innocent lung tumours brought into prominence by mass radiography: (1) *Cartilaginous hamartomas* are composed mainly of cartilage but have peculiar fissures in their substance lined by columnar epithelium. They also contain some fibrous tissue and fat, and if these elements predominate a diagnosis of fibroma or lipoma may be made. In the past, they were usually encountered only at autopsy and were then called "chondromas." Sometimes the cartilage calcifies; radiological recognition is then easy, for a sharply defined and characteristically lobulated opacity is produced. The majority, however, do not become calcified and there is then no way of being sure the tumour is not a peripheral carcinoma, apart from thoracotomy, which therefore becomes imperative. When the lung is handled a hamartoma feels hard, smooth, lobulated and mobile within the lung parenchyma. The lung is incised directly down upon the white glistening surface of the tumour which is then easily shelled out. The bleeding points in the lung are controlled by digital compression and then ligated individually. The raw surfaces are approximated with interrupted catgut sutures. (2) *Vascular Hamartomas*.—Some congenital vascular abnormalities in the lung produce symptoms and give rise to X-ray opacities. If they are symptomless, or if the only symptom is hæmoptysis, it is necessary to distinguish them from malignant tumours; this may demand thoracotomy. There may be telangiectases elsewhere in the body, especially on the lips and mucous membrane of the mouth. A history of hereditary telangiectasia is particularly significant especially when the lesions are multiple.

\* Hamartoma = a tumour-like mass of superfluous tissue of congenital origin.



## CHAPTER 38

### INNOCENT TUMOURS OF THE LUNG

**Adenomas.** - Innocent tumours are rare in comparison with bronchial carcinomas and in many cases the chief indication for their removal is doubt as to their innocence. Of them, the most important is the bronchial adenoma which accounts for about 4 per cent. of all pulmonary neoplasms and is eight times commoner in women than in men. It should be noted that this almost exactly reverses the sex incidence of bronchial carcinoma.

Adenomas grow from the mucus-secreting glands in the deeper layers of the bronchial mucosa and usually arise proximally in the bronchial tree in the walls of either one or other stem bronchus or of the main lobar bronchi. Histologically they consist of cuboidal cells often having a regular acinar arrangement with evidence of mucus secretion, or sometimes of disorderly cellular masses; the cell clumps are held together by highly vascular connective tissue. Superficially they are easily mistaken for anaplastic carcinoma, but mitotic figures are rare and the part of the tumour presenting in the bronchus is usually covered by a layer of bronchial mucosa; unless due weight is given to the clinical findings a mistake in histological diagnosis may occur and lead to pneumonectomy rather than conservative removal.

The patient is most commonly a woman between twenty and forty years of age with a history of repeated hæmoptysis spread over several and sometimes many years. The hæmoptysis is more substantial than the typical streaking seen in carcinoma, but because of the age at which it occurs may lead to a long and fruitless search for tubercle bacilli in the sputum or even to the patient being immured in a sanatorium. As adenomas grow proximally they are likely to cause bronchial obstruction with consequent distal collapse of the lung. Infection follows and gives rise to purulent sputum; although if the growth blocks the bronchus completely the pus cannot be expectorated and soon causes an empyema. Sometimes there is no infection, but instead dammed-up mucus produces a bronchial mucous cyst, or distal bleeding from the tumour forms a hæmatoma. Bronchial obstruction and infection cause bouts of fever, malaise, and purulent expectoration, frequently interpreted as "pneumonia," "influenza," or "bronchiectasis" without discovery of the prime cause. Bronchoscopy is essential to diagnosis, and as most of the growths are within bronchoscopic range a biopsy is generally obtainable for histological confirmation. The adenoma appears as a lobulated raspberry-like tumour in the bronchus and brisk bleeding from it is apt to follow biopsy. If this occurs the operating table is inclined so that the patient's head is dependent and the blood runs up the bronchoscope and

thus does not obstruct the airway. At the same time the blood is aspirated through a wide bronchoscope sucker, and as soon as a view can be obtained of the bleeding site an adrenalin swab is held against it until the flow ceases.

When the presence of an adenoma has been confirmed surgical removal is indicated. As the tumour is innocent, removal should be as conservative as possible, but depends on whether or not damage has been caused to the lung distal to the tumour. In very early cases, it is sometimes feasible to perform merely a bronchotomy. The posterior wall of the affected bronchus is incised and the adenoma is shelled out with a cuff of mucosa around its base, but more often a lobectomy is the best procedure. As a main bronchus is usually involved and patients have a long history, so much damage has sometimes been done to the lung that pneumonectomy is necessary, although it should be avoided if possible. The old practice of fulgurating adenomas at bronchoscopy to clear the bronchial lumen and thus allow the lung beyond to re-expand has no place in modern treatment, for it may cause malignant change. The results of conservative operations are excellent.

**Hamartomas.\*** In the lungs this term is now applied to two not uncommon but quite separate types of innocent lung tumours brought into prominence by mass radiography: (1) *Cartilaginous hamartomas* are composed mainly of cartilage but have peculiar fissures in their substance lined by columnar epithelium. They also contain some fibrous tissue and fat, and if these elements predominate a diagnosis of fibroma or lipoma may be made. In the past, they were usually encountered only at autopsy and were then called "chondromas." Sometimes the cartilage calcifies; radiological recognition is then easy, for a sharply defined and characteristically lobulated opacity is produced. The majority, however, do not become calcified and there is then no way of being sure the tumour is not a peripheral carcinoma, apart from thoracotomy, which therefore becomes imperative. When the lung is handled a hamartoma feels hard, smooth, lobulated and mobile within the lung parenchyma. The lung is incised directly down upon the white glistening surface of the tumour which is then easily shelled out. The bleeding points in the lung are controlled by digital compression and then ligated individually. The raw surfaces are approximated with interrupted catgut sutures. (2) *Vascular Hamartomas*.—Some congenital vascular abnormalities in the lung produce symptoms and give rise to X-ray opacities. If they are symptomless, or if the only symptom is hemoptysis, it is necessary to distinguish them from malignant tumours; this may demand thoracotomy. There may be telangiectases elsewhere in the body, especially on the lips and mucous membrane of the mouth. A history of hereditary familial telangiectasia is particularly significant especially when the lung lesions are multiple.

\* Hamartoma — a tumour-like mass of superfluous tissue of congenital origin.

Single vascular hamartomas occur in which a fistula exists between a pulmonary artery and vein. A cavernous hæmangioma results from the tortuous aneurysmal distension of the adjoining vessels and gives rise to an X-ray shadow, which on screening or kymography is shown to be pulsatile. The short-circuit thus produced causes unoxygenated blood from the pulmonary artery to pass direct into the pulmonary vein and so to the systemic circulation. As much as half the blood volume may pass through the fistula. This results in dyspnœa, cyanosis, polycythæmia, and clubbing of the fingers and toes, and consequently suggests a congenital cardiac defect. If such a lesion is auscultated, a systolic murmur is often audible, which, in contrast with all cardiac systolic murmurs, gets louder on inspiration, when blood is sucked into the "hamartoma" by the concertina action of the lung. The presence of this syndrome is an urgent indication for excision of the lesion, if necessary, by lobectomy.

## CHAPTER 39

### CYSTS OF THE LUNG

**Congenital Bronchogenic Cysts.** An aberration of the outgrowing lung bud may occur at any point. If proximal, a solitary cyst in the mediastinum results, and it is attached to the bifurcation of the trachea by a stalk. It usually has no bronchial communication and, being full of mucus, appears on the radiograph as an opaque spherical tumour. More distally a similar cyst, or group of cysts, may be left in the midst of the



FIG. 278. A bronchogenic cyst containing a fluid level of mucus, and situated in the anterior segment of the left upper lobe. It fluctuated in size with respiration and caused breathlessness. Its wall contained cartilage and elastic tissue and it was lined with cuboidal epithelium.

lung itself. These cysts commonly have a bronchial connection, and are therefore either empty or contain some mucus showing a fluid level in X-rays; their perfectly round fine walls are also revealed so that they are at once recognisable as cysts (Fig. 278). Those cysts in continuity with a main bronchus are prone to become infected, but more distal ones are not. In the former, the mucus becomes purulent and the cyst behaves

Single vascular hamartomas occur in which a fistula exists between a pulmonary artery and vein. A cavernous hæmangioma results from the tortuous aneurysmal distension of the adjoining vessels and gives rise to an X-ray shadow, which on screening or kymography is shown to be pulsatile. The short-circuit thus produced causes unoxygenated blood from the pulmonary artery to pass direct into the pulmonary vein and so to the systemic circulation. As much as half the blood volume may pass through the fistula. This results in dyspnoea, cyanosis, polycythæmia, and clubbing of the fingers and toes, and consequently suggests a congenital cardiac defect. If such a lesion is auscultated, a systolic murmur is often audible, which, in contrast with all cardiac systolic murmurs, gets louder on inspiration, when blood is sucked into the "hamartoma" by the concertina action of the lung. The presence of this syndrome is an urgent indication for excision of the lesion, if necessary, by lobectomy.

## CHAPTER 39

### CYSTS OF THE LUNG

**Congenital Bronchogenic Cysts.** An aberration of the outgrowing lung bud may occur at any point. If proximal, a solitary cyst in the mediastinum results, and it is attached to the bifurcation of the trachea by a stalk. It usually has no bronchial communication and, being full of mucus, appears on the radiograph as an opaque spherical tumour. More distally a similar cyst, or group of cysts, may be left in the midst of the



FIG. 278. A bronchogenic cyst containing a fluid level of mucus, and situated in the anterior segment of the left upper lobe. It fluctuated in size with respiration and caused breathlessness. Its wall contained cartilage and elastic tissue and it was lined with cuboidal epithelium.

lung itself. These cysts commonly have a bronchial connection, and are therefore either empty or contain some mucus showing a fluid level in X-rays; their perfectly round fine walls are also revealed so that they are at once recognisable as cysts (Fig. 278). Those cysts in continuity with the main bronchus are prone to become infected, but more distal ones are not. In the former, the mucus becomes purulent and the cyst behaves

Single vascular hamartomas occur in which a fistula exists between a pulmonary artery and vein. A cavernous hæmangioma results from the tortuous aneurysmal distension of the adjoining vessels and gives rise to an X-ray shadow, which on screening or kymography is shown to be pulsatile. The short-circuit thus produced causes unoxygenated blood from the pulmonary artery to pass direct into the pulmonary vein and so to the systemic circulation. As much as half the blood volume may pass through the fistula. This results in dyspnœa, cyanosis, polycythæmia, and clubbing of the fingers and toes, and consequently suggests a congenital cardiac defect. If such a lesion is auscultated, a systolic murmur is often audible, which, in contrast with all cardiac systolic murmurs, gets louder on inspiration, when blood is sucked into the "hamartoma" by the concertina action of the lung. The presence of this syndrome is an urgent indication for excision of the lesion, if necessary, by lobectomy.

## CHAPTER 30

### CYSTS OF THE LUNG

**Congenital Bronchogenic Cysts.** An aberration of the outgrowing lung bud may occur at any point. If proximal, a solitary cyst in the mediastinum results, and it is attached to the bifurcation of the trachea by a stalk. It usually has no bronchial communication and, being full of mucus, appears on the radiograph as an opaque spherical tumour. More distally a similar cyst, or group of cysts, may be left in the midst of the



FIG. 278. A bronchogenic cyst containing a fluid level of mucus, and situated in the anterior segment of the left upper lobe. It fluctuated in size with respiration and caused breathlessness. Its wall contained cartilage and elastic tissue and it was lined with cuboidal epithelium.

lung itself. These cysts commonly have a bronchial connection, and are therefore either empty or contain some mucus showing a fluid level in X-rays; their perfectly round fine walls are also revealed so that they are at once recognisable as cysts (Fig. 278). Those cysts in continuity with a main bronchus are prone to become infected, but more distal ones are not. In the former, the mucus becomes purulent and the cyst behaves



henceforward exactly like a chronic epithelialised lung abscess, although the sharp definition of the delicate wall as seen in X-rays is not lost and there is little or no inflammatory reaction outside it. The patient suffers from recurrent fever and coughs up purulent sputum and sometimes blood. Most cysts are about 2 or 3 inches in diameter, fluctuating a little in size with respiration, but larger ones when infected may be mistaken



FIG. 279. A congenitally cystic lung.

for loculated empyemas, and may be aspirated or drained. Aspiration is likely to result in a true empyema, as the pleura is contaminated, whereas drainage of a large infected cyst produces a persistent sinus which continues to discharge until the true character of the condition is recognised and the cyst excised.

If developmental disturbances take place more distally still in the ultimate twigs of the bronchial tree, multitudinous small cysts may replace a lobe or even the whole of one lung (Fig. 279). It is this condition which has so often been mistaken for "congenital" bronchiectasis, but in congenital cystic dilatation of the bronchi, peribronchial inflammatory change is wanting; the cysts tend to be bigger and much more uniform than in cystic bronchiectasis, and other congenital defects are often present, as, for instance, in Kartagena's syndrome in which a polycystic right upper lobe is combined with dextrocardia (Fig. 280). Bronchiectasis is an acquired condition, even if it is acquired early in life. It is, of course, possible for a congenitally cystic lobe to become secondarily infected,

when it will give rise to all the symptoms of chronic bronchiectasis; hæmoptysis may occur and, as the lobe is functionless, breathlessness may result. Children with congenital lung cysts are often undersized and "asthmatic"; they have asymmetrical chests and may show early clubbing. The right lung is more commonly affected than the left.

Histologically, bronchogenic cysts can be distinguished from all other cysts by their lining of cuboidal or columnar epithelium, and the presence in their walls of bronchial elements, fibrous and elastic tissue, mucus glands and fragments of cartilage. Kindred anomalies such as conjoined and supernumary ribs, aberrant pulmonary vessels and unusual segmental fissures are also strong evidence of a congenital origin.

Bronchogenic cysts should be excised with careful closure of any bronchial communication. Anomalous vessels may add to the operative risk, but if their presence is anticipated the hazard is diminished. Even long infection produces little change in the character of the cyst wall and surrounding pneumonia is generally absent. Grouped cysts may be removed by segmental resection, but in the case of honeycombed lobes or lung, lobectomy or pneumonectomy is necessary.

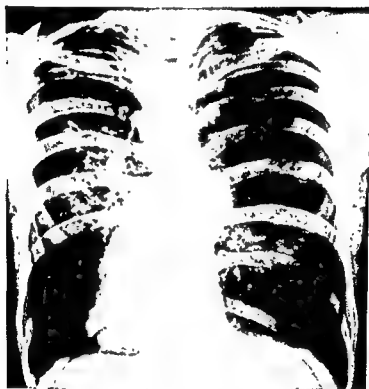


FIG. 280. Kartagena's syndrome: Dextrocardia associated with a congenitally cystic right upper lobe.

**Acquired Alveolar "Cysts" and Spontaneous Pneumothorax.** In contrast with the foregoing cysts, alveolar cysts are really pseudocysts or ruptures of the alveolar pattern to which a positive pressure is added, causing them to balloon (Fig. 281). They are much commoner than congenital cysts. They are associated with degenerative or infective conditions of the lung and often rupture, causing spontaneous pneumothorax (Fig. 282).

The lung is like an elastic sponge inside an accordion. Even under physiological conditions it is possible to raise the intrapulmonary pressure sufficiently to rupture an alveolar wall, and just as a hole punched in an elastic sheet increases when the sheet is stretched so does the cavity produced in the lung. An air cyst is created compressing surrounding

alveoli. If the elastic tissue in the walls of these alveoli is deficient or degenerate, they rupture into the original cyst, increasing its size; also the centrifugal negative suction of the pleural space tends to inflate the cyst still further. To all this may be added the potent force of a check valve mechanism, for if the lumen of the terminal bronchiole communicating with the alveolar cyst is scarred by old disease, such as tuberculosis, or

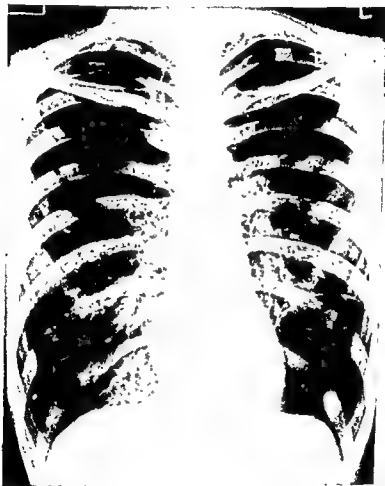


FIG. 281. A ballooning alveolar cyst at the left base.

narrowed by fresh infection, as in staphylococcal pneumonia, air admitted to the cyst during inspiration (when the bronchi dilate) may no longer be able to escape when the bronchi contract on expiration. This phenomenon is accentuated during the deep inspiratory gasp which precedes coughing. As the cyst balloons, it compresses the adjacent bronchioles, increasing the obliquity with which they enter the cyst, and thus worsening the degree of the check-valve mechanism so that the pressure within the cyst becomes more and more positive; its size increases with each gasp and more and more lung is displaced before it as it swells. It may finally fill the thorax and displace the mediastinum, but more often it eventually reaches a

state of equilibrium, when the positive pressure within it is sufficient to force a leak back into a bronchus; the size of the cyst then remains fairly stable, increasing only as neighbouring alveolar walls break down to join the cyst. Clearly the more diseased or degenerate the lung parenchyma, the worse and more generalised will be such cyst formation, but if the initial breakdown is due to some purely local factor in an otherwise healthy lung the pneumatoceles may be small and strictly localised. They are artificially divided into blebs and bullae; the former are subpleural collections of air caused by rupture of alveoli immediately beneath the visceral pleura, and the latter result from confluence of one alveolus with another, suggesting more generalised tissue damage; on the other hand, blebs, which are often seen clustering around apical adhesions or old tuberculous scarring, may be due to purely local stresses. The rupture of either results in a spontaneous pneumothorax, and if, as is usual, the cause persists, repeated leaks result in recurrent pneumothorax, and in conditions affecting both lungs rupture occurs first on one side and then on the other.

The commonest underlying conditions are: (1) The small healed tuberculous scar, usually, but by no means always, confined to one apex and found in most healthy subjects; (2) interstitial emphysema, a progressive generalised condition probably due to a congenital deficiency of elastic tissue in the lung, becoming clinically manifest in young adults; or (3) chronic degenerative emphysema so commonly seen in elderly men, associated with chronic bronchitis and sometimes hyperpneumonia. Although the latter variety of emphysema is so widespread, indeed almost a universal accompaniment of senility, it does not often cause pneumothorax although bullae are frequently present. This is because the layers of the pleura have become adherent owing to repeated mild attacks of inflammation. The majority of cases of spontaneous pneumothorax occur in young adults, and are due to one or other of the first two conditions.

In infancy, a remarkable and common cause of rapidly ballooning pneumatoceles, which frequently rupture into the pleural cavity, is



FIG. 292. Pneumothorax in a newly born infant associated with staphylococcal infection.

alveoli. If the elastic tissue in the walls of these alveoli is deficient or degenerate, they rupture into the original cyst, increasing its size; also the centrifugal negative suction of the pleural space tends to inflate the cyst still further. To all this may be added the potent force of a check valve mechanism, for if the lumen of the terminal bronchiole communicating with the alveolar cyst is scarred by old disease, such as tuberculosis, or

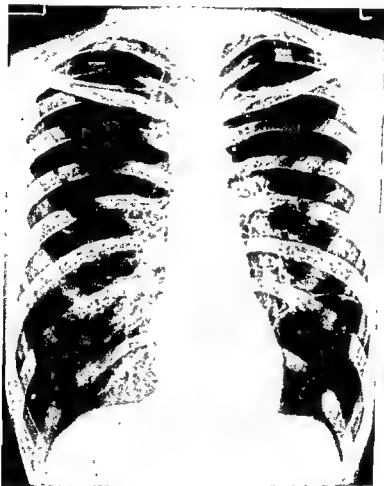


FIG. 281. A ballooning alveolar cyst at the left base.

narrowed by fresh infection, as in staphylococcal pneumonia, air admitted to the cyst during inspiration (when the bronchi dilate) may no longer be able to escape when the bronchi contract on expiration. This phenomenon is accentuated during the deep inspiratory gasp which precedes coughing. As the cyst balloons, it compresses the adjacent bronchioles, increasing the obliquity with which they enter the cyst, and thus worsening the degree of the check-valve mechanism so that the pressure within the cyst becomes more and more positive; its size increases with each gasp and more and more lung is displaced before it as it swells. It may finally fill the thorax and displace the mediastinum, but more often it eventually reaches a

causing breathlessness, it should be removed, even if there is radiological evidence of lesser bullae elsewhere. It is sometimes difficult to distinguish such a "cyst" from a pneumothorax, since in both, lung markings are absent. On close examination, however, one can usually see fine trabeculae crossing a pneumatocele, or distinguish the curvature of its edge at its upper or lower pole. Bullae of this kind, of course, have no lining membrane and no wall except the layer of compressed lung around them, but it is often possible to dissect out this condensation of tissue and almost enucleate the "cyst." At its base a number of bronchial openings will be found and these must be carefully closed. Sometimes a whole lobe is found to be completely replaced by two or three large intercommunicating cysts: lobectomy should then be performed. Such operations are of great benefit and do much to relieve dyspnoea, but it must not be forgotten that the lung left behind is defective and the eventual prognosis is bad.

**Hydatid Cysts of the Lung.** About 25 per cent. of hydatid cysts occur in the lung. As a cyst grows it sometimes causes pain or dyspnoea. Its adventitious layer may cause pressure erosion of a bronchus. Air then separates the true from the false membrane, occasionally producing a characteristic translucent crescent in radiographs. If a cyst is small it may now be coughed up and the patient cured, or a cyst may rupture, so that scolices and hooklets are found in the sputum. The scolices may cause cysts elsewhere. Infection of a cyst often ensues, but does not always kill the hydatid which continues to bud off daughter cysts. Fever, hæmoptysis, and signs of pulmonary suppuration follow infection. Rupture also may occur into the pleural cavity resulting in hydro-pneumothorax and dissemination of daughter cysts over the pleura. Even calcification, which is occasionally seen in the cyst wall, does not necessarily mean that the parasite is dead.

Treatment is directed towards the complete removal of the parasite with maximum conservation of lung. Any procedure which may rupture or disseminate the cyst is avoided, e.g. induction of pneumothorax, transpleural aspiration or bronchoscopy. After accurate radiographic localisation the chest is opened where best access to the cyst can be obtained. The pleural space is completely packed off. Some surgeons use black towels so that spilled daughter cysts can be better detected. The cyst is carefully aspirated with a fine gauge needle attached to a two-way syringe. A small incision is then made directly down to the pericyst. The glistening white hydatid wall is then seen and can be removed without rupture. Any bleeding points on the interior of the pericyst are coagulated with diathermy and any bronchial leaks closed. As the lung re-expands the cavity will soon close of its own accord. Multiple cysts may be dealt with in like manner.

If a cyst has already ruptured and is infected, or if the cyst is very large and associated with bronchiectasis or hæmorrhage, lobectomy may be necessary, although every effort should be made to conserve as much healthy lung as possible.

staphylococcal pneumonia (see under *Abscess of the Lung*). Presumably small abscess cavities are subjected to sudden episodes of tension causing them to expand into pseudocysts. They may have a fluid level of staphylococcal pus in them, but are often quite empty. Many rupture into the pleural cavity causing a pyopneumothorax, but it is usually the positive pressure which causes trouble rather than the infection. The mediastinum is displaced to the opposite side of the chest, breathing becomes laboured and the child cyanosed. Insertion of a needle into the pleural space at once gives relief. The air should be aspirated in order to re-expand the lung. It is very rarely that any other surgical intervention is necessary.

*Spontaneous pneumothorax* occurring for the first time in a young adult is not necessarily an indication of active pulmonary tuberculosis, and calls only for removal of the air and restoration of the normal lung expansion; but if the condition is repeated, and still more if a long history of collapses, sometimes on one side, sometimes upon the other, is elicited, an intercostal thoracotomy should be performed upon the most troublesome side and a search made for the cause. In unilateral cases this is likely to be a small bleb at one apex, related to a pleural adhesion or to some old scarring; often the leaking hole is still visible. The base of the bleb, or group of blebs, is then clamped and cut and the raw area oversewn. If the rest of the lung is perfectly healthy it can be re-expanded and the chest closed. When, however, it is found that blebs and small bullæ are distributed over the surface of the lung and are unrelated to scars, the condition is likely to be one of chronic interstitial emphysema and recurrent pneumothorax is to be expected on either side. Close examination often reveals fine air bubbles seething up between the alveolar lobules as the lung expands, clearly indicating interstitial deficiency. If a ruptured bleb is found it should be undersewn with a mattress suture. The whole layer of the parietal pleura is then stripped off the chest wall and mediastinum and all bleeding points coagulated. Two water-sealed drains are then inserted, one at the top of the thorax to remove air, and one at the bottom for any effusion. Both are connected to low-pressure suction; they may be removed after twenty-four to forty-eight hours. When the lung is fully expanded the chest is closed. By this means the visceral layer of the pleura becomes adherent to the chest wall so that a pneumothorax is no longer possible. Treatment of recurrent pneumothorax by inducing chemical pleurisy with insufflated iodised talc or silver nitrate, and subsequently withdrawing air in the hope of making the lung adhere to the chest wall, besides being extremely painful, is highly unreliable.

The same principles are applied in the treatment of *persistent pneumothorax*, which is commoner in older subjects. If prompt and complete re-expansion of the lung is not secured by aspiration, the chest should be opened and the leak dealt with. The longer such a lung remains collapsed the less likely is re-expansion to be obtained.

When a giant bulla is present, compressing the rest of the lung and

cardiac displacement. Eventually all these manifestations may be produced by mediastinal obstruction.

Investigation of mediastinal tumours begins with postero-anterior, lateral, and if necessary, oblique X-rays. Screening is essential for observing mobility and pulsation. A barium swallow demonstrates displacement or obstruction of the oesophagus. Tomography may be



FIG. 283. The linear shadow in the right lung field is the edge of the left lung which has herniated across the mediastinum following a lobectomy on the right.

helpful in determining exact relationships and in demonstrating cavitation. Bronchoscopy and oesophagoscopy sometimes reveal extrinsic pressure on the structure examined, but are chiefly valuable in the diagnosis or exclusion of intrinsic carcinomas. Induction of an artificial pneumothorax is sometimes carried out to distinguish between a pulmonary and a mediastinal mass; thus in carcinoma of the bronchus the mass falls away from the chest wall with the lung, whereas in mediastinal tumours it remains fixed. Pneumoperitoneum is occasionally of use in defining the relationship of masses abutting on the diaphragm. Thoracoscopy is seldom of value.



## CHAPTER 40

### CYSTS AND TUMOURS OF THE MEDIASTINUM

No part of the body is subject to so great a variety of cysts and tumours—some congenital and many acquired—the differential diagnosis of which embraces every endothoracic disorder. Aneurysms are distinguished from new growths by physical signs, positive serological tests for syphilis, and radiography (fluoroscopy shows an expansile mass and angiography demonstrates continuity with the great vessels). In patients with long-standing cardiospasm, the œsophagus becomes greatly distended and S-shaped, bulging to the left in its lower part, and to the right in its upper part, where it may closely resemble a right-sided mediastinal tumour. The history of dysphagia and demonstration of the dilated œsophagus by a barium swallow elucidate the diagnosis.

By similar methods diaphragmatic hernias, in or beside the mediastinum, are recognised. Herniation of a lung across the midline may resemble a cyst wall (Fig. 283). Scoliosis or vertebral disease causes distortions of the normal radiological appearances. Interpretation of chest X-rays in infants is often extremely difficult because of the mobility of the mediastinal structures and the marked alterations produced by minor rotation. Encysted effusions or empyemas may closely resemble tumours, and carcinoma of the bronchus must constantly be borne in mind in the diagnosis of any abnormal mediastinal shadow. The profusion of lymphatic tissue provides an extensive field for metastatic tumours, masses due to tuberculous adenitis or abscesses, and diseases such as the reticuloses, lymphosarcoma and lymphadenoma.

When all other possibilities have been considered, and eliminated, the wide field of intrinsic mediastinal cysts and tumours remains. Of them all, some 10 to 15 per cent. are likely to undergo malignant change; cysts are prone to secondary infection and may rupture into the pleural cavity or into the lung with fatal consequences; they may establish fistulous communications with the bronchi and behave thereafter as chronic abscesses; enterogenous cysts may ulcerate and perforate; hydatid cysts may disseminate; intrathoracic goitres sometimes cause respiratory and venous obstruction due to sudden hæmorrhage into a cyst, and some become secondarily toxic; 80 per cent. of thymomas are associated with myasthenia gravis and all should be regarded as malignant. Any type of mediastinal growth may cause dyspnoea by pressure upon bronchi or the trachea, or by the space occupied in the pleural cavity. Dysphagia may arise from compression of the œsophagus. Venous engorgement and tachycardia may be produced by obstruction of the great veins and

however, tend to respond much less well to neostigmine before operation and recover less satisfactorily afterwards. It is imperative to treat the tumours with high-voltage irradiation before proceeding to remove them and the gland. When a mediastinal tumour unaccompanied by myasthenic symptoms proves at operation to be a thymoma, deep X-rays should be used subsequently. In view of the much more serious prognosis, it is important to determine in every instance of myasthenia gravis whether or not a tumour is present before the surgical programme is begun.

In postero anterior X-rays a thymoma may be invisible behind the sternal shadow, but if large it sometimes appears as a lobulated mass on one or both sides of it. Lateral films show a thymoma characteristically flattened against the back of the sternum in the upper part of the anterior mediastinum. In contrast to retrosternal goitres, the convexity of the superior pole of the tumour is visible, and thymomas are usually more irregular in outline than the smoothly shaped dermoids which are found a little lower in the front of the mediastinum. This subject, including thymectomy, is further discussed under the Surgery of the Endocrine Glands, Chap. 70.

**Dermoids and Teratomas.** These tumours are almost always found in the anterior mediastinum lying between the back of the sternum and the pericardium. They probably arise from islands of primitive cells carried down by the descent of the heart and great vessels from the point of union of the more caudal branchial arches. Dermoids are derived principally from ectodermal elements whereas teratomas are derived from all three germ layers. Consequently dermoids are usually simple unilocular cysts containing thick gelatinous mucus and sebaceous matter. Ridges protruding inward from the inner wall sometimes contain punched-out lacunæ of tooth buds and sometimes sprout clumps of lank hair. Neural and retinal tissue, both being ectodermal in origin, may also be present. Accessory dermoids are sometimes found nearby on the pericardium, or situated anterior to the sternum, and connected to the main cyst by a stalk in collar-stud fashion. Teratomas are generally solid or polycystic and they are much more complex in structure, containing glandular and skeletal elements.

These cysts and tumours tend to increase in size during adolescence and early adult life, and it is then that they are apt to produce symptoms of mediastinal obstruction. Blood-borne secondary infection of a cyst is common. If the infection is mild it may cause a pyrexia of unknown origin ("P.U.O.") and malaise; a great increase in the adhesions already binding the tumour to surrounding structures results. If the infection is severe the cyst may rupture into the pleura, the pericardium or a bronchus. A bronchial fistula sometimes occurs without evident infection of a cyst and the contents of the cyst are expectorated. Thus hair or sebaceous matter appears in the sputum, confirming the diagnosis; they may also be found on attempted aspiration of the pleura. Once a fistula

When all evidence has been considered, it is seldom possible to be certain of the nature of an abnormal mediastinal mass. It is folly to wait for complications (*vide supra*), or for a symptomless mass to produce symptoms. It is folly to allow something easily removable with a negligible mortality to become perhaps irremovable. Therefore exploration should be undertaken without delay.

Increasing use of chest radiography (including mass radiography) has shown that mediastinal cysts and tumours are common, although the relative frequency of the various lesions has not been determined. If retrosternal goitre is included it probably ranks first; but ganglioneuromas are next and are then followed by bronchogenic cysts, dermoids and teratomas, various tumours of lymphoid tissue, thymomas, pericardial cysts and enterogenous cysts. Of 105 mediastinal tumours removed in the United States Army hospitals in three years 90 were innocent and 15 malignant. Among the innocent ones were 29 neuromas (ganglioneuromas), 23 bronchogenic cysts, 14 dermoids and teratomas and 10 pericardial cysts; the malignant group included 6 teratomas, 4 cases of Hodgkin's disease, 2 lymphoblastomas, 2 thymomas and 1 neurosarcoma.

The common tumours of the *anterior mediastinum* in order from top to bottom are: (1) Intrathoracic goitres, (2) thymomas, (3) dermoids and teratomas, (4) pericardial coelomic cysts. They must be distinguished from carcinomas of the bronchus, growths of the back of the sternum, aneurysms of the first part of the aorta and diaphragmatic hernias through the foramen of Morgagni. Lipomas are rare but are also found anteriorly, related to the cardio-phrenic fold.

In the *posterior mediastinum* the following tumours occur: (1) Ganglioneuromas, (2) enterogenous cysts, and (3) neuroblastomas (in infants). These tumours must be distinguished particularly from carcinomas of the bronchus, encysted effusions or pus, aneurysms of the descending aorta, passive distension of the oesophagus, posterior congenital diaphragmatic hernias, and tumours or abscesses associated with the vertebrae.

In the *central mediastinum* occur: (1) Most of the lymphoid tumours and (2) bronchial cysts. Carcinoma of the bronchus and metastatic deposits in the lymph glands give rise to the main diagnostic difficulties.

**Substernal Goitre** (see Chap. 69). True ectopic thyroids, in which there is no connection with the normally placed gland and correspondingly no vascular supply from the neck, are exceedingly rare and cannot be diagnosed prior to removal.

**Thymomas.** Thymomas are malignant epithelial tumours of the thymus which at first appear to be encapsulated but later erupt, infiltrate surrounding tissue, and eventually metastasize. Their association with myasthenia gravis first led to their removal, and subsequently to thymectomy in patients without a tumour. Thymomas occur in about 15 per cent. of all cases of myasthenia, and myasthenia is present in 75 per cent. of patients with thymomas. The small group who have thymomas,

attachments would be out of reach. Removal may present great difficulty if infection has resulted in the formation of dense and tough adhesions to the parietes and the origins of the great vessels. Dissection in the region of the latter could then result in serious hæmorrhage. Malignant change may add to the hazards, but the aim of surgery should always be complete excision and never the marsupialisation once practised.

**Pericardial Cœlomic Cysts.** Simple, rather lax cysts lined by a single layer of endothelium and filled with clear fluid are not uncommonly found lying anteriorly in the cardiophrenic angle. Seventy per cent. of them are situated on the right side. They may have a pedicle attaching them to the pericardium or even a narrow communication through which fluid can be squeezed. Most of these cysts however have no attachment but lie related to the pericardial fat in the cardiophrenic angle or attached to the anteromedial part of the diaphragm. Because of the clear pericardial fluid they contain they are sometimes called "spring water" cysts. They probably originate by being pinched off from the pneumo-enteric recess of the pleuroperitoneal cavity by the elements of the primitive diaphragm. As a rule they remain symptomless, but if they are large enough dyspnoea results from displacement of the lung. They may then be repeatedly aspirated as "recurrent pleural effusions." If found on routine radiography they are difficult to distinguish from anterior diaphragmatic hernias containing omentum. The induction of a pneumoperitoneum usually resolves this doubt by neatly demonstrating their relationship to the diaphragm (Fig. 285). Most of these cysts are flaccid so that their outline as seen in X-rays is not like that of most cysts but flattened. No instance of infection or of malignant change has been reported. They are easily removed by thoracotomy (usually on the right side).

**Lipomas.** These are rare tumours which occur most often in the cardiophrenic angle anteriorly. They are typically lobulated and may attain a massive size, causing breathlessness.

**Neurogenic Tumours.** Ninety per cent. of posterior mediastinal tumours are neurogenic and arise from the sympathetic chain or its ganglia. For this reason their occurrence elsewhere than in the paravertebral gutter is very rare. There are two tumours which arise from the sympathetic nervous system, the neuroblastoma and the ganglioneuroma. *Neurofibromas* occasionally grow in relationship to the intercostal nerves. They may be single, or they may be multiple (Von Recklinghausen's disease). The phrenic and vagus nerves are sometimes also involved and the chain of lobulated tumours on them can give rise to mediastinal obstruction and be mistaken for malignant disease of the mediastinum. *Neurofibromas* in Von Recklinghausen's disease are much more likely to undergo sarcomatous change than solitary neurofibromas or ganglioneuromas.

*Ganglioneuromas* are rather commoner in the upper than the lower part of the paravertebral gutter, but may occur anywhere along it

is established infection is inevitable and the cyst behaves thereafter as a chronic abscess. Malignant change has been estimated to occur in some 13 per cent. of these cysts and tumours. The proportion is greater in teratomas than in dermoids; thus 70 per cent. of malignant growths arise in the former and 30 per cent. in the latter.

Apart from the expectoration or aspiration of characteristic material (*vide supra*), they are recognised radiologically by their usually ovoid or lobulated shadow in the anterior mediastinum, bulging to one or other side of the sternum, and having, prior to infection, a clearly defined smooth edge (Fig. 284). Successive attacks of inflammation reduce this sharpness

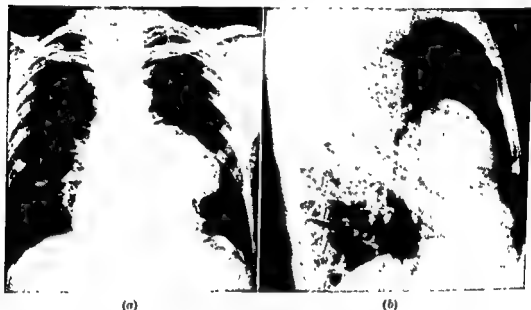


FIG. 284. A mediastinal dermoid cyst. (a) P.A. view. (b) The lateral view demonstrates its anterior situation.

of outline until the tumour resembles an invasive carcinoma. If a fistula is present a fluid level is visible. Teeth can sometimes be seen in X-rays or, more often, there is a regular line of punched-out translucencies caused by teeth buds in the denser germinal ridges. Calcification commonly occurs in the walls of dermoids and must be distinguished from that seen in constrictive pericarditis, goitres, tuberculous lesions or in the wall of a hydatid cyst.

Exploratory puncture should never be done lest it carry infection or, worse still, malignant cells into the pleural cavity. Early exploratory thoracotomy is indicated. This is best carried out through the standard posterolateral incision in the fifth or sixth left interspace. The lung is covered with a wet pack and drawn backwards and the mediastinal parietal pleura is incised anterior to the phrenic nerve. The tumour is inspected and its nature confirmed. It is important to distinguish it from a thymoma or a large retrosternal goitre both of whose vascular

attachments would be out of reach. Removal may present great difficulty if infection has resulted in the formation of dense and tough adhesions to the parietes and the origins of the great vessels. Dissection in the region of the latter could then result in serious hemorrhage. Malignant change may add to the hazards, but the aim of surgery should always be complete excision and never the marsupialisation once practised.

**Pericardial Cœlomic Cysts.** Simple, rather lax cysts lined by a single layer of endothelium and filled with clear fluid are not uncommonly found lying anteriorly in the cardiophrenic angle. Seventy per cent. of them are situated on the right side. They may have a pedicle attaching them to the pericardium or even a narrow communication through which fluid can be squeezed. Most of these cysts however have no attachment but lie related to the pericardial fat in the cardiophrenic angle or attached to the anteromedial part of the diaphragm. Because of the clear pericardial fluid they contain they are sometimes called "spring water" cysts. They probably originate by being pinched off from the pneumato-enteric recess of the pleuroperitoneal cavity by the elements of the primitive diaphragm. As a rule they remain symptomless, but if they are large enough dyspnoea results from displacement of the lung. They may then be repeatedly aspirated as "recurrent pleural effusions." If found on routine radiography they are difficult to distinguish from anterior diaphragmatic hernias containing omentum. The induction of a pneumoperitoneum usually resolves this doubt by neatly demonstrating their relationship to the diaphragm (Fig. 285). Most of these cysts are flaccid so that their outline as seen in X-rays is not like that of most cysts but flattened. No instance of infection or of malignant change has been reported. They are easily removed by thoracotomy (usually on the right side).

**Lipomas.** These are rare tumours which occur most often in the cardiophrenic angle anteriorly. They are typically lobulated and may attain a massive size, causing breathlessness.

**Neurogenic Tumours.** Ninety per cent. of posterior mediastinal tumours are neurogenic and arise from the sympathetic chain or its ganglia. For this reason their occurrence elsewhere than in the paravertebral gutter is very rare. There are two tumours which arise from the sympathetic nervous system, the neuroblastoma and the ganglioneuroma. *Neurofibromas* occasionally grow in relationship to the intercostal nerves. They may be single, or they may be multiple (Von Recklinghausen's disease). The phrenic and vagus nerves are sometimes also involved and the chain of lobulated tumours on them can give rise to mediastinal obstruction and be mistaken for malignant disease of the mediastinum. *Neurofibromas* in Von Recklinghausen's disease are much more likely to undergo sarcomatous change than solitary neurofibromas or ganglioneuromas.

*Ganglioneuromas* are rather commoner in the upper than the lower part of the paravertebral gutter, but may occur anywhere along it

(Fig. 286). They never cause segmental pain : if a tumour otherwise having the appearance of a ganglioneuroma is associated with pain it must be assumed to be a bronchial carcinoma. Since ganglioneuromas arise behind the parietal pleura, they are tightly bound by it to the posterior chest wall and the resultant pressure eventually causes widening of the related intercostal space, and sometimes smooth erosion of the ribs above

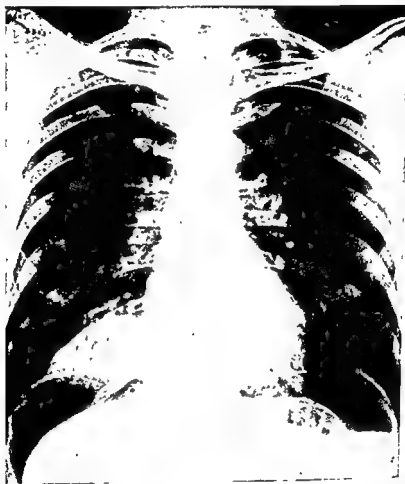


FIG. 285. A pericardial cœlomic cyst. A pneumoperitoneum has been induced to show relation of the cyst to the diaphragm.

and below. Very rarely, part of the tumour may pass through a neural foramen into the spinal canal and result in compression of the cord. Radiologically they show a smoothly rounded "cannon-ball" outline with a sharply defined edge (Fig. 286), and possibly also the rib changes already mentioned. If doubt exists as to whether a tumour is inside or outside the lung, induction of an artificial pneumothorax solves the problem by causing the lung to fall away from the posterior chest wall.

Ganglioneuromas usually give rise to no symptoms but, in the thorax, if they become very large they may cause breathlessness, or if they extend

into the spinal canal paraplegia may result. They have to be distinguished first of all from bronchial carcinomas, which sometimes have surprisingly well-defined edges. Carcinomas of the bronchus lie in the lung and move with it unless they are invading the chest wall, as indicated by continuous intractable pain. Unruptured bronchogenic cysts or even lung abscesses may resemble ganglioneuromas, but they also are in the lung. Furthermore, abscesses are accompanied by malaise and fever and have less well-defined edges. Encysted effusions are not usually as spherical as ganglioneuromas, and a history of past chest infection is usual. Aspiration of the contents will provide a solution. Enterogenous cysts have their own well-defined characteristics, which are described later.

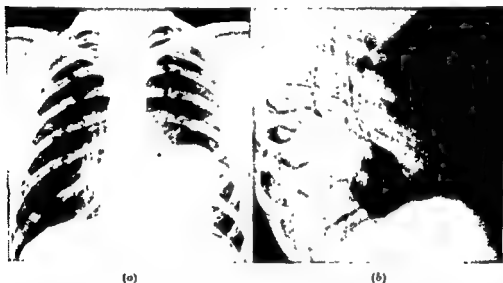


FIG. 286. A typical ganglioneuroma of average size. (a) P.A. view. (b) The lateral view demonstrating its position in the paravertebral gutter.

*Neuroblastomas* are described in Chap. 60.

The removal of neurogenic tumours does not as a rule present any great difficulty and is performed through a posterolateral intercostal thoracotomy unless they are so small that they can be enucleated by a direct extrapleural approach. They at first appear firmly fixed to the chest wall, but as soon as the parietal pleura over them is incised they can be rapidly shelled out and are attached only by a short pedicle to the paravertebral gutter. Tumours having a dumb-bell extension through an intervertebral foramen into the spinal canal are removed by laminectomy followed a few weeks later by thoracotomy.

**Enterogenous Cysts.** The majority of these cysts are seen in infants under one year. They are rarely found in adults. Similar in origin to bronchial cysts, they arise as diverticula from the foregut during foetal development and possess a mucosal lining resembling some part of the



gut, usually the stomach. If there is much active peptic secretion the cyst is virtually an accessory but isolated stomach which rapidly distends. It may interfere with respiratory function, causing increasing dyspnoea and cyanosis, and finally death. If it ulcerates and bleeding follows, hæmoptysis, hæmatemesis or mælæna ensue. On the other hand, if little or no actively secretory peptic mucosa is included, a silent mucus-filled cyst persisting into adult life results.

Enterogenous cysts are found closely related to the œsophagus in the paravertebral gutter and almost always on the right side—presumably

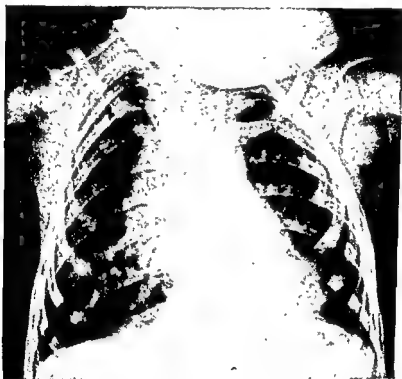


FIG. 287. A large symptomless enterogenous cyst in a child aged four years. It was lined by mucosa closely resembling that of the stomach.

because of the presence of the aorta upon the left (Fig. 287). They may be densely adherent to the parietes, and the wall of the cysts found in infancy are sometimes as thick as that of the stomach and include mucosal and muscular layers. On the rare occasions when such a cyst is seen in adults the cyst wall is thinner. Once a cyst ruptures into a neighbouring hollow viscus a fluid level at once appears in the cyst. Removal is life saving in infancy and is likely to precede the correct diagnosis in the adult.

**Tumours of Lymphoid Tissue.** In view of the profusion of lymphoid tissue it is not surprising that it is often affected both by tumours and granulomas, and by generalised lymphatic disorders. The commonest of these is undoubtedly *tuberculous adenitis*, which always accompanies a primary tuberculous infection, but is then usually transitory. Glandular

enlargement also occurs in the bronchopneumonic complications of measles and whooping-cough in infancy. As at this period of life the bronchi are soft and pliable, the enlarged glands, being closely disposed around the bronchi and disproportionately large and firm, often compress the bronchi and cause atelectasis. If distal infection ensues, the corresponding lung segment becomes bronchiectatic. In later life, tuberculous adenitis may resemble a craggy mediastinal tumour and be associated with a cold abscess; or the glands may ulcerate into neighbouring bronchi, giving rise to serious hæmoptyses. Such glands, when calcified, may actually be roughed up as "broncholiths." Other evidence of tuberculosis, especially the presence of cervical tuberculous adenitis, greatly assists diagnosis.

*Metastatic Deposits.* These most often arise from bronchial cancer; they are exceedingly common and may be more conspicuous than the primary growth. They commonly cause hoarseness from recurrent laryngeal paralysis on the left side, phrenic palsies with paradoxical movement of one or other diaphragmatic dome, dysphagia, or cervical venous distension.

*Lymphosarcoma, lymphadenoma* (syn. Hodgkin's disease), and the *reticulosæ* affect the mediastinal glands, and the first two diseases may be confined to them. If this is so, diagnosis rests on the characteristic blood picture, and Pel-Ebstein pyrexia of lymphadenoma. In other cases, biopsy of an accessible gland in the neck or elsewhere may be conclusive. Lymphosarcoma in young adults produces a massive smoothly lobulated shadow which usually responds rapidly to X-rays, but lymphoblastoma in children may not do so. All these diseases are ultimately fatal.

*Lymphangioma or cystic hygroma* is a rare mediastinal tumour. It is multilocular, intimately connected with surrounding tissues, profusely supplied by blood vessels and not encapsulated. Removal is apt to be a formidable procedure.

*Bronchogenic Cysts.* If a developmental fault occurs in an advancing lung bud a bronchial cyst may result. When this occurs proximally, the cyst is likely to be found in the central mediastinum attached to the carina by a stalk, but it may equally well occur at any other point on the pulmonary tree. A cyst may then be situated in the lung substance itself, or more distally still, cyst formation may result in a congenitally polycystic lobe. Although usually symptomless until secondary infection intervenes, very large cysts are sometimes seen in infancy, causing respiratory distress and cyanosis. If an infected cyst ruptures into the pleural cavity an empyema results and will not heal after the usual drainage; empyema has sometimes followed ill-judged attempts to aspirate an infected cyst for diagnostic purposes.

Radiologically, an unruptured cyst in the mediastinum presents merely as a smoothly rounded opacity and cannot be diagnosed with any certainty prior to thoracotomy. When a fistula is established it at once produces a fluid level, which is seen in X-rays in a rounded cavity with a thin regular

wall. This still has to be differentiated from a breaking-down carcinoma, an abscess (especially staphylococcal), the gut in a diaphragmatic hernia or an hydatid cyst. Bronchial cysts are lined by cuboidal or columnar epithelium containing mucus glands. The walls characteristically contain bronchial elements such as cartilage, elastic tissue and smooth muscle, in disorderly arrangement. Removal is indicated because of the uncertainty of the diagnosis and the risk of infection.

Much rarer tumours of the mediastinum not previously mentioned are *hæmangiomas*, which may be solitary or part of generalised familial telangiectasia, *parathyroid adenoma*, *plasmocytoma*, innocent or malignant, *fibroma* and *chondroma*.

## CHAPTER 41

# THE SURGERY OF PULMONARY TUBERCULOSIS

THE surgical treatment of pulmonary tuberculosis falls into two groups: (a) Those operations designed to relax the diseased lung and so allow it to heal by natural processes; and (b) those in which the object is to extirpate the disease by pulmonary excision. Relaxant operations are therefore an extension of the principles of medical treatment by surgical means. All have for their object the closure of chronic tuberculous cavities which have failed to heal following rest, artificial pneumothorax, pneumoperitoneum, or other measures. The test of the success of surgery is healing of the cavity and conversion of the sputum permanently from positive to negative on repeated and stringent culture for tubercle bacilli. If this criterion is not satisfied, the operation has failed and some other surgical measure must be undertaken.

Before the use of streptomycin and other effective antibiotics, resection of tuberculous lungs was not practicable, for such attempts were inevitably followed either by bronchopleural fistula or by dissemination of the disease. Relaxant operations were therefore often performed on quite unsuitable patients as they were virtually the only surgical methods available. With the use of modern drugs, however, the position of surgery has undergone a revolution, and with it the prognosis and mortality of pulmonary tuberculosis. Relaxant operations, although they still have a most useful role in properly selected cases, have fallen into second place. Thus excisions now account for about 80 per cent. of all operations for pulmonary tuberculosis.

## RELAXANT OPERATIONS

The lung resembles a three-dimensional elastic sponge and therefore relaxation to be effective must be accomplished in three dimensions. It is of little or no value merely to remove ribs, thus compressing the lung from side to side while leaving it stretched from apex to base, or to induce an artificial pneumothorax in the presence of apical adhesions. Indeed, as such adhesions are most likely to occur where the disease is worst, extra strain is thrown upon the very area most in need of relaxation and disaster is likely to ensue. On the other hand, effective relaxation of a lung is well demonstrated by a perfect artificial pneumothorax in which no adhesions are present or in which those existing at induction have been cut.

When a tuberculous lesion in the lung breaks down to form a cavity, three things tend to make it bigger. Firstly, the lung is itself elastic and

is held in equilibrium with the ebb and flow of the chest wall by a considerable negative pressure between the layers of the pleura. The effect of a gap in this balanced mesh is rather like one made in a gently stretched rubber sheet—a pin-prick soon becomes a wide gap. Secondly, the size of a cavity may increase by active tuberculous ulceration of its wall. Thirdly, the lumen of the bronchi expands on inspiration and contracts on expiration, so that if a bronchus is partly blocked or distorted by disease air may be able to enter a cavity during inspiration but be trapped within it when expiration occurs. Thus tension may soon be built up so that the cavity balloons with every inspiration, especially with the deep one which precedes a fit of coughing. It will be seen, then, that "relaxation" as applied to the closure of cavities implies a good deal more than mere "rest" for the lung, although rest of the patient plays a most important part in the treatment of tuberculosis.

The induction of artificial pneumothorax and pneumoperitoneum are minor surgical procedures usually performed by physicians, and it is therefore not proposed to discuss their techniques here. It is noteworthy that about 40 per cent. of tuberculous empyemas follow ill-judged induction of a pneumothorax. If pleural adhesions are present, as in the majority of cases, the surgeon divides them, for whereas cavities close in about 60 per cent. of perfect A.P.'s, they do so in only 25 per cent. of those with undivided adhesions. Although radiography shows the presence of adhesions, it cannot be relied on to indicate their extent or their divisibility. Therefore when adhesions are suspected or known to be present, thoracoscopy should be carried out.

**Thoracoscopy and Section of Adhesions.** An adequate degree of collapse must have previously been achieved. The patient's chest is prepared as for a major operation, including shaving the axillary hair. Omnopon  $\frac{1}{2}$  gr. (20 mg.) is given one hour beforehand. The patient is placed on the operating table in the lateral position similar to that adopted for induction of A.P. The body is arched over a sandbag or rubber pad, and the arm arranged to expose the axilla (Fig. 288). Two types of thoracoscope are in common use: (a) One with two cannulae, the telescope passing down one, the cautery down the other; and (b) one with a single cannula carrying both telescope and cautery. Usually the most convenient place to insert the instrument is high in the axilla. Sterile towels are arranged, and a suitable intercostal space is selected and infiltrated with local anaesthetic down to and including the pleura. The surgeon assures himself that sufficient pleural space exists at the point selected by measuring the distance of the lung from the chest wall by passing a light blunt probe down a short hollow needle. The skin is nicked over this point with a scalpel and the large trocar and cannula is then firmly thrust into the pleural space. The trocar is withdrawn, and replaced by a lighted telescope. The pleural cavity is now inspected (Fig. 289). If it is unsafe to cut the adhesions because of direct adherence



FIG. 288. The position for thoracoscopy.

of the lung to the chest wall or mediastinum, the thoracoscope is withdrawn, the puncture hole is sutured, a firm dressing is applied, and the patient returned to bed. As an A.P. in such circumstances is unlikely to achieve its end, and holds the threat of empyema, it will probably be soon abandoned and some other form of treatment adopted.

If, however, the adhesions are safely divisible, they are severed by the cautery as close to the chest wall as possible, or they may be enucleated from it to lessen the risk of burning the lung. Bleeding vessels in the

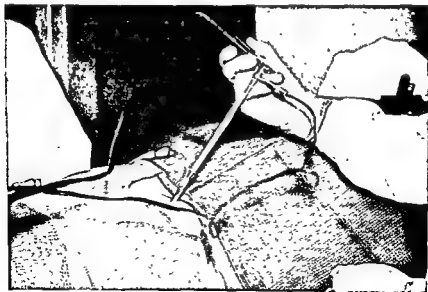


FIG. 289. The thoracoscope with its cautery has been passed through the cannula into the pleural cavity.

is held in equilibrium with the ebb and flow of the chest wall by a considerable negative pressure between the layers of the pleura. The effect of a gap in this balanced mesh is rather like one made in a gently stretched rubber sheet—a pin-prick soon becomes a wide gap. Secondly, the size of a cavity may increase by active tuberculous ulceration of its wall. Thirdly, the lumen of the bronchi expands on inspiration and contracts on expiration, so that if a bronchus is partly blocked or distorted by disease air may be able to enter a cavity during inspiration but be trapped within it when expiration occurs. Thus tension may soon be built up so that the cavity balloons with every inspiration, especially with the deep one which precedes a fit of coughing. It will be seen, then, that "relaxation" as applied to the closure of cavities implies a good deal more than mere "rest" for the lung, although rest of the patient plays a most important part in the treatment of tuberculosis.

The induction of artificial pneumothorax and pneumoperitoneum are minor surgical procedures usually performed by physicians, and it is therefore not proposed to discuss their techniques here. It is noteworthy that about 40 per cent. of tuberculous empyemas follow ill-judged induction of a pneumothorax. If pleural adhesions are present, as in the majority of cases, the surgeon divides them, for whereas cavities close in about 60 per cent. of perfect A.P.'s, they do so in only 25 per cent. of those with undivided adhesions. Although radiography shows the presence of adhesions, it cannot be relied on to indicate their extent or their divisibility. Therefore when adhesions are suspected or known to be present, thoracoscopy should be carried out.

**Thoracoscopy and Section of Adhesions.** An adequate degree of collapse must have previously been achieved. The patient's chest is prepared as for a major operation, including shaving the axillary hair. Omnopon  $\frac{1}{2}$  gr. (20 mg.) is given one hour beforehand. The patient is placed on the operating table in the lateral position similar to that adopted for induction of A.P. The body is arched over a sandbag or rubber pad, and the arm arranged to expose the axilla (Fig. 288). Two types of thoracoscope are in common use: (a) One with two cannulae, the telescope passing down one, the cautery down the other; and (b) one with a single cannula carrying both telescope and cautery. Usually the most convenient place to insert the instrument is high in the axilla. Sterile towels are arranged, and a suitable intercostal space is selected and infiltrated with local anaesthetic down to and including the pleura. The surgeon assures himself that sufficient pleural space exists at the point selected by measuring the distance of the lung from the chest wall by passing a light blunt probe down a short hollow needle. The skin is nicked over this point with a scalpel and the large trocar and cannula is then firmly thrust into the pleural space. The trocar is withdrawn, and replaced by a lighted telescope. The pleural cavity is now inspected (Fig. 289). If it is unsafe to cut the adhesions because of direct adherence

tuberculous pus into the opposite lung with consequent spread of the disease. The maintenance of the normal position of the heart and mediastinum after operation is also rendered difficult by unilateral paralysis of the diaphragm.

The diaphragm is the most mobile part of the thoracic wall and hence is very important from a physiological point of view. It is reprehensible to render it parietic, especially as the advantages gained from doing so are extremely uncertain, and there are usually more effective methods of obtaining the desired results. Therefore nowadays the phrenic nerve should never be crushed, for the patient's surgical future is thereby prejudiced.

**Thoracoplasty.** This operation, which used to account for 90 per cent. of all major surgery in pulmonary tuberculosis, remains the most effective operation in the relaxant group. Furthermore, modifications of it are widely used following pulmonary resections for tuberculosis and non-malignant conditions to prevent development of emphysema in residual lobes, and to maintain the mediastinum in its normal position after pneumonectomy. It permanently "converts" the sputum in 80 per cent. of properly selected cases of tuberculosis. Patients should be between the ages of twenty to forty years and suffering from a chronic tuberculous cavity which should be of moderate size, situated in the upper and outer thirds of the lung and irresponsive to other treatment. The opposite lung should either be free from disease or such disease as is present must be of very limited extent and under control. The patient should be gaining, or at any rate maintaining, weight, and the sedimentation rate should be low or declining. The patient's general condition should be otherwise satisfactory and there should be no breathlessness, for a full seven-rib thoracoplasty is likely to diminish the vital capacity by about one-third. The patient's morale must be good and he should be capable of understanding the implications of the operation, for his intelligent co-operation is of great importance. Other patients in whom thoracoplasty may be indicated are those with fibrocaseous disease causing deviation of the trachea and traction upon the opposite lung. It is also indicated after pulmonary resection in order to maintain the central position of the mediastinum, but for this purpose apicolysis is omitted and the first rib spared.

Removal of ribs results in a soft chest wall with paradoxical respiratory movements (i.e. sucking inwards on inspiration and blowing outwards with expiration). It follows that extensive removal of ribs at one time is incompatible with life. The patient would neither be able to breathe properly nor to expel the secretions in his lungs by coughing; therefore death would occur from drowning if not from respiratory failure. It is thus necessary that thoracoplasty must be performed in stages, usually at a fortnight's interval, so that the soft part of the chest wall may become more rigid in the intervening time and the patient is not over-



adhesions are coagulated. As the lung relaxes, further sheets or cords of adhesions become accessible and the patient is placed in whatever position is necessary to expose them, or the instrument is moved to another intercostal space. When the lung has been freed, the wound is closed and the intrapleural pressure adjusted.

During the operation a nurse is seated by the patient's head, beyond the anæsthetic screen, to keep operation sheets from his face, to observe his colour and respirations, and to reassure him. The post-operative posture is carefully supervised for the first twenty-four hours. If, for example, the adhesions were chiefly in the left axilla, the patient should lie on the right side to prevent the lung coming in contact with the raw area and forming new adhesions, or if the adhesions were attached to the left side of the mediastinum the patient is propped up and inclined to the left side. The nursing staff must therefore be informed of the desired posture in each instance. There is a greater likelihood of coughing air into the tissues than after induction of an artificial pneumothorax as the wound in the parietal pleura is larger and re-adherence of the lung, therefore, more likely. Coughing should be kept to a minimum by the use of suitable drugs and the patient told to press his hand firmly over the dressing when he finds he cannot suppress his cough. Vomiting has much the same effect as coughing. It is often caused by opiates which should therefore be used sparingly, or not at all if sensitivity is known to exist. If *surgical emphysema* occurs, as indicated by puffiness at the base of the neck and on the chest wall itself, with tenderness and a characteristic crackling sensation to light touch, a refill of the pneumothorax may be necessary to prevent expansion of the lung.

The pulse rate should be carefully charted during the first twelve hours, as hæmorrhage can occur from one of the divided adhesions or from the trocar wound. Should blood accumulate in the chest it must be aspirated completely, and the loss of blood, if considerable, replaced by transfusion. The patient is also taken back to the theatre for re-insertion of the thoracoscope and coagulation of bleeding vessels.

**Phrenic Crush.** In the event of an unsatisfactory A.P., or to reinforce the effect of a pneumoperitoneum, physicians have in the past often had recourse to phrenic paralysis to raise the level of the diaphragm and thus help to relax the affected lung. The nerve is found in the neck as it runs from without inwards over the surface of the scalenus anterior muscle and is crushed between the jaws of a pair of artery forceps. *The resulting palsy, however, although intended to be temporary, is frequently permanent.* This constitutes a grave handicap when, as is likely, the patient has later to undergo major surgery. Thus if a thoracoplasty or upper lobectomy is done the remaining lower lobe is very liable to collapse, for the patient is unable efficiently to cough up basal secretions. This is always serious, and particularly so in tuberculosis, as an atelectatic lobe is very likely to become infected. Also ineffective coughing may result in spill-over of

tuberculous pus into the opposite lung with consequent spread of the disease. The maintenance of the normal position of the heart and mediastinum after operation is also rendered difficult by unilateral paralysis of the diaphragm.

The diaphragm is the most mobile part of the thoracic wall and hence is very important from a physiological point of view. It is reprehensible to render it parietic, especially as the advantages gained from doing so are extremely uncertain, and there are usually more effective methods of obtaining the desired results. Therefore nowadays the phrenic nerve should never be crushed, for the patient's surgical future is thereby prejudiced.

**Thoracoplasty.** This operation, which used to account for 90 per cent. of all major surgery in pulmonary tuberculosis, remains the most effective operation in the relaxant group. Furthermore, modifications of it are widely used following pulmonary resections for tuberculosis and non-malignant conditions to prevent development of emphysema in residual lobes, and to maintain the mediastinum in its normal position after pneumonectomy. It permanently "converts" the sputum in 80 per cent. of properly selected cases of tuberculosis. Patients should be between the ages of twenty to forty years and suffering from a chronic tuberculous cavity which should be of moderate size, situated in the upper and outer thirds of the lung and irresponsive to other treatment. The opposite lung should either be free from disease or such disease as is present must be of very limited extent and under control. The patient should be gaining, or at any rate maintaining, weight, and the sedimentation rate should be low or declining. The patient's general condition should be otherwise satisfactory and there should be no breathlessness, for a full seven-rib thoracoplasty is likely to diminish the vital capacity by about one-third. The patient's morale must be good and he should be capable of understanding the implications of the operation, for his intelligent co-operation is of great importance. Other patients in whom thoracoplasty may be indicated are those with fibrocaseous disease causing deviation of the trachea and traction upon the opposite lung. It is also indicated after pulmonary resection in order to maintain the central position of the mediastinum, but for this purpose apicolysis is omitted and the first rib spared.

Removal of ribs results in a soft chest wall with paradoxical respiratory movements (i.e. sucking inwards on inspiration and blowing outwards with expiration). It follows that extensive removal of ribs at one time is incompatible with life. The patient would neither be able to breathe properly nor to expel the secretions in his lungs by coughing; therefore death would occur from drowning if not from respiratory failure. It is thus necessary that thoracoplasty must be performed in stages, usually at a fortnight's interval, so that the soft part of the chest wall may become more rigid in the intervening time and the patient is not over-

adhesions are coagulated. As the lung relaxes, further sheets or cords of adhesions become accessible and the patient is placed in whatever position is necessary to expose them, or the instrument is moved to another intercostal space. When the lung has been freed, the wound is closed and the intrapleural pressure adjusted.

During the operation a nurse is seated by the patient's head, beyond the anæsthetic screen, to keep operation sheets from his face, to observe his colour and respirations, and to reassure him. The post-operative posture is carefully supervised for the first twenty-four hours. If, for example, the adhesions were chiefly in the left axilla, the patient should lie on the right side to prevent the lung coming in contact with the raw area and forming new adhesions, or if the adhesions were attached to the left side of the mediastinum the patient is propped up and inclined to the left side. The nursing staff must therefore be informed of the desired posture in each instance. There is a greater likelihood of coughing air into the tissues than after induction of an artificial pneumothorax as the wound in the parietal pleura is larger and re-adherence of the lung, therefore, more likely. Coughing should be kept to a minimum by the use of suitable drugs and the patient told to press his hand firmly over the dressing when he finds he cannot suppress his cough. Vomiting has much the same effect as coughing. It is often caused by opiates which should therefore be used sparingly, or not at all if sensitivity is known to exist. If *surgical emphysema* occurs, as indicated by puffiness at the base of the neck and on the chest wall itself, with tenderness and a characteristic crackling sensation to light touch, a refill of the pneumothorax may be necessary to prevent expansion of the lung.

The pulse rate should be carefully charted during the first twelve hours, as hæmorrhage can occur from one of the divided adhesions or from the trocar wound. Should blood accumulate in the chest it must be aspirated completely, and the loss of blood, if considerable, replaced by transfusion. The patient is also taken back to the theatre for re-insertion of the thoracoscope and coagulation of bleeding vessels.

**Phrenic Crush.** In the event of an unsatisfactory A.P., or to reinforce the effect of a pneumoperitoneum, physicians have in the past often had recourse to phrenic paralysis to raise the level of the diaphragm and thus help to relax the affected lung. The nerve is found in the neck as it runs from without inwards over the surface of the scalenus anterior muscle and is crushed between the jaws of a pair of artery forceps. *The resulting palsy, however, although intended to be temporary, is frequently permanent.* This constitutes a grave handicap when, as is likely, the patient has later to undergo major surgery. Thus if a thoracoplasty or upper lobectomy is done the remaining lower lobe is very liable to collapse, for the patient is unable efficiently to cough up basal secretions. This is always serious, and particularly so in tuberculosis, as an atelectatic lobe is very likely to become infected. Also ineffective coughing may result in spill-over of

rhomboids are divided in the line of the incision, or alternatively their tendinous origin from the vertebral spines may be detached. A few of the fibres of the latissimus dorsi are also divided. The scapula can then be retracted forwards to display the upper thoracic cage and can easily be maintained in this position by a single rib spreader of the Tudor Edwards type placed between it and the 4th or 5th ribs.

The part of the serratus anterior muscle arising from the 2nd rib is detached with cutting diathermy, and the fat and loose areolar tissue of



FIG. 290. The position of the patient for thoracoplasty.

the axilla thus exposed is pushed forward with a finger so that the anterior ends of the upper three ribs can be clearly seen. The periosteum of the ribs is then incised longitudinally with the diathermy and stripped free with a rugine from the costotransverse joints behind to the costal cartilages in front. The costotransverse ligaments of the 2nd and 3rd ribs are cut with a chisel, while the index finger of the left hand holds the soft tissue covering the lung out of danger. The necks of ribs are now divided with a rongeur similarly guarded and the ribs lifted forward so that the soft tissue can be cleared from their under-surfaces. Both 2nd and 3rd costochondral junctions are cut and the ribs removed.

An excellent view should then be obtained of the whole of the 1st rib. After the periosteum along its lateral border has been incised with cutting diathermy the rib is stripped carefully along its upper and lower surfaces (Fig. 291a). The attachment of the scalenus anterior muscle, together with the medial attachment of the periosteum, is also stripped off. The

taxed by a more extensive operation. During the days preceding operation the patient receives instruction in diaphragmatic breathing and any faulty posture is corrected. The nature of the operation, its staging, the importance of post-operative coughing, and the necessity for maintaining correct posture of the head and neck, and carrying out full arm movements, are carefully explained. Either general or local anaesthesia may be employed, but the latter is preferable for blood loss is much less, due to the adrenaline in the mixture and, as the patient is conscious, the cough reflex is unimpaired, and therefore if tuberculous pus from the newly relaxed cavity should spill into the trachea, it is coughed up and the risk of seriously contaminating the opposite lung avoided; and furthermore, the patient is able to cough at the end of the operation while still on the table and also immediately on returning to bed. This helps to prevent spill-over and atelectasis of the lower lobe on the operated side. Collapse of this lobe is the commonest post-operative complication and calls for prompt bronchoscopy.

Meticulous asepsis is essential in thoracoplasty. The patient should be receiving 600 mg. of ascorbic acid daily to aid tissue repair. A streptomycin "screen" is not usually required for the type of case suitable for thoracoplasty. During the operation a blood transfusion is given.

Premedication for an average adult consists of Omnopon 20 mg. ( $\frac{1}{2}$  gr.) with hyoscine (scopolamine) 0.4 mg. ( $\frac{1}{15}$  gr.), given  $1\frac{1}{2}$  hours before operation, and injection of Omnopon, 20 mg. ( $\frac{1}{2}$  gr.),  $\frac{1}{2}$  hour before.

Regional anaesthesia is then carried out. A solution is used of 250 ml. of  $\frac{1}{2}$  per cent. Xylocaine with 1 ml. of 1/1,000 adrenaline added just before use. The skin and muscles along the line of incision are infiltrated and a paravertebral block of the first to sixth intercostal nerves is also done. A vagal block is desirable.

The patient lies on the operating table in the lateral position, held securely at the hips by special rests. The uppermost arm hangs freely forwards and is protected from the chest rest and table edge by pads (Fig. 290). If the premedication has been well judged the patient remains asleep or drowsy throughout the operation, but can still cough when required.

The standard thoracoplasty involves removal of varying lengths of the upper seven ribs, for if fewer ribs are taken away the scapula is unable to sink medially against the mediastinum and insufficient collapse of the lobe is obtained. The incision begins just below the upper border of the trapezius muscle about level with the superior angle of the scapula and runs caudally mid-way between the latter and the vertebral spines. It then curves round the inferior angle of the scapula and runs forward for a short distance in the line of the 6th rib. It is of the greatest importance to begin the incision high to obtain a good exposure of the 1st rib, but it is unnecessary to carry the incision far anteriorly. The trapezius and the

deep fascia of the neck, called Sebileau's bands. These pass down between the first thoracic contribution to the brachial plexus (lying closely related to the neck of the 1st rib) and the subclavian artery, and also in the angle formed by this artery and its internal mammary branch. These bands must therefore be divided before the apex of the lung can relax (Fig. 291b).

As the dissection is carried upwards from the neck of the 2nd rib a groove is entered formed by the first thoracic nerve's contribution to the brachial plexus posteriorly and the endothoracic fascia in front. Great care must be taken not to injure this nerve trunk, which now lies bare. As the first of the Sebileau's bands is cut the subclavian artery immediately beyond it is exposed. There is danger when the forceps are passed beneath the next band of injury to the internal mammary artery (Fig. 291b). A band of periosteum lying over the surface of the superior vena cava at the point where the subclavian vein enters it must be cut. The phrenic nerve entering the thorax at this point becomes visible. In patients with much induration due to long-standing tuberculosis, the nerve is very apt to lift away with the medial aspect of the lung and pleura and be mistaken for fibrous tissue attaching them to the mediastinum. The phrenic nerve may therefore be inadvertently divided. The dissection continues forward and the anterior attachments of the periosteum of the upper ribs are cut as they are encountered. Finally, the dissection is carried down the mediastinum with increasing ease until the azygos vein on the right side or the arch of the aorta on the left is visible. All the mediastinal structures are thus exposed (Fig. 292). The apicolysis is now complete and the upper lobe of the lung is lying relaxed in the thorax covered by its two layers of pleura, the endothoracic fascia, and laterally the intercostal muscles and periosteum (Fig. 292). The artificial cavity above is called Semb's space; after operation it becomes partially filled with fluid, which provides a useful "plombage," helping to prevent re-expansion and re-ascent of the lobe. The muscle layers and skin are closed and the first stage is complete.

A substantial pad of wool is placed in the axilla and held firmly in place by Elastoplast bandages to control paradoxical movement of the soft chest wall. At regular intervals during the first twenty-four hours, this soft area is also supported with manual pressure by the nurses, who encourage the patient to cough and thus expel the mucus or mucopus which would otherwise accumulate in the lung. Next day the patient learns to support the area with his own hands. He also raises his arm fully above his head and begins to move his shoulder freely (Fig. 293). The patient has previously been taught to keep his head and neck slightly overcorrected *towards* the side of the operation, for sagging to the opposite side encourages the scoliosis which thoracoplasty patients tend to develop and which is the source of deformity sometimes seen in neglected patients. A scoliosis or any other marked deformity following thoracoplasty is a sign of neglect.

subclavian artery and the lowest trunk of the brachial plexus lie behind and the subclavian or innominate vein lies in front of the attachment of scalenus anterior. The posterior fibres of the rhomboid ligament attached to the anterior end of the rib and costal cartilage are divided. After the medial aspect of the neck of the rib has been thoroughly freed, the costotransverse ligament is cut and the neck divided with special shears. The rib is then disarticulated from its cartilage and removed.

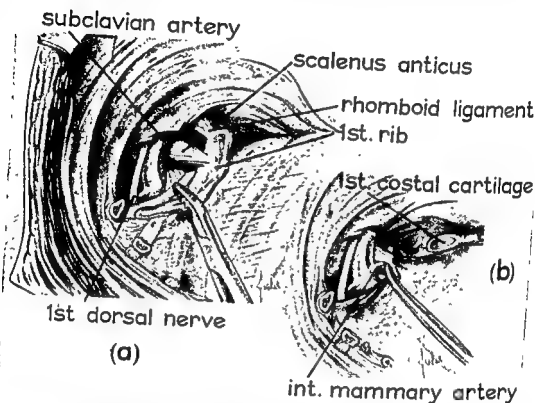


FIG. 201. (a) The periosteum has been stripped from the first rib; the neck cut at the costo-transverse joint, and the rib pulled out to show the attachments of the scalenus anterior muscle and the rhomboid ligament. (b) The first rib having been removed, its deep relations become visible, and Sebilleau's fascial bands are divided (the forceps is under one of them).

The upper three ribs have now been resected subperiosteally from their costotransverse joints behind to their cartilages in front and lateral, but not concentric relaxation has been obtained. Therefore, to complete the operation, *apicolysis* must be carried out. The upper three intercostal bundles (of nerves and vessels) are divided as they emerge from between the necks of the ribs and a plane of dissection developed between the intercostal muscles and the endothoracic fascia which covers the parietal pleura. At the thoracic inlet this fascia is known as Sibson's fascia. It covers the apex of the lung and is suspended by interdigitations of the

during the apicectomy, it is safer (and is in any case more usual) to do the thoracoplasty in three stages.

At the second operation the wound is reopened, the accumulated serum is aspirated from the Serratus space and the apex of the lung remobilized should it have risen in the thorax and become re-adherent. The posterior three-quarters of the 4th and 5th ribs are then resected in the usual



FIG. 293. A patient fully extends his arms the morning after a first-stage thoracoplasty.



FIG. 294. Minimal deformity after a seven-rib thoracoplasty. The scapula now lies against the mediastinum.

way and the chest re-closed. If, however, it is now desired to embed the scapula and complete the thoracoplasty the posterior halves of the 6th and 7th ribs are also removed. Otherwise this is done as a third- and final-stage operation a fortnight later (Fig. 294).

Formerly thoracoplasties involving 8, 9 or 10 ribs were commonly performed. However, no more than 7 ribs should be resected, for if it is thought that removal of more ribs is necessary to obliterate a cavity the wrong operation is being contemplated; the patient would be more suitable for pulmonary resection. When however the disease is limited to the apex of the lung and is minimal in extent, satisfactory collapse may



At the end of a fortnight the back is straight, the arm movements are full, the lower lobe of the lung is well aerated, the paradoxical movement of the chest wall is much diminished, and the wound is healed. The patient is ready for the second-stage thoracoplasty. The extent of this

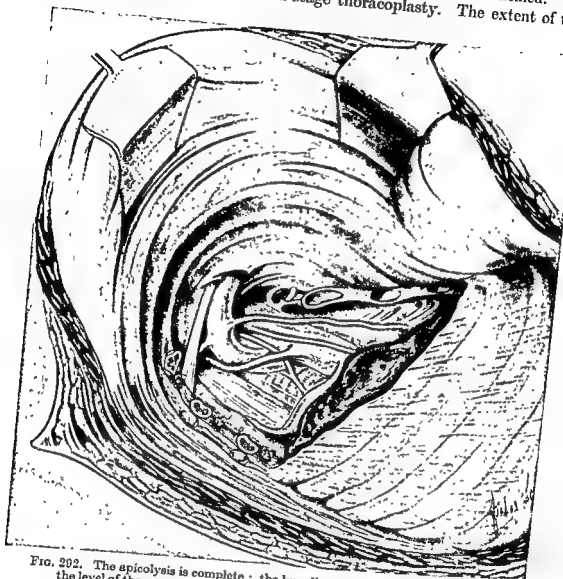


FIG. 202. The spicolysis is complete; the lung lies relaxed in the thorax at about the level of the azygos vein, and the mediastinal structures are exposed. From back to front they are: The first thoracic nerve's contribution to the brachial plexus; the œsophagus; the trachea; the subclavian artery giving off its vertebral and internal mammary branches and being crossed by the vagus; the superior vena cava, crossed by the phrenic nerve and the internal mammary artery running to the back of the sternum.

varies according to the condition of the patient and the degree of mobility of the lung found during the first stage. If the previous operation has caused little upset, the patient is fit, and the disease chronic and fibrotic in type, it may be decided that the thoracoplasty can be completed in two stages. If the patient is old, a little dyspnoeic, or the lung had a very free excursion

but with a much shorter incision. The posterior few inches of the 3rd or 4th ribs are then resected subperiosteally and the deep layer of the periosteum and the endothoracic fascia incised. The parietal pleura is then visible and is gently stripped off the chest wall with the finger to create a space between it and the endothoracic fascia. When this space is big enough a single rib spreader is inserted to provide better access and the dissection is carried over the apex until the whole upper part of the lobe is displaced downwards covered by its two adherent layers of pleura. With the aid of a malleable light and a dissecting swab on forceps, the stripping is completed until the lung is mobilised down to the level of the 5th rib. Bleeding points are coagulated and occasional strands of fibrous tissue may have to be cut with scissors. The operation is unsuitable for anyone with severe or long-standing disease and a hard fibrotic apex, or for patients in whom active tuberculosis is so near the periphery of the lobe that there may be danger of contamination of the space created.

On the day following operation some serum will have accumulated in the space and must be aspirated as it collects and replaced by air. Post-operative bleeding into the space is a serious hazard and is the reason for meticulous haemostasis during operation. If blood does accumulate, however, and cannot be aspirated because of clotting, it may be necessary to re-open and clean out the space. Air refills are continued, with an A.P. apparatus, until a positive pressure of about 20 or 25 cm. of water has been built up, and they are required thereafter once a month or even less frequently. This is the only instance in which air is admitted to the chest under positive pressure. Once the cavity has closed and the sputum has become negative, refills are continued for several years until sound healing is assured. They may then be stopped. The air in the space re-absorbs and much of the collapsed apex re-expands.

There is danger of contamination of such a space either by tuberculosis or by pyogenic organisms, and extra-pleural operations should not be done unless prolonged skilled after-care is available, for constant attention is needed to see that the space does not become obliterated by an effusion or lack of refills.

**Extra-periosteal Plastic Plombage.** To avoid some of the disadvantages of extra-pleural pneumothorax, attempts were made in the past to fill the space with vegetable oil (oleothorax) or paraffin wax (wax plombage). The usual result was either secondary infection or ulceration of the foreign substance into the lung and its subsequent piecemeal expectoration. Non-irritating plastics were later employed instead, but at first they suffered a similar fate to that of the other materials. Attempts were therefore made to increase the safety of the operation by interposing more tissue between the prosthesis and the diseased lung. Instead of the parietal pleura only, the periosteum over the upper five ribs, the intercostal muscles, the endothoracic fascia and the parietal pleura were all

be obtained by a five-rib thoracoplasty, leaving the scapula in its normal position riding outside the 6th and 7th ribs. When thoracoplasty is performed to obliterate dead space in the thorax left after resection of a lobe, or after pneumonectomy, and thus to prevent over-stretching of the residual lung and mediastinal displacement, the number of ribs resected clearly depends on the size of the space to be filled ; but in these circumstances it is best to spare the 1st rib with its muscular insertions as there is less tendency to deformity.

After each stage the usual precautions are taken against surgical shock, although blood transfusion is usually required only during or after the first stage, for the initial wound is re-opened almost without bleeding for the other stages. The dangers of paradoxical chest movement have already been mentioned and must be adequately controlled. In elderly patients, or those who are emphysematous or bronchitic, it may be necessary to stage thoracoplasty at a slower tempo. It is always of great importance to maintain good aeration of the lower lobe on the side of the operation, and if collapse occurs (as shown by a rise in pulse rate and temperature, impairment of percussion, diminished air entry, mediastinal shift and opacity on X-ray), bronchoscopy must be promptly carried out—if necessary with the patient in bed. After aspiration of the secretions blocking the bronchus the lobe re-expands. *Such a complication is much more probable if the phrenic nerve has previously been interfered with.*

Occasionally spread of disease to the opposite lung may appear soon after operation, due to spill-over from the tuberculous cavity or to re-activation of existing contralateral disease. This event is of grave significance and demands immediate administration of antibiotics and the suspension of further surgery. If three or four months after thoracoplasty the patient's sputum is still positive on culture the operation has failed and resection of the underlying lobe is indicated without delay.

**Extra-pleural Pneumothorax.** In carefully selected cases this operation has a very limited but useful application, especially as it is the only major surgical procedure the effects of which are reversible. It has a bad reputation in England, partly because it has in the past been applied to unsuitable patients, and because the after-care requires much skill and application; but this type of collapse therapy is still employed in Swiss and Scandinavian clinics.

A small chronic sub-apical cavity (i.e. not too peripheral), without much surrounding disease, may be treated by means of an extra-pleural pneumothorax when other methods, such as induction of an A.P., are contraindicated. In some patients in whom bilateral disease is present, bilateral "extra-pleurals" are acceptable when resection or thoracoplasty are not, or in whom it is desired to perform a resection upon a badly diseased lobe on one side and less severe disease in the contralateral apex must first be brought under control.

The posterior ends of the upper ribs are exposed as for thoracoplasty

for thoracoplasty, except that the degree of relaxation obtainable is not so complete.

The surgical approach is similar to that for extra-pleural pneumothorax, although usually it is unnecessary to resect any ribs. The periosteum is incised over the upper five ribs and stripped from them (the periosteum of the first rib is stripped from its under surface only) and the related intercostal bundles are secured and divided posteriorly near the costotransverse joints. It is then possible to perform an apicolysis in very much the same manner as in thoracoplasty. When the desired degree of collapse has been obtained, two adjacent ribs are spread a little and polyethylene balls are introduced until they just fill the space between the ribs and the detached muscles and periosteum. The wound is then closed. It will be noted that this procedure avoids two disadvantages of thoracoplasty: (a) The plastic plombage prevents paradoxical excursion of the lung, and (b) as no ribs are removed there is no subsequent deformity.

### PULMONARY EXCISION

There is much to be said for extirpation of diseased tissue as opposed to leaving it behind and promoting healing by relaxant measures, but the problems of excision in pulmonary tuberculosis are far from simple. Tuberculosis at its inception is often widespread in the lung and is at no time strictly confined to one pulmonary segment or lobe. Radiography reveals only the more advanced lesions in the lung. There are always others unperceived. Furthermore, if lung is excised, it is doubly important in patients with phthisis to avoid over-distension of the remaining lung, not only to prevent emphysema, but to avoid re-kindling other quiescent foci. Hence thoracoplasty is often required after an excision has been carried out. Also, resection of lung carries with it certain risks and complications not associated with relaxant operations, such as bronchial fistula and persistent air leaks. On the other hand, excision is applicable to many tuberculous conditions for which thoracoplasty is quite useless. It has immensely enlarged the scope of surgery in the treatment of tuberculosis, now accounting in one form or other for about 80 per cent. of major operations performed for this disease.

It is desirable to leave the maximum amount of healthy lung tissue compatible with effective extirpation of the disease. Sometimes, such as with totally "destroyed" lungs, pneumonectomy is necessary; often a whole lobe may have to be resected when it is much cavitated or bronchiectatic, but in most cases the removal of one or more pulmonary segments is sufficient to eradicate a cavity or a solid agglomeration of tuberculous tissue. All resections, whatever their scope, must be done under antibiotic cover, usually of streptomycin and P.A.S., but if the subject is resistant to these drugs Viomycin, Marsalid, or Isoniazid may be employed. The drugs should be administered for at least six weeks

stripped down in one thick layer on to the lung, and the plombage inserted between them deeply and the bare ribs superficially (Fig. 295). Various prostheses are used : (a) *Polystan sponge*, which has the disadvantage that should infection occur the sponge is difficult to remove ; (b) *polythene bags* which are stuffed with broad polythene ribbon to produce the degree of tension required ; and (c) *polyethylene balls*, about the size of golf balls,

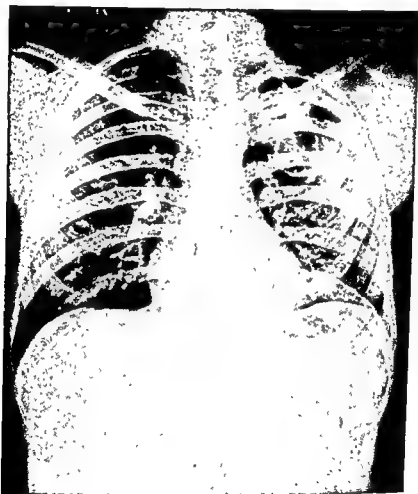


FIG. 295. A left extrapariosteal plombage with one variety of polyethylene balls.

have the advantage that they are easily inserted and can be readily added or subtracted to fill the irregular space completely and to produce just the right degree of plombage. The balls are pierced with cylindrical holes so that any effusion occurring does not produce additional displacement. This otherwise excellent modification of extra-pleural pneumothorax differs from the original, for the periosteum is displaced downwards, and therefore bone reforms in the new position causing the collapse to be permanent. It is in fact a substitute for thoracoplasty rather than for extra-pleural pneumothorax, and the indications for it are similar to those

healed foci at the apex. Pneumonectomy, followed by thoracoplasty, is indicated, for surgery offers the only hope of survival. The patients are often in poor physical condition and may be already rather short of breath. The diversion of unoxygenated blood, however, from a diseased and non-functioning lung to the opposite normal lung in which it can take up oxygen, may render patients much less breathless after operation than they were before. Furthermore, such patients if left inadequately treated are likely to develop amyloid disease. Any albuminuria should be an extra spur to surgery rather than the reverse. (7) The presence of a *bronchopleural fistula*, or a *tuberculous empyema* associated with a positive sputum, when the disease is confined to one lobe or lung. When, however, a tuberculous empyema exists with a negative sputum and the disease in the underlying lung is healed, decortication is indicated instead of resection. Re-expansion of the lung and restoration of function and mobility follow. If, on the other hand, the patient is coughing up the contents of an empyema because of the presence of a fistula, or if an empyema is associated with active disease in the lung beneath, lobectomy or pneumonectomy with total pleurectomy and removal of both empyema and the diseased lung is indicated.

The complications following resection for pulmonary tuberculosis are substantially the same as those after resection for other conditions, with the following additional ones: (a) Spread of disease to other parts of the lung is an extra hazard. Therefore antibiotic cover must be sustained and effective, and rest must be complete and prolonged (patients remaining in bed for at least three to four months, and often longer). (b) Post-operative bronchopleural fistula, once the scourge of thoracic surgery, is now rare, but remains rather more common after resections for tuberculosis than for other lesions. If it occurs from the bronchial stump, the best treatment is prompt thoracotomy and careful resuture of the leaking bronchus. The latter should be covered by an intercostal muscle graft if this has not previously been done, and an apical thoracoplasty is performed to bring the chest wall in close apposition with the point of amputation of the bronchus. If there is only a small but persistent air leakage from a raw area of lung, exploration and simple suture of the leaking point may be sufficient. As many segmental resections leave considerable areas of raw lung, and as these areas may have become unusually emphysematous or affected by fibrosis from old disease, persistent leaks of this type constitute the most troublesome complication. Therefore at the time of operation the raw surfaces are carefully tested for leaks by saline irrigation while the anaesthetist maintains a low positive pressure. Bubbles are seen coming from leaks and fine mattress sutures are inserted round them. The best safeguard against leakage, however, is complete expansion of the surrounding lung and its adherence to the chest wall. This process (provided obvious leaks are dealt with at the time of operation) is encouraged by the insertion of two water-sealed drainage tubes, a

before operation (streptomycin is given in doses of 1 gm. daily), but usually the patient has been receiving them much longer. The surgical procedures are in all cases exactly the same as those described under Pulmonary Resections (Chap. 42). Following operation, the patient continues with the course of antibiotic drugs and remains in bed for a minimum period of three months. Subsequent lateral thoracoplasty is often necessary, if any substantial dead space remains in the thorax, to protect the residual lung from emphysema.

The indications for excision in pulmonary tuberculosis are as follows :

(1) *A Failed Thoracoplasty*.—If culture of the sputum remains positive three to four months after thoracoplasty the operation has failed and excision of the affected lobe is imperative. Lobectomy after thoracoplasty presents no great difficulty although entry into the chest is not so easy as when a normal rib is resected. The post-operative disturbance is generally less.

(2) *The Presence of a Chronic Tuberculous Cavity in a Position Unsuitable for Thoracoplasty*.—(a) Cavities in the apex of the lower lobe or in one of the basal segments ; (b) a cavity in an upper lobe close to the mediastinum ; or (c) a very large cavity perhaps occupying most of the lobe. Appropriate segments or lobes are excised.

(3) *Tuberculous Bronchiectasis*.—In patients with long-standing disease the bronchi in the affected lobe or segment may have developed bronchiectasis, which is the source of positive sputum although no actual tuberculous cavitation is present. It is useless merely to compress such an area. Therefore as soon as bronchiectasis has been revealed by bronchography, resection of the affected part of the lung is indicated.

(4) *Solid Lesions* or "*Tuberculomas*."—Only removal can possibly provide a satisfactory solution. On the other hand, solid foci, sometimes quite inactive, are very common in the lungs of patients who have had tuberculosis and it would be quite unjustifiable to remove them all. The circumstances under which they should unhesitatingly be excised—assuming them to be the predominant lesion present—are : (a) If they are associated with positive sputum or hæmoptysis ; (b) if they show radiological signs of increase in size or alteration of edge, or if tomography reveals them to be cavitated ; (c) if they are much bigger in diameter than 3 cm., because experience proves the larger lesions to be almost invariably troublesome ; (d) if, being unassociated with any other evidence of tuberculosis, doubt exists as to their identity, particularly if the patient is a man and over forty, as the risk of the lesion being a carcinoma is then very considerable. Calcification in a lesion provides good grounds for leaving it alone, but there are exceptions to this rule.

(5) *Persistent collapse of a lobe* or the presence of *tuberculous bronchostenosis* due to endobronchial disease. These conditions are identified radiologically and by bronchoscopy.

(6) *A So-called "Destroyed" Lung*.—It is not uncommon to see patients in whom one lung is wholly cavitated, or opaque and functionless, and in whom the opposite lung is free from disease or shows perhaps a few

with lobectomy or pneumonectomy according to the location and extent of the active tuberculous lesion in the lung. The extrapleural plane of dissection is developed round the chest. Sometimes this presents great difficulty, but as the pulmonary hilum is approached it is generally free of the intense fibrosis encountered elsewhere, and isolation and ligature of the vessels can be carried out without undue impediment.

Those patients in whom the disease in the underlying lung is healed, and whose sputum is therefore consistently negative, are suitable for removal of the empyema by decortication and re-expansion of the lung. A standard posterolateral thoracotomy is performed, usually with removal of the rib most centrally situated over the empyema, although it is better if rib resection can be avoided altogether. The abscess cavity is at once opened widely, all the pus and fibrinous slough within it are sucked out, and its limits are defined. The parietal extrapleural plane is then found and the whole tough carapace stripped manually or, if necessary, by sharp dissection from the chest wall up to and including the angles of return on to the lung or the mediastinum. Bleeding during this phase may be severe and adequate provision must be made for blood replacement. Hot towels are packed into the freed areas. If the diaphragm is involved, it too should be cleared, but the process is often more difficult over it than elsewhere, and it is important that its musculature should not be damaged. On the mediastinal aspect the greatest care must be taken not to injure the phrenic nerve directly with instruments, by stretching, or by the use of diathermy coagulation nearby. When the parietes are clear of thickened pleura, the layer overlying the lung is deliberately incised until the visceral plane of cleavage is reached. This plane is developed by sharp dissection, which unavoidably results in some superficial alveolar air leaks, and dissection is continued until the whole lung is liberated and all the fibrous cortex removed except where it is densely adherent over areas of old disease, when it is better left undisturbed as an isolated plaque. It should, however, be cleared from the fissures, and from the folds in which the lung parenchyma is found imprisoned. The degree to which the anaesthetist is able to re-expand the lung at the end of the operation depends upon the amount of old disease and fibrosis within it, but if this is minimal practically complete restoration of normal expansion should be obtained. Dependent and apical water-sealed drainage tubes are inserted before the chest is closed, and both are connected with continuous suction which is maintained until X-rays show the lung to be everywhere in apposition with the chest wall. If residual air spaces become isolated, it may be necessary to empty them by separately inserted intercostal drainage tubes. If fluid or air is allowed to accumulate between the lung and the chest wall, thickening recurs and the result is disappointing. Every effort must now be made to obtain and to preserve mobility of the chest wall and of the diaphragm. Exercises for both are done hourly by the patient alone and also twice a day under the instruc-



dependent one to drain off any effusion, and an apical one for the removal of air. Continuous suction is applied to both. These tubes are usually removed after forty-eight hours when the lung is seen radiographically to be fully expanded. As most segmental resections are performed on one or other upper lobes, and it is therefore at the apex that leaking air usually collects, it is helpful to nurse such patients lying on their operated sides in a slight head-down position for the first three or four postoperative days. Patients should have become accustomed to this position prior to operation. By these means the lung is displaced upwards, adheres to the parietes, obliterates the space, and seals off potential air leaks early.

**Tuberculous Empyema.** *Some 40 per cent. of tuberculous empyemas follow induction of artificial pneumothorax, usually one in which adhesions persist and in which attempts to divide them have failed.* As empyema is a disaster, it is evident that the induction of artificial pneumothorax must be well judged and skilfully managed, and never regarded as a form of minor therapy to be embarked upon light-heartedly. At the first sign of fluid accumulation in the pleural cavity, or if there is other evidence (such as contra-selective collapse) that the treatment is ineffective or unsatisfactory, refills are abandoned.

If pus forms in the pleural space of a patient with pulmonary tuberculosis, it must be aspirated at once in the hope of securing re-expansion of the lung and its early adherence to the chest wall with consequent obliteration of the space. In early cases persistent efforts along these lines have a good chance of success, but very often the rapid increase in pleural thickening over the lung and the chest wall render both quite immobile and permanently prevent further re-expansion.

Under these circumstances—and they are the rule rather than the exception—a drainage tube should never be inserted as is done in non-tuberculous empyema, for not only will it be required permanently, but it will probably also lead to secondary infection of the space by pyogenic organisms. Likewise the performance of thoracoplasty over a tuberculous empyema in an effort to obliterate it by bringing down the chest wall on to the lung, instead of expanding the latter towards the former, is usually futile, and adds still more to the impairment of function. Fibrosis and thickening of both layers of the pleura are so rapid and so extreme in tuberculosis that only their surgical removal by decortication provides any hope of restoring normal function.

Decortication is a major and sometimes formidable operation so that the patient must be in a sufficiently good general condition to withstand it. In addition, there must be no active disease in the opposite lung. If active disease is still present in the lung beneath the empyema, as evidenced, for example, by a positive sputum, re-expansion of such a lung, or lobe, is clearly contraindicated, and the procedure should then be a total pleurectomy with resection of the underlying diseased area: i.e. removal of the empyema by stripping it from the chest wall, coupled

## CHAPTER 42

### PULMONARY RESECTIONS

**Thoracotomy.** Two positions of the patient are in common use, lateral and prone. The choice depends on whether or not an intrabronchial blocker is employed by the anaesthetist. A blocker is contraindicated (a) in young children, for their bronchi are too small, (b) in patients who have bilateral suppuration, (c) in the presence of a bronchopleural fistula, and (d) when there is very serious risk of spill-over of secretions from the diseased into the healthy lung. In all these instances in which use of a

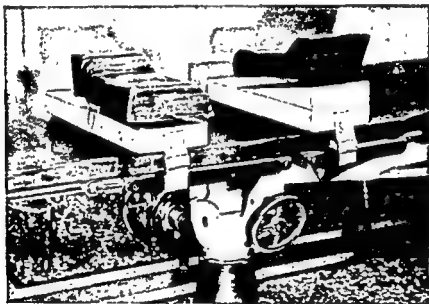


FIG. 296. The chest and pelvic rests employed in the "face-down" thoracotomy position.

blocker is unsuitable the prone position is used. The patient lies upon pelvic and upper chest rests arranged to secure a gentle gravity drainage up the bronchial tree so that secretions are easily aspirated from the trachea (Fig. 296). The thoracotomy side lies slightly overlapping the edge of the table towards the surgeon. The patient's arm hangs vertically downwards on this side (Fig. 297). The advantages of this position are : (1) Safety from spill-over, (2) the avoidance of intrabronchial blockers, (3) the surgeon can operate seated. The disadvantages are : (1) Loss of some freedom of access to the chest, including the hilar structures, (2) cardiac movement is more prominent, for the lung lies upon the pericardium, (3) infants with soft chest walls sometimes do not tolerate the

tion of a physiotherapist. Spreading of the narrowed intercostal spaces can be obtained by the patient being made to lie for a considerable part of the day with the healthy side of the thorax arched over pillows. The after-care of patients who have had a decortication is at least as important as the operation itself, and all the advantages gained by the latter will be forfeit if rapid lung expansion, conscientious exercises, and postural correction are neglected.

If necessary, part of the inferior edge of *rhomboideus major* is also divided. All bleeding points are coagulated with diathermy. Many of the vessels can be secured before they are divided.

For standard pneumonectomy or lobectomy the best access is gained by resection of the whole length, from neck to costal cartilage, of the 6th rib. The periosteum over the rib is first incised with cutting diathermy and then stripped from it with a curved elevator. Little if any bleeding occurs and Doyen's retractors are not required. The rib is divided at its neck and costochondral joint by means of a guillotine costotome. The periosteal bed is then carefully opened and the pleural space entered.



FIG. 298. The lateral position. The chest rests upon a padded right-angled support, and the pelvis is fixed by special attachments in front and behind. The bandages round the hips and head provide additional stability if it is desired to tilt the table laterally towards the surgeon.

If it is desired later to cover the bronchial stump with an intercostal muscle graft, this is next cut, incorporating the intercostal artery, vein and nerve of the resected rib, but leaving enough muscle on the upper edge of the 7th rib to make subsequent closure of the thoracotomy easy.

Wound cloths are inserted, and the pleural cavity widely exposed with the help of a Finochietto rib-spreader. At this point care must be taken to divide any adhesions between the lung and chest wall as they are brought into view, and to increase the rib spread slowly and gently so that ribs are not broken in the process. The freed lung is now examined, and if a growth is present its operability and the scope of the operation are determined.

**Pneumonectomy (Left).** The pleural reflection round the hilum is incised and small blood vessels are coagulated. The pulmonary ligament is divided from the diaphragm to the inferior pulmonary vein, and the latter is cleaned and isolated (Fig. 299). A pneumonectomy, or

position well before the thorax is opened and just after it is closed (prompt entry and exit are therefore indicated).

In adults, except when contraindications exist (*vide supra*), the lateral position is generally used. The patient is placed with the back close to, and parallel with, the surgeon's side of the table. The dependent side of the chest lies on an L-shaped, sponge-rubber covered rest which gently arches the chest and spreads the uppermost ribs (Fig. 298). The upper arm, which is used in the administration of the intravenous drip, hangs loosely over the far side of the table cephalad to the vertical limb of the above-mentioned rest. Two other vertical supports, one against the thigh

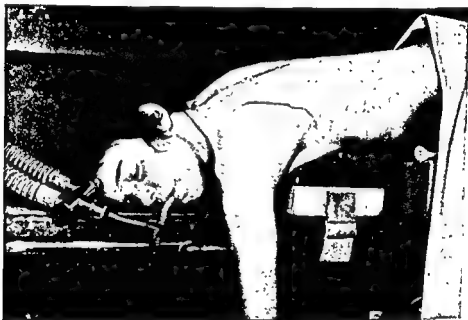


FIG. 297. The "face-down" position. The inclination of the thorax is sufficient to ensure gravitational drainage. The vertebral border of the scapula has been marked as it falls naturally forward to expose the ribs.

and one in the knee flexure, fix the body in the lateral position and allow the whole table to be tilted towards the surgeon if required (Fig. 298).

The line of incision passes obliquely round the angle of the scapula from an inch lateral to the vertebral spines posteriorly to the costal cartilages anteriorly, and follows roughly the line of the 6th rib. The line of the incision, and the two or three nerve roots above and below this line, are infiltrated with local anæsthetic. This procedure reduces bleeding, shock and the need for much general anæsthesia. Following the skin incision, the latissimus dorsi is divided, exposing the angle of the scapula and the triangle of fatty areolar tissue formed by the anterior edge of the trapezius behind and the posterior border of serratus anterior in front. This triangle is opened and both muscles cut to lay bare the ribs. The scapula may now be elevated and the ribs counted from above down.

tends to facilitate access to another (Fig. 301). [When patients are face down (i.e. a blocker is not used) it is good practice to secure, divide and suture the bronchus before ligating the vessels, but when this is done traction on the lung must be avoided, as it is then attached to the mediastinum only by the vessels.] The pulmonary artery is cleared and the branches of the vagus nerve lying on its surface cut (Fig. 302). Among these branches is a small vessel requiring ligation. During this procedure, and indeed at all times, care must be taken not to damage the left recurrent laryngeal nerve as it parts company from the main vagus trunk and

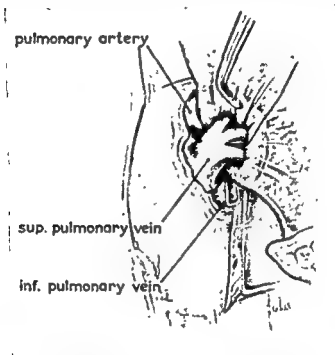


FIG. 300. Exposure and ligation of superior pulmonary vein. Note the stump of the inferior pulmonary vein previously ligatured.

loops round the ligamentum arteriosum on its way back to the neck (Fig. 302). When the artery is thoroughly defined, it is freed digitally gently from the underlying bronchus. Forceps are passed round it and it is doubly ligated close to the ligamentum so that an adequate cuff is left. As the 25-gauge thread is tightened it should be felt to "bite" into the artery wall.

Only the bronchus now remains. Closely attached to its anterior and posterior walls course tortuous and rather friable bronchial arteries. If these have not already been ligated during the hilar dissection they are dealt with now, and all the related lymphatic glands are separated from the mediastinum and carried up with their areolar tissue on to the lung.

O'Shaughnessy forceps, is passed round the vein to secure a 25-gauge linen thread delivered in "bow-string" fashion, and the vessel doubly ligated as close to the heart as possible. A third ligature is tied distally near the lung and the vein cut to leave a sound cuff at its cardiac end. The lung, grasped by Duval forceps (see in Fig. 299), is now drawn towards the surgeon and the superior pulmonary vein exposed and thoroughly cleaned of areolar tissue and pericardial fibrous reflexions.

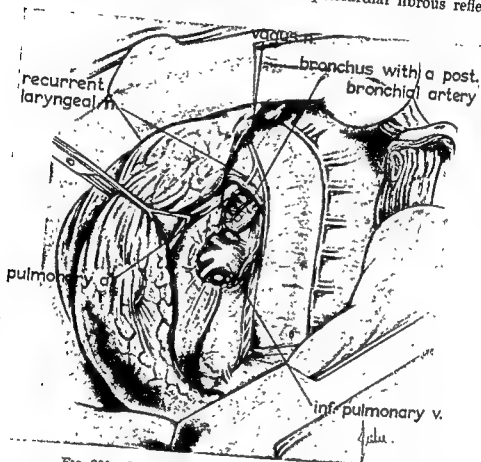
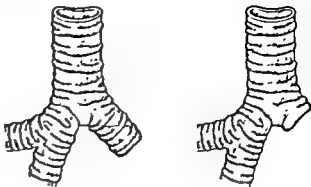


FIG. 299. Exposure of the inferior pulmonary vein.

A pneumonectomy forceps is passed between the superior pulmonary vein and the bronchus (to which it is intimately related posteriorly), and the vein is ligated and divided (Fig. 300). It is better to deal with the veins first as division of the inferior pulmonary vein mobilises the lung and makes subsequent procedures safer, and the superior pulmonary vein obscures the pulmonary artery to some extent, though much more so on the right side than the left. Furthermore, if a cancer is present, the real risk of malignant emboli being produced during manipulation is avoided by early ligation of the veins. On the other hand, whatever makes an operation easier is usually best. Therefore, the structures of the hilum may be dealt with in the most convenient order. Division of one structure

FIG. 303. It is essential to divide the bronchus flush with the trachea so that no blind pouch is left in which secretions can collect and infection occur.



Those at the carina will have to be freed from the opposite bronchus, and a bronchial artery ligated just to the left of the angle of the carina. The bronchus is now clean and a *curved untoothed bronchus clamp* is lightly applied obliquely across its origin from the trachea (as the intrabronchial blocker, when present, is withdrawn), in such a fashion that no residual stump is left (Fig. 303). The *spiked crushing clamp* is placed distally (Fig. 304); the bronchus is cut across and the lung removed. Complete hæmostasis is established, the stump closed by a number of interrupted gauge 00 linen sutures and the clamp taken off. While the anæsthetist maintains a positive pressure the stump is bathed in saline so that any air leak may be detected and remedied; a sucker is always available to

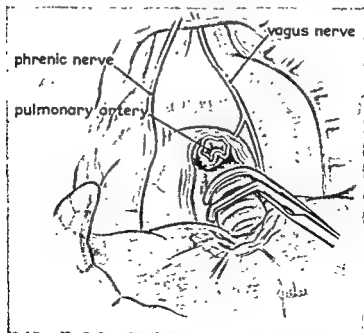


FIG. 304. The artery has been divided and the bronchus clamps have been applied.



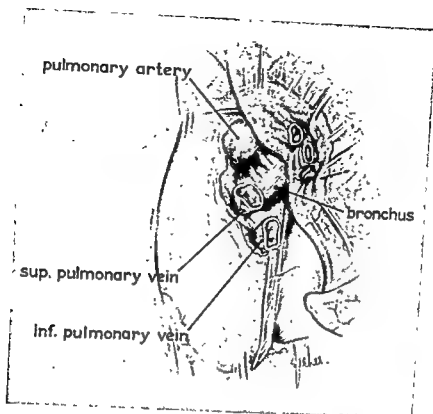


FIG. 301. After division of the veins, the anterior aspect of the bronchus is clearly defined.

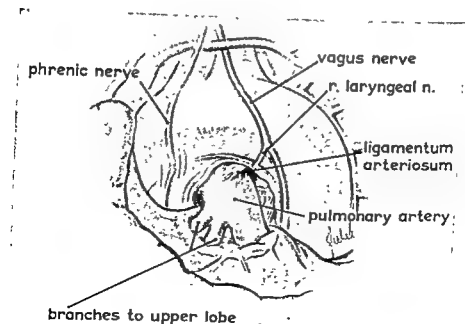


FIG. 302. The pulmonary artery is exposed.

contributes to safety, to ligate and divide the vein first. It will now be seen that a triangular fibrous reflexion from the pericardium overlies the base of the artery and is attached to a lymphatic gland lying at the bifurcation of the artery into upper and lower divisions (Fig. 305.) This fold must be lifted from the arterial wall by forceps passed beneath it, and cut, thus exposing the main trunk of the artery, from which the rest

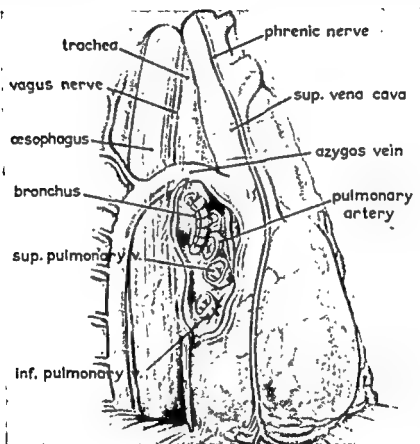


FIG. 306. General view of the right mediastinum after removal of the lung, but prior to sewing on a muscle graft.

of the tissue is peeled by swab dissection. As on the left side, the artery is gently freed by the index finger from its bed, ligatures are passed round it and tied as proximally as possible. Each of the two divisions is separately secured distally.

As the right bronchus is shorter than the left and does not lie so much beneath the aortic arch, it is easier to clamp and divide it at its most proximal point. A large constant bronchial artery lies on its posterior aspect and is secured early in the operation when the pleural reflexion and vagal branches are divided. A view of all divided structures is shown in Fig. 306.

remove excess saline and prevent possible aspiration. When absolute closure is assured, the muscle graft, which during the operation has been wrapped in a saline swab and allowed to hang from the posterior end of the wound, is delivered and sewn securely over the stump. If a graft is not employed, a flap of pleura should be mobilised and used to cover the stump instead. Penicillin is dusted over the stump.

Three holes are now drilled at intervals along the 7th rib, and pericostal sutures of doubled No. 4 chromic catgut are passed through them and round the 5th rib above. A rib approximator draws the two ribs together and is removed after the sutures have been tied. The intervening intercostal muscles are sewn up with a continuous No. 2 chromic cat-

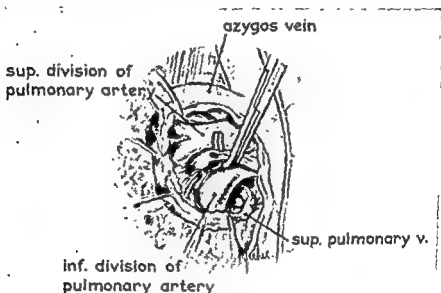


FIG. 303. Exposure of the right pulmonary artery. The superior pulmonary vein has been divided.

gut stitch, and the other muscle layers are similarly closed. Interrupted nylon is employed for the skin and subcutaneous layers. Drainage is not required in pneumonectomy, but when the dressing is in place, the rests removed, and the patient on his back, the intrapleural pressures of the operated side are measured with a Morland needle and Maxwell box, and enough air is added or withdrawn to make the pressures atmospheric (e.g. a swing of plus 10 to minus 10 cm. on the manometer scale).

**Pneumonectomy (Right).** This differs from the operation on the left in minor anatomical points only. The pulmonary ligament and inferior pulmonary vein are dealt with as before. The pulmonary artery instead of entering the hilum as one large trunk bifurcates into a smaller upper and much larger lower division just under cover of the superior vena cava, and of these divisions the inferior lies almost wholly behind the superior pulmonary vein (Fig. 305). It greatly simplifies dissection, and therefore

pressure in the bronchus, having first removed his intrabronchial blocker. Any leak is thereby detected and can be dealt with. The stump is covered by an intercostal muscle graft sewn in place over it and passing underneath the main pulmonary artery. Two water-sealed drains are left in the chest, one draining the effusion from the base of the chest, and a narrower one allowing air to escape from the apex.

**Left Lower Lobectomy.** The pulmonary ligament is cut and the inferior pulmonary vein defined and cleared. Its position is marked by a small lymphatic gland which is found constantly at its inferior border (Fig. 299). After the vein has been ligated and cut, the interlobar fissure

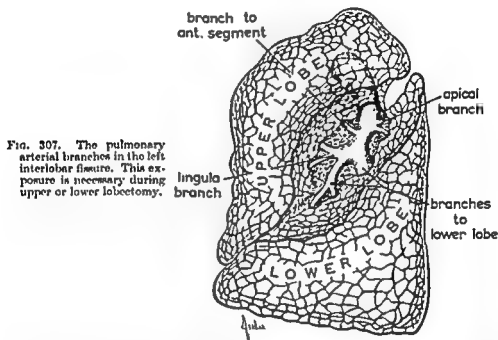


FIG. 307. The pulmonary arterial branches in the left interlobar fissure. This exposure is necessary during upper or lower lobectomy.

is opened and dissected down until the pulmonary artery is found lying in its depths (Fig. 307). Its branches to the lower lobe, usually comprising two main trunks and a smaller vessel to the apical segment (it may come off as the pulmonary artery curves round the bronchus before it actually enters the fissure) are tied and divided. A bronchial artery is always present closely attached to the anterior surface of the bronchus which is now exposed. This vessel is dissected free and tied, or under-run by a suture. The rest of the bronchus is cleared, divided, and closed. It is sufficient to leave one basal water-sealed drain in position before closing the chest.

**Right Upper Lobectomy.** The superior pulmonary vein is dissected clear and the part supplying the upper lobe ligated. Great care is taken not to damage or occlude its branch to the middle lobe. The pulmonary

**Intrapericardial Pneumonectomy.** If a growth extends so far proximally that it involves the pericardium, or makes extrapericardial division difficult and hazardous, the pericardium is widely opened behind the line of the phrenic nerve, and the pulmonary veins are secured and tied on the atrial wall itself. On the left, the ligamentum arteriosum should be cut (after displacing the recurrent laryngeal nerve laterally) in order to obtain a safer cuff and to ligate the pulmonary artery close to its origin. The pericardium behind the hilum is similarly opened and the whole involved area removed with the lung. This procedure greatly simplifies the technique of the operation, but cardiac irregularities are much commoner. Furthermore, if the defect in the pericardium on the left side is large and cannot be closed, there is a risk of the apex of the heart prolapsing through it. Some surgeons routinely employ intrapericardial ligation in all malignant cases, combined with a form of block dissection of the mediastinal tissues, but owing to the anatomy of the lymphatic drainage of the lung it is doubtful if this confers much benefit, and comparative survival figures do not support the practice.

### LOBECTOMY

(Figs. 307 and 308)

**Left Upper Lobectomy.** The superior pulmonary vein is first ligated (Fig. 300). The pleural reflection over the pulmonary artery is cut and the surface of the vessel thoroughly cleared. In long-standing inflammation, such as tuberculosis or bronchiectasis, the artery is found in a sheath of tissue which must deliberately be incised and opened before the vessel wall is reached. This step is essential, and failure to perform it is the cause of most subsequent difficulties. Further dissection is relatively easy and the branches of the artery to the upper lobe can readily be identified, ligated and divided (Fig. 307). A sufficient length of each branch must be freed to provide an adequate cuff, and as the vessels are themselves thin-walled and often friable care must be taken to avoid evulsion from the main artery by excessive traction on the lung. For this reason also it is safer to tie their distal extremities before placing the proximal ligature. As these branches are tied in succession, the main artery is followed round the hilum of the lung into the interlobar fissure where the branches to the anterior and lingular segments are usually found (Fig. 307).

The upper lobe bronchus is cleared and the small bronchial arteries on its anterior and posterior surfaces are ligated or undersewn. When the upper lobe bronchus has been thoroughly defined down to its origin from the stem bronchus, clamps are applied as proximally as possible and it is cut between them. Its proximal end is closed by interrupted sutures (No. 90 linen thread) flush with the main bronchus. Saline is run into the chest to cover the stump while the anaesthetist maintains a positive

pulmonary vein. The fissure is then opened at the apex of the middle lobe, and the main arterial trunk to the lower lobe identified and dissected clear (Fig. 308). As this vessel is followed upwards its middle lobe branch at once comes into view. In about half the cases there are two branches, one just above the other. The bronchus is finally divided and the lobe removed. One basal drain is employed.

**Right Lower Lobectomy.** The pulmonary ligament is divided and the inferior pulmonary vein ligated and cut. The fissure is opened and the branches of the artery to the lower lobe are identified and secured. Care is taken not to injure the middle lobe branch. The artery to the apical segment often arises well above this level on the opposite side of the main trunk and may have to be separately ligated. One basal drain is left in place.

**Segmental Resections.** Whenever possible healthy lung is preserved. In the treatment of pulmonary tuberculosis it is often possible to remove an isolated cavity or solid focus by segmental resection alone. The procedure is similar for all the segments. First the branches of the pulmonary artery supplying the lobe of the diseased segment are carefully defined. The one running to the segment itself is identified and secured. The lobar bronchus is then dissected out and followed distally until it gives off the segmental branch. This is lightly clamped and the rest of the lobe inflated by the anæsthetist. The segment can then be stripped off its neighbours by a combination of traction and blunt dissection. The tributaries from the segment to the pulmonary vein are tied as they are encountered (Fig. 309). On the left side the apical and posterior segments of the upper lobe share a common bronchus and artery for a short distance and may therefore have to be removed together. If, however, dissection can be carried sufficiently far distally it is often possible to separate them.

The only disadvantage of segmental resection is that when a good deal of chronic disease exists near the fissures, or there is emphysema, the raw surfaces left may produce troublesome air leaks. Every care is taken therefore at the time of operation to close such leaks by under-sewing them. As most segmental resections are performed on the upper lobes the apex of the pleural cavity is usually drained. Sometimes it is an advantage to nurse such patients in a head-down position for the first three or four days in order to displace any air and effusion at the apex, and promote adherence of the lung to the chest wall. A great advantage of segmental resections is that they avoid the necessity of subsequent apical thoracoplasty in tuberculous patients. If, for example, a left upper lobectomy be done in such cases, thoracoplasty is usually indicated to prevent overstretching of the lower lobe, but if the removal of one or even two segments proves sufficient, then enough of the upper lobe is left to avoid this need.

**Preoperative Care.** Before any pulmonary resection the patient is

artery is defined as in a pneumonectomy, and the smaller of its two main trunks, supplying most of the upper lobe, is ligated and cut (Fig. 305). A lymph gland is constantly found in the bifurcation and this is dissected free of the remaining arterial trunk, which runs down to enter the fissure and supply the other lobes (Fig. 305). The arterial branch running to the posterior segment of the upper lobe is sometimes found coming off the artery in this position and can be secured, but usually it leaves the main trunk in the fissure (Fig. 308) (occasionally as a branch of the artery to the apical segment of the lower lobe). It courses to its segment parallel

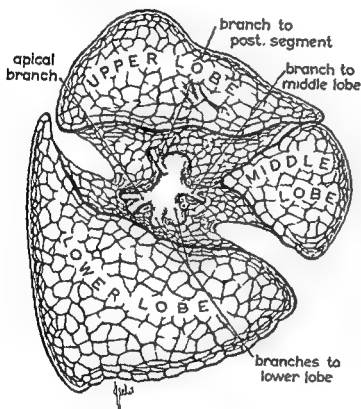


FIG. 308. The pulmonary arterial branches in the right interlobar fissure. This exposure is necessary during upper, middle or lower lobectomy.

with and just inferior to the upper lobe bronchus. This artery is thus usually secured by opening the interlobar fissure, but if this presents difficulties it is better to define and divide the upper lobe bronchus, and approach the artery to the posterior segment from above. The bronchial blocker is then removed and the upper lobe peeled from the inflated middle lobe. The fissure between the two lobes is usually incomplete. Care is taken not to damage the middle lobe artery (Fig. 308) which is often closely related to the ligated stump of the superior pulmonary vein. Upper and lower water-sealed drains are left in place at the end of the operation.

**Middle Lobectomy.** First define and ligate the middle lobe vein which joins the superior pulmonary vein. Occasionally it enters the pericardium as an independent vessel, or less often flows into the inferior

sensitivity is determined, and all operations are performed under suitable antibiotic cover, which is begun three or four days preoperatively and continued for a week or ten days afterwards. If there is lung sepsis, such treatment begins sooner and is continued for as long as may be necessary postoperatively. Anaemia must always be corrected, especially as it may contribute greatly to dyspnoea. Resection of lung should not be undertaken unless the haemoglobin level is at least 80 per cent. Blood lost at operation is replaced as it occurs.

**Postoperative Care.** The principal postoperative aim in thoracic surgery is the restoration as rapidly as possible of normal respiratory function. Essentially, this depends upon two factors, the maximum mobility of the chest wall and diaphragm, and the complete expansion of the remaining part of the lung. To secure expansion of the lung, the bronchial tree must be kept clear of secretions, and the pleural space free of air and fluid (unless, of course, a *pneumonectomy* has been performed). During operation the anaesthetist continually aspirates the bronchi, and at the end of operation it is usual to bronchoscope the patient and meticulously clear all mucus, blood or pus from the bronchial tree before returning the patient to the ward. If there is evidence of bronchial obstruction at any time during the immediate postoperative or convalescent periods, or even of accumulating secretions which the patient is unable to expel by his own efforts of coughing or with the help of postural drainage, further prompt bronchoscopy and aspiration are indicated. Following lobectomy or segmental resection, adequate water-sealed drainage tubes are left in the chest to evacuate both air and fluid from the pleural space as quickly as possible in order to secure early re-expansion of the remaining healthy lung (Fig. 310). This is aided by the addition of a suction pump to the drainage system. Adherence of the expanded lung to the chest wall is the most effective means of sealing off minor pulmonary air leaks. Usually the drainage tubes can be removed in twenty-four or forty-eight hours if portable radiography confirms full aeration of the lungs. Drainage is also promoted by coughing which is therefore encouraged at regular intervals. The negative pressure in the pleural cavity is indicated by the fluid meniscus which rises and falls in the drainage tube with respiration, and if this free excursion ceases, the tube has been blocked and must be cleared. Sometimes the obstruction is due to blood clot which can be milked down the tube or expressed from the inner end, or it is due to kinking of the tube at the patient's back rest, or to a misplaced safety pin on the bedside. But if the block is permanent, or the swing ceases because the lung has fully expanded and sealed off the end, the tube should be taken out and any residual air or fluid removed by aspiration. It is essential that air should not be allowed to enter the chest when the drainage tube is withdrawn. Therefore, after the holding suture has been cut, the skin on both sides of the tube is pinched between finger and thumb and the patient exhales and holds his



taught those breathing exercises needed afterwards, for they are easier to learn and to perform before they are impeded by pain. Physiotherapy is not only designed to improve the patient's vital capacity by increasing rib and diaphragmatic excursions, but also to teach dissociated move-

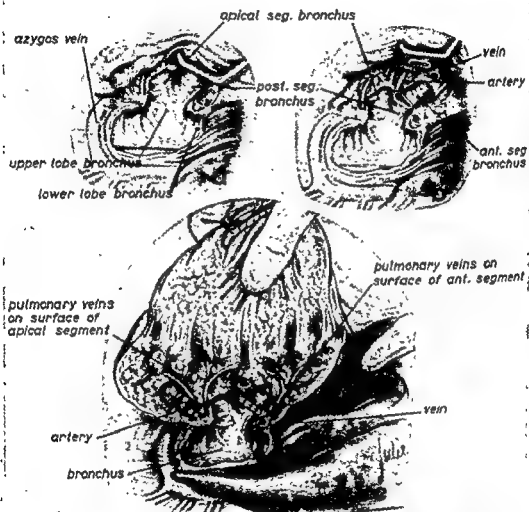


FIG. 309. (a) For the removal of the posterior segment of the right upper lobe, the upper lobe bronchus is first defined and its posterior segmental branch identified. (b) In this instance the artery to the segment lies behind the bronchus and is not readily accessible. Therefore the bronchus is secured and divided first. Both vein and artery are then tied and cut as they are encountered. (c) While the anaesthetist keeps the neighbouring segments of the lobe inflated, the segment to be removed is gently peeled away. The intersegmental veins help to define the boundaries of the segment.

ments of either half of the thorax and even the upper or lower part of either half. In addition, faulty posture is corrected and systematic postural drainage carried out when necessary (e.g. in obstructive or necrotic growths associated with sepsis, bronchiectasis, or lung abscess). The predominant flora of the sputum are cultured, their antibiotic

artery, than from a pulmonary one in which tension is much lower, but if a ligature slips from a large pulmonary vessel the resulting hæmorrhage is swift and catastrophic. It is vital therefore to see that all ligatures are secure. Major trunks should be doubly ligated and a safe cuff left beyond the ligature. When any doubt as to the safety of the ligature arises the vessel is transfixed and ligated proximally.

Before the advent of antibiotics (penicillin in non-tuberculous infections and streptomycin in tuberculosis) all resections of the lung were attended by a prohibitively high incidence of post-operative bronchopleural fistula, leading perhaps to fatal aspiration of the effusion on the side of operation into the opposite lung, but inevitably to contamination of the pleural cavity and the formation of an empyema. Although these disasters are now infrequent, they remain complications to be feared and avoided. The bronchus is amputated in such a fashion that no stump is left in which a potentially infected puddle of mucus may collect (Fig. 303). Meticulous care is taken in suturing the bronchus and in testing its closure afterwards, also the suture line is finally covered, preferably with an intercostal muscle graft, but failing that with a pleural flap. In non-tuberculous patients a bronchopleural fistula is most likely to occur during the first two post-operative weeks; therefore it is a wise precaution during this time to keep the fluid in the pleural cavity below the level of the bronchial stump so that if a fistula occurs, aspiration into the remaining lung is less likely. After this danger period, when sound healing is assured, it is an advantage after pneumonectomy to allow the pleural space to fill with serum, as this maintains the mediastinum in a reasonably central position without recourse to the periodic air refills which are otherwise necessary. The risk of a fistula after resection for tuberculosis is somewhat greater than after resection for non-tuberculous conditions and continues for a longer period postoperatively. Any resistance to the antibiotics used greatly increases this hazard.

The development of a bronchial fistula is usually signalled by a slight hæmoptysis, followed by a rise of temperature, an increase of dyspnoea, and eventually the expectoration of some of the serous fluid (which is stained brown by changed blood) from the pleural cavity. If the fistula is large, perhaps involving the whole of the end of the stump, there is seldom doubt about the diagnosis, but if it is small its existence can be proved decisively by measuring the intrapleural pressures on the affected side with the manometer of an A.P. apparatus. If a small fistula is present the pressure is usually atmospheric or slightly positive. Three hundred to 400 ml. of air are withdrawn and new measurements made over a period of about five minutes to see whether a negative swing is obtained and maintained. If the negative pressure is not maintained during this time the presence of a leak is proven. Methylene blue may also be injected into the pleural space and a watch kept for its appearance in the patient's sputum pot.

breath while the drain is smoothly and quickly pulled out. A *tulle gras* dressing is firmly strapped over the closed wound.

Patients leave the theatre wearing a light but well-fitting plastic oxygen mask which is used for as long as necessary, though as a rule it is not long required after consciousness has been fully regained. Oxygen is, however, available at the bedside at all times. A mask that does not fit or is not comfortable—in edentulous patients it is not always easy to secure a good fit—is worse than useless. As well as the measures usually employed to combat shock after any major operation, care must be taken to see that a sudden fall in blood pressure does not occur in the first few



FIG. 310. A water-sealed drainage tube in position after a left upper lobectomy. Note the way in which the pillows are arranged, and the tube is threaded through the slats of the back rest.

hours after the patient's return to bed. If an extensive extrapleural strip has been performed there may be considerable widespread reactionary oozing from the chest wall. A large volume of blood may gradually be lost in this way so that additional transfusion and aspiration are required. When there are patent drainage tubes they should provide early warning of bleeding, and although drains are not usually employed when a *pneumonectomy* has been performed, it is sometimes expedient to use one after difficult extrapleural pneumonectomy for pulmonary tuberculosis or bronchiectasis, provided careful watch is kept to ensure atmospheric intrapleural pressure. The treatment of continued bleeding is sealing the responsible vessels. Blood transfusion is not an alternative but may be supplementary. Bleeding is more likely to occur from one of the higher pressure systemic vessels, such as a bronchial or an intercostal

## CHAPTER 43

### DIAPHRAGMATIC HERNIAS

**DIAPHRAGMATIC** hernias are divided into congenital and acquired types. The former result from deficiencies in the diaphragm left during its somewhat complex development, the latter are commonly due to weakness of the œsophageal hiatus, or rarely to direct or indirect trauma.

**Congenital Hernias.** The coelom is divided into pleural and peritoneal cavities by the fusion of the septum transversum of the ventral mesentery with elements from the third and fourth cervical and lateral myotomes, and fusion of the dorsal mesentery with the pleuroperitoneal membranes posteriorly. Failure of any of these diverse elements to unite leaves a foramen through which herniation of abdominal contents into the chest may occur: (1) The commonest anterior deficiency is a semilunar gap, sometimes bilateral, through which the internal mammary vessels pass to reach the abdominal wall. Its flat anterior boundary is formed by the back of the sternum or the lower costal cartilages, and its curved posterior boundary by the edge of the diaphragm. The gap is called the foramen of Morgagni. As the opening is anterior it is usually the transverse colon or the omentum which herniates (Fig. 311). (2) The posterior defect which represents a continued patency of the pleuroperitoneal canal is commoner on the left side (as the liver protects the right) and is called the foramen of Bochdalek. (3) In new-born infants and small children hiatus hernias very similar to those producing symptoms in adults are encountered and are presumably due to a congenital defect of the diaphragmatic sphincter since they are often associated, within a few days of birth, with regurgitation at the beginning of feeds and persistent vomiting. The child becomes starved and dehydrated and much time may be lost in treating it for "acidosis," "cyclical vomiting" or even "pyloric stenosis." With acid reflux, an intense œsophagitis occurs and sometimes causes hæmatemesis, malæna or, more seriously still, a stricture to which the infant œsophagus seems more prone than that of the adult. Such a stricture does not often appear before the sixth month. Hernias of this type should be investigated and treated on the same lines as those occurring in the adult, but it is even more urgent to deal with them before the formation of a stricture which would enormously complicate their management.

In addition to a defect of the sphincter, the œsophageal hiatus may be abnormally wide and allow the whole, or a large part of the stomach to enter the chest. In these circumstances the fundus rotates and presents in the right pleural cavity. (4) The central tendon of the diaphragm is sometimes wanting, then bowel herniates on the left

The diagnosis of a fistula calls for prompt action. The patient is warned not to lie on the side of his intact lung in order to keep any fluid on the opposite side away from the leaking bronchus until the fluid can be completely aspirated. The withdrawn fluid is cultured and if necessary a change made in the covering antibiotics, some of which must also be injected into the space in the hope of sterilising it. If the breakdown in the bronchus has occurred early, the best treatment is to reopen the chest at once, identify the leak, and sew it up anew, but if much time has elapsed and infection has become established, the chances of success are very poor. In patients who have had a pneumonectomy it will probably be necessary to resect a rib and insert a dependent drainage tube as for an empyema, and this is likely to be permanent. But if a fistula occurs at a lobar stump and there is still expansible lung on the side of the operation, an apical thoracoplasty may succeed in bringing the chest wall into close apposition with the leak, thus sealing it off. Occasionally a small leak may heal spontaneously with the aid of antibiotics, and this is greatly aided if a muscle graft has been employed.

Following segmental resections, any air leaks are usually from the raw surfaces of the rest of the lobe. They can be very troublesome although they do not as a rule lead to early infection as does a direct bronchial leak. They are best controlled by securing the early expansion of the remaining homolateral lung, and the consequent sealing of parenchymal leaks by adherence of the lung to the chest wall. This is accomplished by drainage of both air and fluid, if necessary, assisted by suction, and the maintenance of full aeration by coughing and by respiratory exercises. Posture may also help. Thus the head-down position maintained for some days after apical resections assists the leaking parenchyma to adhere to the chest wall. If, nevertheless, a bronchiolar leak persists, two courses remain: the thoracotomy may be reopened, the leak identified and sewn up; or a limited thoracoplasty can be done involving perhaps the removal of two or three ribs over the affected area.

Special problems in postoperative management are discussed in the chapters dealing with the conditions for which pulmonary resections are performed.

cause, for the diaphragm is the most mobile and therefore the most important part of the chest wall. A large hiatal hernia, therefore, is a limited kind of sucking pneumothorax (Fig. 313).

Any of the hernias mentioned may result in bowel strangulation in infancy with all the characteristic signs of intestinal obstruction, but complicated by breathlessness and cyanosis if the deficiency is large, and



FIG. 312. The liver herniating through a deficient central tendon of the right side of the diaphragm.

in these circumstances surgical repair of the diaphragm is life saving. A tube should always be passed to aspirate the stomach contents to diminish its bulk. After location of the hernia by means of a barium meal it is approached by thoracotomy. The head of the table is tilted up to aid reduction of the hernia, and the defect is repaired either by suturing the edges together if it is small, or by darning the gap with fascia lata strips or floss silk if large.

If no obstruction occurs these hernias may persist into adult life. They may, however, be the source of dyspnoea, or vague abdominal discomfort,

and liver on the right (Fig. 312). The gap may be so large that the diaphragm is represented only by a strip of muscle around the periphery of the thorax, and abdominal viscera fill the chest.

As a rule congenital hernias have no sac, but occasionally they may be covered with a sheath of peritoneum or pleura. An intermediate stage occurs in "*eventration*" of the diaphragm when one dome, almost always



FIG. 311. Hernia of large bowel through the foramen of Morgagni (barium enema).

the left or a part of it, is very high in the chest, usually with the stomach below it. The affected dome may move paradoxically and this has led to a theory of idiopathic phrenic palsy. In fact, the eventrated part results from an extensive deficiency in the muscle, covered only by pleura and sometimes some tenuous fibrous tissue; the paradoxical movement is due to the absence of innervated muscle. The apparent thickness of such eventrations as seen on X-rays is due to contact with the stomach wall. Such defects do not as a rule affect the bowel. They often result in breathlessness and collapse and bronchiectasis of the lung above; these effects occur with any substantial diaphragmatic defect whatever its

diaphragmatic hernia is the protrusion of part of the stomach through the œsophageal hiatus. This is promoted by obesity, poor musculature, and pregnancy, and is therefore commonest in fat middle-aged women. The principal sphincter at the cardia is the right crus of the diaphragm, which is arranged like a sling round the lower end of the œsophagus, increasing the angle at which the gullet enters the stomach when it contracts. In addition, the medial fibres of the right crus surround the lower end of the œsophagus and therefore on inspiration (when the intrathoracic negative suction is greatest) normally prevent the stomach entering the chest by closing round the orifice in scissor fashion. These are the main factors preventing regurgitation of stomach contents into the œsophagus. When a sliding hiatus hernia is present and part of the stomach lies above the diaphragm, this mechanism can no longer operate, and the reflux which then occurs is the source of the characteristic symptoms. Regurgitation can take place in the absence of a hernia, but it may sometimes give rise to a hernia, for it has been demonstrated that the vagal stimulation resulting from reflux causes shortening of the longitudinal œsophageal muscle and consequent ascent of the cardia. The influence of raised intra-abdominal pressure in obesity and pregnancy is probably more important, especially as such subjects usually have poor musculature as well. It may be significant that many patients with a hiatus hernia have associated gall bladder or duodenal disorders which could be a source of vagal stimulation. Gastro-œsophageal regurgitation is probably responsible for the symptoms in 25 to 30 per cent. of all patients complaining of dyspepsia, and in the majority, though not in all, a sliding hiatus hernia can be demonstrated radiologically. Since the cardia is protruded through a wide hiatus, the hernia has only a frontal sac and is likely to reduce itself spontaneously when the patient stands erect. It is, therefore, essential that during a barium meal examination the patient should bend down and touch the toes while standing laterally against the X-ray screen. Alternatively, the Trendelenburg position can be used to demonstrate the protrusion and accompanying reflux. Failure to carry out these manœuvres results in the condition not being recognised, and the unfortunate patients are condemned to become chronic "dyspeptics." Retrosternal or epigastric pain and flatulence, "heart-burn" and acid regurgitation without nausea, brought on by bending or recumbency and relieved by standing upright, or by using several pillows at night, are characteristic of hiatus hernia.

The pain associated with a sliding hiatal hernia and its accompanying reflux œsophagitis is not directly related to food, but rather to posture, coming on when the patient bends down or goes to bed. It is recurrent and burning in character, usually high in the epigastrium or behind the lower end of the sternum, but sometimes it is felt precordially and radiating to the back, the neck or the left arm. As bending is frequently connected with effort, such as scrubbing floors, or gardening, it is very



or they may only be discovered on routine radiography ; then, if containing only omentum or liver, they resemble intrathoracic tumours. It is, however, much commoner for breaking-down growths and other abscesses to be mistaken for diaphragmatic hernias, although abscesses are very common whereas diaphragmatic hernias are rare.

**Traumatic Hernias.** These are caused directly by penetrating wounds,



FIG. 313. "Eventration" of the left diaphragm. The patient was very short of breath and the whole left lung severely bronchiectatic. The structure resembling the diaphragm here is in fact the stomach wall covered only by attenuated amuscular diaphragm.

or indirectly by crushing injuries of the chest resulting in rupture of the central tendon of the left dome and extrusion of the stomach and intestine into the left side of the chest, usually with accompanying hæmo-thorax. Both direct and indirect injuries of the diaphragm call for operation with reposition and repair of injured bowel if necessary, and reconstruction of the diaphragm. As the left side is usually affected, rupture of the spleen is a commonly associated injury.

**Hiatus Hernia.** Much the commonest and most important variety of

expected from operation. It is well worth while restoring the cardia to its normal position and repairing the hernia even when much œsophagitis and spasm are present providing there is not an organic fibrous stricture. In most cases complete relief of symptoms follows, and if the barium swallow is repeated after operation it will be seen that no reflux occurs and that the appearance of the mucosa has returned to normal.

**Operation for Hiatus Hernia.** With the patient in the left lateral position the seventh rib is resected subperiosteally and the pleural space entered. The lower lobe of the left lung is packed upwards with a warm wet towel and retracted. The layers of pleura constituting the inferior pulmonary ligament are opened thus exposing the œsophagus and the bulging gastric hernia protruding through the hiatus, which is seen and felt to be patulous. It is partially covered by a sac of peritoneum. This is dissected free and the lower end of the œsophagus mobilised sufficiently far up to make reposition easy. This is nearly always possible, and is only difficult when real shortening has taken place due to cicatrization ("congenital short œsophagus" is a fiction). Care must be taken while freeing the œsophagus not to open the opposite pleura, but if this is inadvertently done the hole must be stitched up again. A length of plastic tape is passed round the œsophagus lifting it from the mediastinum (Fig. 314a). The central tendon of the diaphragm is picked up and incised for a short distance towards the hiatus so that its undersurface can be seen. The reflexion of peritoneum on to it from the stomach is cut and the oval muscle sling (right crus) of the hiatus is then clearly visible (Fig. 314a). A curved forcep is passed up between this and the stomach wall from below; the tapes round the œsophagus are grasped and drawn down and through the diaphragmatic incision, reducing the herniation and keeping the cardia in place (Fig. 314b). Fine interrupted linen thread sutures are then placed at intervals around the cardia anchoring it to the adjacent undersurface of the diaphragm. The deficiency in the hiatus is now repaired (Fig. 314b). The œsophagus is held forward and the substantial posterior gap between the muscular insertions of the right crus inspected. These muscles, which encircle the œsophagus in a well-defined ring anteriorly, diverge posteriorly and curve downwards in an arc to their origins on the bodies of the first three lumbar vertebræ. The left-hand margin is clearly visible (if all the fatty areolar tissue has been properly cleaned from the hiatus), but the right edge lies medially, and may not at first be so apparent. It can, however, always readily be felt between index finger and thumb, and drawn into view. A series of stout interrupted No. 25 linen thread sutures are inserted, but at first are left untied. They extend from the most posterior part of the hiatus—a sound bite of tissue being secured on either side—to a point close behind the œsophagus (Fig. 314b). They are then drawn together so that an estimate of the resulting size of the hiatus can be made, and if this is satisfactory they are tied. The closure of the hiatus should be snug but

easy to confuse this pain with that of angina pectoris. Indeed the nerve paths of both types of pain are the same (experimental distension of the stomach decreases coronary flow). Furthermore, electrocardiographs taken when a hernia is present may indicate coronary insufficiency, but the tracings are normal when the hernia is reduced. Nazum found a hiatus hernia present in 25 per cent. of patients with the diagnosis of angina pectoris, but in only 12 per cent. of patients given barium meals for other reasons.

Much less common than the sliding hiatus hernia is the paræsoophageal type in which the *fundus* of the stomach rolls up through the hiatus to lie beside the œsophagus, while the cardia remains in its normal place below the diaphragm. As reflux does not then occur, the only symptoms to which such a hernia gives rise are substernal discomfort, belching, and intermittent dysphagia. More serious and consistent dysphagia results from the œsophagitis caused by acid reflux in the sliding variety, and this in turn may proceed to actual peptic ulceration at the lower end of the gullet. The œsophagitis causes spasm which may be intense and make the passage of a bougie difficult even though no true stricture or ulceration is present. The mucosa is reddened, swollen, superficially excoriated, and bleeds easily. If a true peptic ulcer is present insidious bleeding, with occult blood in the stools, is not infrequent, and frank hæmatemesis or malæna sometimes occur. As well as swelling of the mucosal and muscular coats there is a marked inflammatory reaction in the mediastinal tissues related to the œsophagus, which thus becomes fixed and adherent to the pleuræ, the diaphragm, the pericardium and the aorta, and in some instances perforation takes place into the pleural cavity with fistula formation and empyema. More catastrophically still such ulcers have been known to penetrate the heart or aorta. At best, long-standing œsophagitis and ulceration cause fibrosis and real shortening of the œsophagus, as opposed to the apparent shortening which accompanies spasm. As healing takes place an organic stricture is likely to remain. It is probable that the vast majority of innocent strictures of the lower end of the œsophagus are so caused, and as repair of a hernia presents grave if not insurmountable difficulties in the presence of severe chronic œsophagitis or ulceration, there is every reason to deal promptly with sliding hiatus hernias before œsophagitis is established.

After a sliding hernia has been diagnosed by the means described above, the patient is œsophagoscoped in order that the extent of the herniation, the degree of œsophagitis and spasm, and the presence of ulceration or stricture can be ascertained. It is also essential to exclude the possibility of a carcinoma of the œsophagus. In most hiatus hernias the instrument can be passed easily through the œsophagus into the stomach, but if there is much spasm due to inflammation this may not be possible, and indeed it is unwise to try; a large gum elastic bougie is tried instead. If the mucosa is actively inflamed much improvement is to be

expected from operation. It is well worth while restoring the cardia to its normal position and repairing the hernia even when much œsophagitis and spasm are present providing there is not an organic fibrous stricture. In most cases complete relief of symptoms follows, and if the barium swallow is repeated after operation it will be seen that no reflux occurs and that the appearance of the mucosa has returned to normal.

**Operation for Hiatus Hernia.** With the patient in the left lateral position the seventh rib is resected subperiosteally and the pleural space entered. The lower lobe of the left lung is packed upwards with a warm wet towel and retracted. The layers of pleura constituting the inferior pulmonary ligament are opened thus exposing the œsophagus and the bulging gastric hernia protruding through the hiatus, which is seen and felt to be patulous. It is partially covered by a sac of peritoneum. This is dissected free and the lower end of the œsophagus mobilised sufficiently far up to make reposition easy. This is nearly always possible, and is only difficult when real shortening has taken place due to cicatrisation ("congenital short œsophagus" is a fiction). Care must be taken while freeing the œsophagus not to open the opposite pleura, but if this is inadvertently done the hole must be stitched up again. A length of plastic tape is passed round the œsophagus lifting it from the mediastinum (Fig. 314a). The central tendon of the diaphragm is picked up and incised for a short distance towards the hiatus so that its undersurface can be seen. The reflexion of peritoneum on to it from the stomach is cut and the oval muscle sling (right crus) of the hiatus is then clearly visible (Fig. 314a). A curved forcep is passed up between this and the stomach wall from below; the tapes round the œsophagus are grasped and drawn down and through the diaphragmatic incision, reducing the herniation and keeping the cardia in place (Fig. 314b). Fine interrupted linen thread sutures are then placed at intervals around the cardia anchoring it to the adjacent undersurface of the diaphragm. The deficiency in the hiatus is now repaired (Fig. 314b). The œsophagus is held forward and the substantial posterior gap between the muscular insertions of the right crus inspected. These muscles, which encircle the œsophagus in a well-defined ring anteriorly, diverge posteriorly and curve downwards in an arc to their origins on the bodies of the first three lumbar vertebræ. The left-hand margin is clearly visible (if all the fatty aveolar tissue has been properly cleaned from the hiatus), but the right edge lies medially, and may not at first be so apparent. It can, however, always readily be felt between index finger and thumb, and drawn into view. A series of stout interrupted No. 25 linen thread sutures are inserted, but at first are left untied. They extend from the most posterior part of the hiatus—a sound bite of tissue being secured on either side—to a point close behind the œsophagus (Fig. 314b). They are then drawn together so that an estimate of the resulting size of the hiatus can be made, and if this is satisfactory they are tied. The closure of the hiatus should be snug but

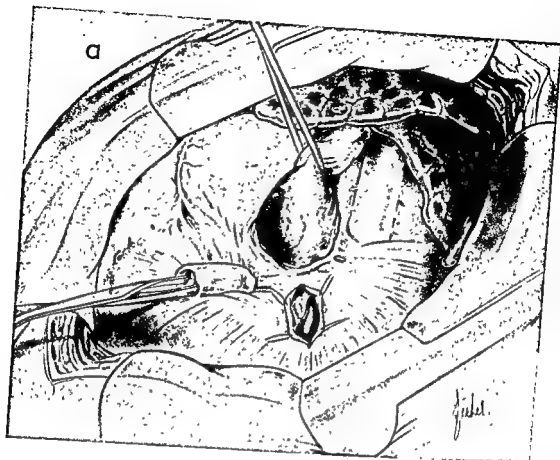


FIG. 314. (a) The oesophagus and the hiatus hernia itself have been mobilised and the central tendon of the diaphragm incised. (b) The hernia is now reduced and the crural deficiency repaired. (c) Operation completed.

not tight. The œsophagus now lies in close apposition to the anterior ring of muscle of the hiatus, and with a firm, solid block of tissue behind it. The tape is removed from the œsophagus and the incision in the tendon of the diaphragm closed with mattress sutures (Fig. 314c). The pleura is not closed over the mediastinum. The lung is re-expanded and the chest closed without drainage.

On no account should the phrenic nerve be interfered with at any stage of this operation. The practice of crushing it to make reposition easier or as a routine procedure is reprehensible. The diaphragm is the most mobile and therefore valuable part of the chest wall, especially in the elderly who have lost most of their rib mobility, and to paralyse the diaphragm deliberately is indefensible. Without its mechanism it becomes difficult or impossible for the patient to expel the secretions in the base of his lung, so that atelectasis and infection result, sometimes with fatal consequences. At best the patient is deprived, often permanently, of a substantial part of the vital capacity of one lung, for 40 per cent. of crushed phrenic nerves never recover. For the same reason treatment of hiatal hernia by phrenic crush alone is to be deplored. It is true that some patients are thereby relieved of their symptoms because the diaphragm rises so high that the hernia is, as it were, overtaken, and the cardia returns to the abdomen, but there can be no justification for depriving a man of his breath to cure his indigestion.

In those cases in which a true stricture has formed and there is serious difficulty in swallowing, dilatation with bougies should be tried, and the patient instructed to avoid bending, to sleep always propped up with several pillows, and to take alkalis. Dilatation, however, may result only in increased regurgitation and further œsophagitis, and therefore in a proportion of cases it is necessary to excise the stricture. This comprises a transthoracic partial gastrectomy and œsophagogastrostomy. At first sight this would appear to be merely reconstituting the original lesion, as the stomach is again brought above the diaphragm. On these grounds some surgeons perform œsophagojejunostomy, but in the procedure just described *most of the acid-secreting portion of the stomach has been removed and both vagi are divided*. In practice the results of excision are satisfactory.

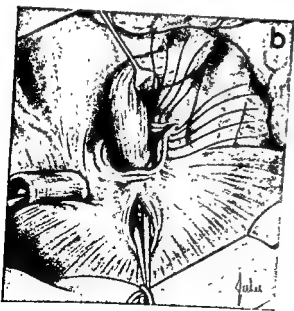
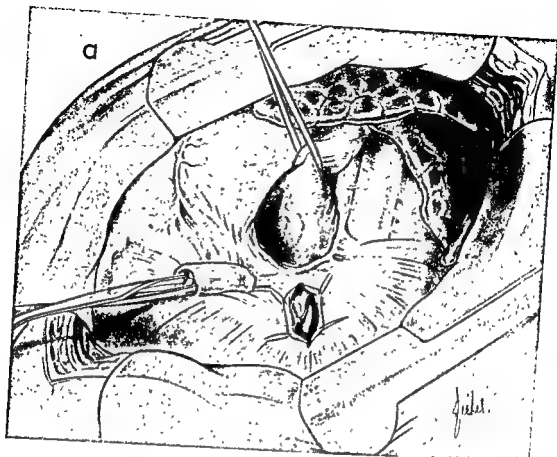


FIG. 314. (a) The oesophagus and the hiatus hernia itself have been mobilised and the central tendon of the diaphragm incised. (b) The hernia is now reduced and the crural deficiency repaired. (c) Operation completed.

site of damage is the posterior wall in the cervical region, but instrumentation in the presence of malignancy also often leads to rupture elsewhere. As in bronchoscopy, the removal of foreign bodies must be planned according to the object to be removed. Sharp points or cutting edges are drawn up within the instrument to prevent laceration of the oesophageal wall (Figs. 315 and 316). If endoscopic removal proves to be hazardous, thoracotomy should be performed instead, usually on the right side. The oesophagus is incised over the object which is then extracted, the mucosa



FIG. 316. A dental plate of difficult shape lodged in the upper third of the oesophagus. Such objects are sometimes best broken up before extraction. (X-ray by Dr. J. M. Stewart.)

and muscle wall are repaired, and the chest closed. The presence of an aneurysm or recent swallowing of a corrosive is an absolute contra-indication to passage of an oesophagoscope.

If there is any suspicion of damage during oesophagoscopy, the patient should be given penicillin, and a close watch kept for tenderness, redness or swelling in the supraclavicular region or suprasternal notch. Surgical emphysema as shown by crepitus in these situations is diagnostic of a tear in the oesophagus; an X-ray may show air or fluid, or both, in one or other pleural cavity. There may be fever and a rising pulse rate. Sometimes perforation is accompanied by rigidity of the upper abdominal muscles, severe pain in the chest or abdomen, and shock. These signs accompany oesophageal tears from any cause. Rarely "spontaneous"



## CHAPTER 44

### THE ŒSOPHAGUS

#### ŒSOPHAGOSCOPY AND RUPTURE OF THE ŒSOPHAGUS

ŒSOPHAGOSCOPY is an indispensable step in the investigation of all patients with dysphagia, the commonest intrinsic causes of which are carcinoma, hiatus hernia with œsophagitis, cardiospasm, innocent stricture and large pharyngeal pouch. As well as growths of the gullet and

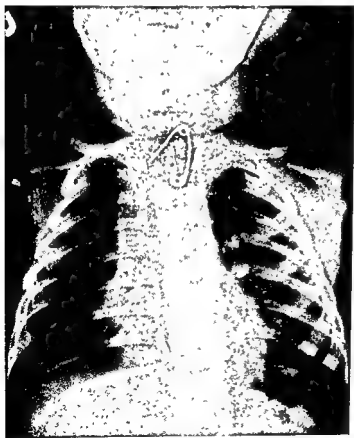


FIG. 315. An open safety pin in a child's gullet. In such a position the pin should be seized at the loop and withdrawn with the point trailing. (X-ray by Dr. J. M. Stewart.)

cardiac end of the stomach, bronchial carcinomas and their mediastinal metastases often infiltrate and obstruct the œsophagus. Œsophagoscopy is also used for the removal of foreign bodies, dilatation of strictures and cardiospasm, and the insertion of radon needles or of a Souttar's tube in some inoperable cases of cancer.

The procedure is much more dangerous than bronchoscopy and should therefore be carried out only by those specially trained. The vast majority of perforations of the œsophagus follow œsophagoscopy ; the commonest

result of acid regurgitation. The great majority are acquired in later life in the same way and are therefore found at the lower end of the œsophagus, often associated with an hiatus hernia needing surgical correction. The treatment of this type of stricture is discussed under hiatus hernia. Other non-malignant strictures follow the swallowing of corrosives or scalding water, or occasionally the prolonged impaction of a foreign body, or operations upon the œsophagus. Like simple strictures elsewhere in the body they respond to, and are best treated by, periodic dilatation, and they tend to recur ("once a stricture, always a stricture").



FIG. 317. Stricture at junction of middle and lower thirds of œsophagus after the swallowing of a corrosive. (X-ray by Dr. J. M. Stewart.)

The pharyngeal muscles open widely during the act of deglutition so that when caustics or strong acids are swallowed, or a child drinks from the spout of a boiling kettle, the resultant stricture tends to be well down the œsophagus, below the level of the aortic arch (Fig. 317). In those who survive, the formation of stricture is indicated by slowly increasing dysphagia, although in some patients obstruction is sudden, due to solid food becoming impacted in the narrowed part. The nature of the lesion and its extent is determined by a barium swallow followed by œsophagoscopy. In contrast with what happens in cardiospasm, the œsophagus never becomes *markedly* dilated as a result of fibrous stricture; a channel, even if very narrow, remains; it is lined by mucosa and can be dilated.

rupture is seen after severe vomiting, when the rent is usually longitudinal and situated just above the diaphragm posterolaterally on the left side. Ingestion by mouth must be stopped at once, the site of injury explored, and the necessary repair carried out. The chest is then closed, water-sealed drainage is established, and the lung re-expanded. Gastrostomy is not as a rule required as fluids by mouth can usually be started again after twenty-four to forty-eight hours.

The most useful œsophagoscope is the Negus type which has a wide flattish bore, and two lamps set obliquely in the walls at the proximal end. It is 45 cm. in length, but a shorter one of 35 cm. should also be available for use when only the upper reaches of the œsophagus are to be examined. Powerful suction and several wide-bored aspirating tubes must always be at hand together with a selection of biopsy and grasping forceps, gum elastic bougies and a hydrostatic dilator bag.

Oesophagoscopy is best performed under a good general anæsthetic given through an intratracheal tube. The patient's head lies in a bronchoscopy head-rest, with the shoulders overlapping the end of the table, but instead of the head being extended as in bronchoscopy, it should be somewhat flexed upon the neck, and the neck in turn flexed upon the trunk. Flexion of the neck is particularly important as extension causes the cervical vertebral bodies to press forward against the posterior œsophageal wall, and if an instrument is then passed there is great risk of damage to the œsophageal wall (this can be produced not only by the advancing beak of the œsophagoscope but also by backward pressure of the tube against the vertebræ). The instrument is passed into the mouth, usually to one side, and guided downwards with the fingers of the left hand which also prevent it bearing upon the teeth. The epiglottis is identified and lifted forward. The beak is then passed into the pharynx behind the arytenoids to the back of the cricoid cartilage. This is the difficult stage and the one during which most injuries occur; passage of the instrument is greatly facilitated at this level by the anæsthetist grasping the whole larynx in his hand and gently lifting it forward. The lumen at once opens up and can be followed down—always under direct vision—aided by frequent aspiration. *No force must ever be applied*; if the instrument does not slide forward easily it is either going in the wrong direction or a stricture has been encountered. When the instrument passes below the level of the aortic arch the patient's head is slightly extended and moved to the right, and there is usually a little upward lift of the beak needed as it passes into the cardia. This point is recognised by the abrupt change in the appearances of the mucosa and reflux of gastric juice. Measurements of any abnormalities encountered are made in centimetres from the upper incisors or gums.

---

**Simple Strictures.** Non-malignant strictures of the œsophagus are rarely congenital; most of those seen in small children are probably the

of the cells of Auerbach's plexus as seen histologically is probably a secondary phenomenon occurring late in the disease and not, as has been suggested, the cause of the disorder. Cardiospasm affects women rather more than men. Dysphagia is the characteristic symptom and comes on in early adult life. The onset is usually insidious but occasionally sudden. It has been estimated that about 20 per cent. of patients with dysphagia suffer from cardiospasm. The distended oesophagus usually contains a considerable quantity of undigested food, which may spill over during sleep and be aspirated into the lung, giving rise to chronic suppurative pneumonitis. Thus patients may seek advice for pulmonary manifestations (cough, purulent sputum, and sometimes low-grade fever) instead of dysphagia.

Patients complain that at first solid food seems to stick at the level of the lower end of the sternum, but after a pause, or drinking, it passes on. Later, semi-solids and even fluids are held up for longer and longer periods and often regurgitated unchanged. There is not the slightest evidence of psychoneurosis in patients with cardiospasm as some observers have suggested, but when the disorder has been present for a long time nutrition may be gravely impaired and patients are weak and wasted.

*Investigations.* Plain radiography of the chest is followed by a barium swallow. The former shows the presence of any lung infection and the latter shows a wide column of the barium held up at the cardia and a thin trickle of barium reaching the stomach (Fig. 318). The administration of amyl nitrite usually produces a temporary relaxation of the circular muscle and allows barium to flow through, thus helping to distinguish the obstruction from that caused by neoplastic or other types of stricture.

Oesophagoscopy is, however, essential to exclude a carcinoma or peptic ulcer at the lower end of the oesophagus. Before this is carried out, and certainly before any anaesthetic is given, the gullet must be washed out to prevent overflow into the lungs.

*Treatment.* The treatment of cardiospasm by the passage of mercury-filled bougies is ineffective and outmoded. It causes discomfort and inconvenience and drastically curtails social life, for patients can never dine out with friends, travel, or visit a restaurant.

If the patient is seen early in the course of the disease it is worth dilating the affected part of the oesophagus with a Negus hydrostatic bag. This is passed empty down the oesophagoscope on an introducer until it is firmly gripped by the constricting muscles at the lower end of the oesophagus. The bag is then filled slowly with water to stretch the muscle fibres fully and evenly. This procedure affords complete and permanent relief in many early cases. When it fails, or if the method is not indicated because of the severity or duration of the disorder, nutrition is first improved, any lung infection is treated, and operation is subsequently carried out.

*Heller's Operation.* The oesophagus is washed out thoroughly daily for

Dilatation is first attempted during œsophagoscopy ; a fine gum elastic bougie is threaded through, followed by larger sizes. Future dilatations should be carried out by the patient if sufficiently co-operative. The advantages of self-treatment are that it can be done as often as necessary, calling for no anæsthetic, and, furthermore, it is helped by the swallowing mechanism which is in abeyance during œsophagoscopy. The patient sits up, takes some deep breaths, and then passes a small gum elastic bougie, softened in warm water and lubricated with olive oil, into the mouth, and begins to swallow it as soon as it reaches the pharynx. The lips are closed round the bougie and its passage is helped by continued swallowing and gentle manipulation. Force must never be used, and the process must never cause pain. Initially only fine bougies can be passed, but the size is gradually increased, and the largest bougie swallowed should be left in position as long as possible. At first, this should be done three or four times a day, but later the intervals may be lengthened. Solid food is encouraged as each bolus acts itself as a dilator. Usually this method succeeds, but if it does not, or if ulceration is present, physiological rest is called for and therefore a gastrostomy should be performed. After a time dilatation may prove quite easy. In more difficult cases the retrograde method should be tried : a ureteric catheter, or even a thread, is swallowed, and the end recovered through the gastrostomy. To it is attached a bougie with an eye in the tip, and this is drawn back up the œsophagus guided by the thread or catheter until it passes the stricture. This is easier than passage from above because the lumen of the œsophagus below the stricture is a narrow cone leading to the orifice, whereas the lumen above the stricture may be wide and irregular. Once a way through is established, bougies can be swallowed and the gastrostomy allowed to close.

Surgery is reserved for those strictures which resist dilatation or for those patients who refuse to dilate them. If a stricture is confined to a very narrow segment it may be possible to excise it locally, though this is the very type most suitable for dilatation, otherwise the best operation is some form of intrathoracic œsophagogastronomy as performed for carcinoma of the œsophagus or cardia.

**Cardiospasm.** The essential mechanism in cardiospasm is idiopathic spasm of the circular muscle fibres at the lower end of the œsophagus. In some cases, though very rarely, the circular muscle in this situation is hypertrophied, but never to the extent seen in congenital pyloric stenosis ; usually no macroscopic change whatever is apparent. At first, contractility of the rest of the œsophagus is normal or even hyperactive, but with continued obstruction to the passage of food passive distension occurs, peristalsis is lost, and the gullet may assume vast proportions. It lies " S " shaped in the mediastinum so that it bulges to the left inferiorly and to the right in the superior mediastinum. Thus it may be mistaken radiographically for an intrinsic mediastinal tumour. The degeneration

of Auerbach's plexus as seen histologically is probably a phenomenon occurring late in the disease and not, as has been a cause of the disorder. Cardiospasm affects women rather than men. Dysphagia is the characteristic symptom and comes on with age. The onset is usually insidious but occasionally sudden. It is estimated that about 20 per cent. of patients with dysphagia have cardiospasm. The distended œsophagus usually contains a quantity of undigested food, which may spill over during inspiration into the lung, giving rise to chronic suppurative pneumonia. These patients may seek advice for pulmonary manifestations (purulent sputum, and sometimes low-grade fever) instead of

complain that at first solid food seems to stick at the level of the sternum, but after a pause, or drinking, it passes on. Solids and even fluids are held up for longer and longer periods regurgitated unchanged. There is not the slightest evidence of obstruction in patients with cardiospasm as some observers have thought when the disorder has been present for a long time. They are gravely impaired and patients are weak and wasted.

*Diagnosis.* Plain radiography of the chest is followed by a barium meal. The former shows the presence of any lung infection and the latter a wide column of the barium held up at the cardia and a thin column reaching the stomach (Fig. 318). The administration of nitroglycerine usually produces a temporary relaxation of the circular muscle allowing barium to flow through, thus helping to distinguish the disorder from that caused by neoplastic or other types of stricture. Roentgenoscopy is, however, essential to exclude a carcinoma or peptic ulcer at the lower end of the œsophagus. Before this is carried out, and before any anæsthetic is given, the gullet must be washed out to allow barium to flow into the lungs.

*Treatment.* The treatment of cardiospasm by the passage of mercury is ineffective and outmoded. It causes discomfort and distress and drastically curtails social life, for patients can never leave their homes, friends, travel, or visit a restaurant.

*Hydrotherapy.* If the patient is seen early in the course of the disease it is worth attempting to dilate the affected part of the œsophagus with a Negus hydrostatic bag. The bag is inserted empty down the œsophagoscope on an introducer until it is stopped by the constricting muscles at the lower end of the

The bag is then filled slowly with water to stretch the muscle evenly. This procedure affords complete and permanent relief in many early cases. When it fails, or if the method is not indicated by the severity or duration of the disorder, nutrition is first maintained and any lung infection is treated, and operation is subsequently

*Operation.* The œsophagus is washed out thoroughly daily for

Dilatation is first attempted during œsophagoscopy ; a fine gum elastic bougie is threaded through, followed by larger sizes. Future dilatations should be carried out by the patient if sufficiently co-operative. The advantages of self-treatment are that it can be done as often as necessary, calling for no anæsthetic, and, furthermore, it is helped by the swallowing mechanism which is in abeyance during œsophagoscopy. The patient sits up, takes some deep breaths, and then passes a small gum elastic bougie, softened in warm water and lubricated with olive oil, into the mouth, and begins to swallow it as soon as it reaches the pharynx. The lips are closed round the bougie and its passage is helped by continued swallowing and gentle manipulation. Force must never be used, and the process must never cause pain. Initially only fine bougies can be passed, but the size is gradually increased, and the largest bougie swallowed should be left in position as long as possible. At first, this should be done three or four times a day, but later the intervals may be lengthened. Solid food is encouraged as each bolus acts itself as a dilator. Usually this method succeeds, but if it does not, or if ulceration is present, physiological rest is called for and therefore a gastrostomy should be performed. After a time dilatation may prove quite easy. In more difficult cases the retrograde method should be tried : a ureteric catheter, or even a thread, is swallowed, and the end recovered through the gastrostomy. To it is attached a bougie with an eye in the tip, and this is drawn back up the œsophagus guided by the thread or catheter until it passes the stricture. This is easier than passage from above because the lumen of the œsophagus below the stricture is a narrow cone leading to the orifice, whereas the lumen above the stricture may be wide and irregular. Once a way through is established, bougies can be swallowed and the gastrostomy allowed to close.

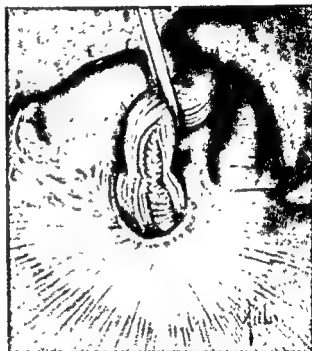
Surgery is reserved for those strictures which resist dilatation or for those patients who refuse to dilate them. If a stricture is confined to a very narrow segment it may be possible to excise it locally, though this is the very type most suitable for dilatation, otherwise the best operation is some form of intrathoracic œsophagogastrostomy as performed for carcinoma of the œsophagus or cardia.

**Cardiospasm.** The essential mechanism in cardiospasm is idiopathic spasm of the circular muscle fibres at the lower end of the œsophagus. In some cases, though very rarely, the circular muscle in this situation is hypertrophied, but never to the extent seen in congenital pyloric stenosis ; usually no macroscopic change whatever is apparent. At first, contractility of the rest of the œsophagus is normal or even hyperactive, but with continued obstruction to the passage of food passive distension occurs, peristalsis is lost, and the gullet may assume vast proportions. It lies " S " shaped in the mediastinum so that it bulges to the left inferiorly and to the right in the superior mediastinum. Thus it may be mistaken radiographically for an intrinsic mediastinal tumour. The degeneration

oesophagus, and for at least 2 inches on to the stomach wall. Operative failures are due to insufficient depth and length of this incision. The chest is closed without drainage. After twenty-four hours on fluids, patients are given minces and purées, and after three or four days a normal solid diet. Patients are usually able to get up within forty-eight hours and leave hospital in ten days. The results of this operation are excellent and the risks negligible. The condition does not recur.

**Carcinoma of the Oesophagus.** Cancer of the oesophagus is about eight times commoner in men than in women, thus mirroring the sex incidence

FIG. 319. Heller's operation completed. Note the incision in the muscle is left unsutured.



of cancer of the bronchus (males to females : 10 to 1), but is much less frequent than carcinoma of the bronchus, comprising some 5 per cent. of cancers and accounting for rather more than 2,000 deaths annually in England. Most of these growths are squamous-celled, ranging from well-differentiated keratinising tumours to very anaplastic ones, but at the lower end of the oesophagus adenocarcinomas occur growing either upwards from the stomach or arising in ectopic gastric mucous membrane. Carcinomas are peculiarly prone to extend unseen upwards and downwards in the submucous layer of the oesophagus. Therefore, it is always necessary to leave a substantial cuff of apparently healthy tissue on both sides of a resected neoplasm. Patients, however, tend to die of starvation rather than of extension of the tumour. In 1,025 post-mortems, Ochsner found no evidence of metastatic spread in 418 cases (40·7 per cent.). Fifty per cent. of growths in the lowest third and 30 per



a week preceding operation. The left side of the chest is entered through a standard posterolateral incision in the seventh intercostal space and the lower lobe of the lung packed upwards. The layers of the inferior pulmonary ligament are opened and the lower end of the œsophagus mobilised mostly by blunt dissection, but care is taken not to open the opposite pleura. A plastic tape is then passed round the œsophagus and the hiatus cleared of areolar tissue so that an inch or so of the cardia can be gently drawn up and exposed. Only occasionally is it necessary to



FIG. 318. Barium in the œsophagus shows great but smooth and symmetrical dilatation. The constriction is at the level of the diaphragm. (X-ray by Dr. J. M. Stuart.)

make a small incision in the diaphragm to obtain an adequate exposure of the gastro-œsophageal junction; the incision is later closed with interrupted sutures. The œsophagus is held firmly with the fingers of the left hand and the longitudinal and circular muscle coats are incised until the mucosa appears in the gap (cf. Rammstedt's operation for pyloric stenosis). A vascular ring at the cardia is tied and divided (Fig. 319). The pressure of the fingers puts these muscle fibres on the stretch and the mucosa bulges conspicuously outward as soon as they are divided. It is, of course, important, and generally quite easy, to avoid injury to the mucosa, but if a hole is inadvertently made it is at once repaired with a few fine interrupted sutures. Danger lies only in an unnoticed wound of the mucosa. The myotomy should extend for about 3 inches up the

infallible, for peptic ulceration may produce a remarkably similar appearance. Oesophagoscopy is indispensable in all cases of dysphagia and particularly when malignancy is suspected. Great care must always be exercised because of the increased risk of perforation in the presence of a carcinoma. When a growth or ulceration is encountered, an estimate is made of its mobility or fixation, and its distance measured from the incisor teeth. A biopsy is also carried out. *Bronchoscopy* must always be performed to exclude infiltration of the left main bronchus or erosion with fistula formation.



FIG. 520. Filling defect of the oesophagus due to a carcinoma at junction of middle and lower thirds. Contrast thus appearance with that seen in cardio-sperm and simple stricture. (X-ray by Dr. J. M. Stewart.)

**Operation.** Whenever possible an attempt should be made completely to excise the neoplasm, or at least to restore the patient's ability to swallow if radical extirpation is impracticable. When even local removal is impossible a short circuit should be established. Multiple stage operations and ante-thoracic (i.e. subcutaneous) reconstructions are outmoded. Gastrostomy is sometimes unavoidable, but should certainly not be done if the patient's condition can be sufficiently restored without it. A Stamm inverted inkpot type of gastrostomy with an indwelling feeding tube is best. It must be remembered that a gastrostomy does little to palliate the condition of a patient who cannot swallow saliva.

Prior to operation, at least a week to ten days must be devoted to

cent. in the middle third of the œsophagus metastasise below the diaphragm. Therefore, radical operations must include removal of the regional glands along the lesser curve of the stomach and the left gastric artery. Twenty per cent. of carcinomas of the œsophagus arise in the superior third of the organ (from the upper end to the arch of the aorta), 37 per cent. in the middle third (from the arch of the aorta to the inferior pulmonary vein), and 43 per cent. in the lowest third.

*Symptoms.* As with most other cancers, symptoms are often absent until late in the disease. Patients may notice that a longer time is taken over meals (which may be left unfinished) long before definite dysphagia occurs, or there may be vague discomfort described as "indigestion." Also among early symptoms may be an unproductive cough, arising from uneasiness in the gullet. Later, difficulty is experienced in swallowing dry solids; then there is occasional regurgitation, and dysphagia with moist foods, and finally with semi-solids. Often patients can localise with fair accuracy the level behind the sternum at which food is arrested. Occasionally a sudden inability to swallow may be the first and only symptom. To the wasting of the early stages of the disease is later added the much more rapid wasting of inanition, and patients may present themselves in a state of severe starvation and dehydration. Eventually they are unable to swallow even their saliva.

Pain between the scapulae, if boring in character, is likely to presage an inoperable growth. Persistent fever arises from infection in a necrotic and eroding growth. Hoarseness spells involvement of the left recurrent laryngeal nerve. Rectal examination must never be omitted for it may reveal peritoneal metastasis in the pelvis. As the venous return from œsophageal and high gastric areas enters the inferior vena cava directly instead of passing through the portal system, deposits in the lungs may precede metastasis to the liver. If any of these obvious contraindications to surgery are present, or the age and condition of the patient forbid it, palliative treatment is indicated.

*Investigations.* Following complete physical examination, the chest is X-rayed and the patient screened after a barium swallow. The passage of the barium is observed, and any delay, spasm, filling defect or disorder of peristalsis noted. The patient is then put in the Trendelenburg position and the fundus and cardia of the stomach are outlined to show any tendency to herniation or regurgitation. If a stricture is present this manœuvre also allows its lower margins to be examined. In œsophageal carcinoma, dilatation above the stricture is seldom marked and never occurs to the extent seen in cardiospasm. Furthermore, the stricture may be at any level, whereas cardiospasm only occurs at the lower end of the œsophagus (Fig. 320). The outline of a carcinoma is usually irregular and asymmetrical. A narrow *tortuous* track of barium winds through the lumen left by the growth.

Although a barium swallow is often diagnostic, it is by no means

infallible, for peptic ulceration may produce a remarkably similar appearance. Oesophagoscopy is indispensable in all cases of dysphagia and particularly when malignancy is suspected. Great care must always be exercised because of the increased risk of perforation in the presence of a carcinoma. When a growth or ulceration is encountered, an estimate is made of its mobility or fixation, and its distance measured from the incisor teeth. A biopsy is also carried out. *Bronchoscopy* must always be performed to exclude infiltration of the left main bronchus or erosion with fistula formation.



FIG. 320. Filling defect of the oesophagus due to a carcinoma at junction of middle and lowest thirds. Contrast this appearance with that seen in cardiospasm and simple stricture. (X rays by Dr. J. M. Stewart.)

**Operation.** Whenever possible an attempt should be made completely to excise the neoplasm, or at least to restore the patient's ability to swallow if radical extirpation is impracticable. When even local removal is impossible a short circuit should be established. Multiple stage operations and ante-thoracic (i.e. subcutaneous) reconstructions are outmoded. Gastrostomy is sometimes unavoidable, but should certainly not be done if the patient's condition can be sufficiently restored without it. A Stamm inverted inkpot type of gastrostomy with an indwelling feeding tube is best. It must be remembered that a gastrostomy does little to palliate the condition of a patient who cannot swallow saliva.

Prior to operation, at least a week to ten days must be devoted to

restoring the patient's nutrition and electrolyte-fluid balance. Transfusion may be necessary for anaemia.

Growths at the lower end of the oesophagus (i.e. below the level of the inferior pulmonary vein) are best approached by a combined left postero-lateral thoracotomy and laparotomy. The incision is made along the line of the seventh rib, and continued across the upper part of the left rectus muscle and, if need be, downwards in the midline. The latter part of the incision is completed after resection of the ribs and opening the thorax has demonstrated the operability of the growth, or the possibility of performing a short-circuit. The costal cartilage is then divided and the diaphragm opened in radial fashion from the hiatus outwards to the periphery, where the muscles of the anterior abdominal wall and the peritoneum are divided. [If more room is required, or if it is decided to perform a radical removal of stomach, spleen and tail of pancreas, this incision can be extended even further down the midline to just above the umbilicus. The operating table is tilted to the left for this part of the operation. The liver, the omentum, and the glands along the lesser curvature of the stomach are carefully examined for secondary deposits.

In the thorax, the lung is packed away with a moist towel and the layers of the inferior pulmonary ligament opened to expose the lower end of the oesophagus containing the growth. This is mobilised, mostly by blunt dissection, and care is taken not to open the closely related opposite pleura. When several centimetres of healthy tissue above the growth are free, a tape is passed round the gullet and attention transferred to the abdomen. Here it is almost always convenient to remove the spleen first. The greater omentum is ligatured and divided along most of the greater curvature of the stomach. The lesser omentum is detached in the same way. The gastro-epiploic vessels are preserved. The left gastric artery is then doubly ligated as closely as possible to the coeliac axis. The lower end of oesophagus and stomach are now freely mobile and a decision can be made as to the extent of the resection. As a rule this will include the growth with at least 4 or 5 cm. of healthy oesophagus above it, the fundus of the stomach, and about two-thirds of the lesser curve together with the related lymph glands and omentum. Clamps are therefore applied obliquely across the stomach to include these areas and the stomach is divided between them. A swab is tied round the upper clamp, which is turned back into the chest. The lower part of the stomach is closed with two continuous layers of catgut on atraumatic needles. The upper end of the remaining stomach formed by the line of section with the greater curve (Fig. 321) is brought up without tension into the mediastinum to lie behind the healthy upper part of the still intact oesophagus, and is anchored there by a series of fine interrupted linen sutures. A small circular incision is made through the serous coat of the stomach at a convenient point on its anterior surface to match the size of oesophageal lumen. All subserous blood vessels entering the area are

underrun with fine linen sutures which are then tied. This greatly facilitates making the anastomosis. A series of posterior interrupted sutures now approximate the muscle of the back of the oesophagus and the seromuscular coat of the stomach along the posterior edge of the stoma; incisions are then made across the oesophagus and through the gastric



FIG. 321. The distal part of the stomach has been delivered into the thorax and anchored there, and the oesophagus (from which the growth has not yet been resected) brought down on to its anterior surface. A stoma is being made in the stomach wall and the subserous vessels leading into it have been under-run.

mucosa, and a further line of interrupted sutures including both mucosa and muscle layers inserted. The distal part of the oesophagus, including the growth, and the upper part of the stomach, are now removed and the anterior layers of the anastomosis completed in the same way as the posterior layers. A few extra stitches are inserted between the muscle coats of the two organs to reinforce and cover the anastomosis which is also wrapped in omentum, if any is available, and dusted with penicillin. The diaphragm is repaired and stitched to the stomach wall to form a new

restoring the patient's nutrition and electrolyte-fluid balance. Transfusion may be necessary for anæmia.

Growths at the lower end of the œsophagus (i.e. below the level of the inferior pulmonary vein) are best approached by a combined left postero-lateral thoracotomy and laparotomy. The incision is made along the line of the seventh rib, and continued across the upper part of the left rectus muscle and, if need be, downwards in the midline. The latter part of the incision is completed after resection of the ribs and opening the thorax has demonstrated the operability of the growth, or the possibility of performing a short-circuit. The costal cartilage is then divided and the diaphragm opened in radial fashion from the hiatus outwards to the periphery, where the muscles of the anterior abdominal wall and the peritoneum are divided. If more room is required, or if it is decided to perform a radical removal of stomach, spleen and tail of pancreas, this incision can be extended even further down the midline to just above the umbilicus. The operating table is tilted to the left for this part of the operation. The liver, the omentum, and the glands along the lesser curvature of the stomach are carefully examined for secondary deposits.

In the thorax, the lung is packed away with a moist towel and the layers of the inferior pulmonary ligament opened to expose the lower end of the œsophagus containing the growth. This is mobilised, mostly by blunt dissection, and care is taken not to open the closely related opposite pleura. When several centimetres of healthy tissue above the growth are free, a tape is passed round the gullet and attention transferred to the abdomen. Here it is almost always convenient to remove the spleen first. The greater omentum is ligatured and divided along most of the greater curvature of the stomach. The lesser omentum is detached in the same way. The gastro-epiploic vessels are preserved. The left gastric artery is then doubly ligated as closely as possible to the celiac axis. The lower end of œsophagus and stomach are now freely mobile and a decision can be made as to the extent of the resection. As a rule this will include the growth with at least 4 or 5 cm. of healthy œsophagus above it, the fundus of the stomach, and about two-thirds of the lesser curve together with the related lymph glands and omentum. Clamps are therefore applied obliquely across the stomach to include these areas and the stomach is divided between them. A swab is tied round the upper clamp, which is turned back into the chest. The lower part of the stomach is closed with two continuous layers of catgut on atraumatic needles. The upper end of the remaining stomach formed by the line of section with the greater curve (Fig. 321) is brought up without tension into the mediastinum to lie behind the healthy upper part of the still intact œsophagus, and is anchored there by a series of fine interrupted linen sutures. A small circular incision is made through the serous coat of the stomach at a convenient point on its anterior surface to match the size of œsophageal lumen. All subserous blood vessels entering the area are

cervical region) are not amenable to the procedures just described, but, being comparatively superficial, are easily accessible to deep X-rays. As surgical removal usually involves laryngectomy and a complicated plastic repair, the mortality is high and the results disappointing. Equally good results and more satisfactory palliation is achieved by treating them routinely by radiotherapy.

*Post-operative Management.* Drainage tubes can usually be removed after twenty-four or forty-eight hours when radiographs show the lung to be completely expanded. Subsequent effusions are aspirated. During the first forty-eight hours blood replacement, usually comprising 3 or 4 pints, is completed and the patient is then given about 3 litres of 4 per cent. dextrose in N 5 saline a day, to which is added 250 mg. ascorbic acid, 150 mg. nicotinic acid and 5 mg. riboflavin. The quantity of intravenous fluids administered, however, depends on the blood chemistry and clinical state of the patient. The mouth is regularly cleaned with water and glycerine and penicillin lozenges are given. Nothing is swallowed at this stage. However, providing no ileus is present, sips of water are allowed on the third day, and 1 oz. of glucose fruit drinks hourly the next day. On the fifth and sixth days, 2-oz. feeds of peptonised milk with glucose and egg can be given hourly, rising to 4 oz. by the end of the week. Amounts are then increased, but still given two-hourly, until, by the tenth or eleventh day, more varied semi-solid diet is begun with greater intervals between feeds. At the end of two weeks solids are taken, and in another week a normal diet is given.

Throughout the early stages great attention is paid to full expansion and aeration of the lungs, and the patient is made, and assisted, to cough hourly when awake. Atelectasis and accumulating secretions call for prompt bronchoscopy performed under local anaesthesia in the patient's bed. Breathing and leg exercises, taught before operation, are persevered with. Analgesics are given in the first days to prevent pain on exercising or coughing. Antibiotics are continued for about ten days. As many of the patients are elderly and frail, auricular fibrillation is not uncommon and must be controlled promptly.

In the early post-operative phase, paralytic ileus, signalled by distension and absent bowel sounds, must be watched for, and is an indication for further intravenous therapy and continuous aspiration of stomach contents through a Ryle's tube which some surgeons leave *in situ* at the end of the operation. Ileus may be mimicked by pylorospasm which is apt to follow section of both vagi and gives rise to distension, although auscultation confirms the presence of bowel sounds. Pylorospasm is often relieved by a 25 to 50 mg. pastille of Mechothane taken sublingually after each feed. Later the vagotomies may cause troublesome diarrhoea. By far the most serious, and usually fatal, complication of the operation is leakage from the anastomosis. This leads at once to the appearances of air and fluid in the pleural cavity and a marked deterioration in the state



hiatus. The stitches are placed close together to prevent herniation of the bowel into the thorax. The abdomen and chest are then closed. A water-sealed drain is left in the chest after the lung has been fully re-expanded. The layers of the mediastinum should not be sutured, so that any leakage from the anastomosis finds its way into the left pleural cavity and is drained.

Growths in the middle third of the œsophagus are best approached through a right thoracotomy. A combined thoraco-abdominal approach is not possible on the right because of the presence of the liver. This method has been criticised on the grounds that (a) the chest must first be opened to determine operability of the growth itself; (b) the abdomen must then be opened to look for secondaries and to mobilise the stomach; and (c) the chest must be reopened to perform the anastomosis. But if the operation is palliative these arguments lose force. Access to any part of the œsophagus is much easier on the right than on the left where the aortic arch forms a barrier. On the right, only the azygos vein needs to be divided, and the mediastinal pleura opened, to expose the whole length of the œsophagus without impediment. Therefore, for growths other than those at the lower end of the œsophagus (*vide supra*), a separate midline supra-umbilical laparotomy is first performed. A search is made for secondary deposits in the liver and elsewhere. The stomach is mobilised as before right up to the œsophageal hiatus, the margin of which is divided between ligatures to make sufficient room for subsequently drawing up the stomach. The abdomen is then closed and, after a rest of a quarter of an hour or so to minimise shock, the patient is placed in position for a standard right thoracotomy. The level of the rib to be resected depends to some extent upon the position of the growth, but generally speaking removal of the sixth gives good access. The œsophagus, including the growth, is mobilised as before and gentle traction put upon its lower end to deliver the stomach into the right side of the thorax. Some pleural and peritoneal reflections round the hiatus require division, but afterwards the stomach can be brought high into the chest and an anastomosis completed at almost any level. The resection and subsequent junction are carried out in the same way as on the left side, except that as a rule it is not necessary to remove so large a portion of the stomach.

When œsophageal growths are inoperable owing to extensive involvement of adjacent mediastinal tissues, every effort should be made to perform a short circuit to restore swallowing. The part of the gullet mobilised above the neoplasm is brought into apposition with the stomach which is anchored in the mediastinum without resection. A stoma is then created at a convenient position in the stomach wall and the upper divided end of the œsophagus anastomosed to it. The lower cut end of the œsophagus is closed in two layers and invaginated. The growth being inoperable is left undisturbed.

Carcinomas occurring in the superior third of the œsophagus (in the

and as such a tumour is essentially mobile its presence is not felt with the oesophagoscope. If scars of previous ulceration are seen on the mucosa covering the tumour, it is important, in view of the surgical measures indicated, not to perform a biopsy and so impair the integrity of the mucosa.

The treatment of choice is enucleation of the leiomyoma. As the growth is encapsulated, this presents no technical difficulty providing the correct diagnosis has been made. The principal error to be avoided is unnecessary resection of the oesophagus. Every precaution must be taken not to breach the mucosa during the enucleation. The latter is facilitated by the passage of a stomach tube which can readily be felt as a landmark in the oesophageal lumen.

**Pharyngeal Pouches.** These are produced by herniation of mucosa between the circular and oblique muscle fibres of the inferior constrictor of the pharynx and are thought to be due to long standing dysfunction of the cricopharyngeal sphincter (Fig. 322). Their discovery is therefore usually preceded by a long, although perhaps vague, history of dysphagia. During this time the mucous membrane of the pharynx is slowly extruded through its posterior wall. When eventually a pouch has formed it gradually displaces the oesophagus forward and food more readily enters the pouch than the gullet, which therefore receives nothing until the pouch is full. The pouch thus becomes bigger and bigger and more and more dependent, so that food stagnates in it and swallowing becomes increasingly difficult. The patient often finds relief by pressing on the side of the neck, or by tensing the neck muscles, and so emptying the pouch. The patient, who is usually an elderly man, complains of food sticking just above the level of the sternal notch, gurglings and eructations of foul-tasting material, and attacks of choking at night due to overflow of the pouch contents.

A barium swallow confirms the presence of the pouch which usually presents on the left. Any accompanying anaemia (as in the Plummer-Vinson Syndrome) is treated. Oesophagoscopy demands great care, for the instrument tends to enter the pouch and may easily rupture it. If the pouch is large enough to justify surgery, it is first washed out through a soft rubber catheter and penicillin solution instilled into the cavity. Parenteral antibiotics are also given.

**Operation.** The patient is placed in position as for thyroidectomy, and the pouch approached either by a collar incision or by an incision along

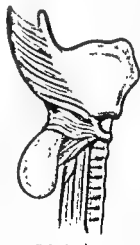


FIG. 322. The mucosal pouch extruded between the fibres of the inferior constrictor. (After Negus.)

of the patient. Aspiration of the fluid reveals œsophageal contents. An empyema soon results. When such a breakdown is not due to faulty stitching, it is more likely to be caused by ischæmia of the stomach than of the œsophagus. If the accident is recognised early and the condition of the patient permits, the best hope of survival lies in reopening the chest and attempting to repair the fistula. If circumstances do not allow this, the only alternative is to cease oral feeding, aspirate the stomach, insert a water-sealed drainage tube into the chest, and endeavour by continuous suction to maintain full expansion of the lung and hope that the leak will heal. The outlook, however, is usually poor.

**Innocent Tumours of the Oesophagus.** Innocent tumours of the œsophagus are rare, though not so rare as once was thought, and over 70 per cent. of them are leiomyomas (tumours of the smooth muscle coat). Growths of the mucosal or submucosal layers project into the lumen as they grow and are therefore drawn further and further from the wall of the œsophagus by peristaltic action until their attachment becomes attenuated into a pedicle which sometimes allows spectacular regurgitation of the tumour into the patient's mouth. Such tumours arise mostly just below the level of the cricoid or in the lowest third of the œsophagus and comprise polypi, papillomas, adenomas, fibromas, epithelial cysts, lipomas and some leiomyomas. As they obstruct the lumen they are most likely to cause dysphagia as a presenting symptom, or they may ulcerate and bleed.

Intramural benign tumours, on the other hand, rarely cause dysphagia, for unlike carcinomas they do not give rise to a stricture even when they almost completely encircle the œsophagus. The mucous membrane over these tumours often ulcerates however, giving rise to obscure but occasionally quite massive hæmorrhages into the alimentary canal, or as their bulk increases they may press upon an adjacent bronchus and cause collapse of a lower lobe of the lung or a pleural effusion. The great majority of these growths are leiomyomas arising in the lowest third of the œsophagus and extending in the plane between the circular and longitudinal muscle coats. Sometimes they appear multicentric or diffuse in form, involving large segments of the œsophagus, but characteristically they are circumscribed even when large. A diffuse tumour arouses suspicion of a leiomyosarcoma.

Radiologically, a leiomyoma may be seen as a lobulated opacity in the posterior mediastinum, moving on deglutition. During a barium swallow the mucosal folds are seen to be ironed out over the tumour, whereas those opposite to it are normal. The flow of barium past the tumour is unimpeded because the other wall of the œsophagus dilates. Sometimes a distinct shelf is formed where the normal œsophageal wall meets the rigid segment involved by growth. Oesophagoscopy is often negative, the instrument passing the growth effortlessly, as does swallowed barium,

and as such a tumour is essentially mobile its presence is not felt with the oesophagoscope. If scars of previous ulceration are seen on the mucosa covering the tumour, it is important, in view of the surgical measures indicated, not to perform a biopsy and so impair the integrity of the mucosa.

The treatment of choice is enucleation of the leiomyoma. As the growth is encapsulated, this presents no technical difficulty providing the correct diagnosis has been made. The principal error to be avoided is unnecessary resection of the oesophagus. Every precaution must be taken not to breach the mucosa during the enucleation. The latter is facilitated by the passage of a stomach tube which can readily be felt as a landmark in the oesophageal lumen.

**Pharyngeal Pouches.** These are produced by herniation of mucosa between the circular and oblique muscle fibres of the inferior constrictor of the pharynx and are thought to be due to long-standing dysfunction of the cricopharyngeal sphincter (Fig. 322). Their discovery is therefore usually preceded by a long, although perhaps vague, history of dysphagia. During this time the mucous membrane of the pharynx is slowly extruded through its posterior wall. When eventually a pouch has formed it gradually displaces the oesophagus forward and food more readily enters the pouch than the gullet, which therefore receives nothing until the pouch is full. The pouch thus becomes bigger and bigger and more and more dependent, so that food stagnates in it and swallowing becomes increasingly difficult. The patient often finds relief by pressing on the side of the neck, or by tensing the neck muscles, and so emptying the pouch. The patient, who is usually an elderly man, complains of food sticking just above the level of the sternal notch, gurglings and eructations of foul-tasting material, and attacks of choking at night due to overflow of the pouch contents.

A barium swallow confirms the presence of the pouch which usually presents on the left. Any accompanying anaemia (as in the Plummer-Vinson Syndrome) is treated. Oesophagoscopy demands great care, for the instrument tends to enter the pouch and may easily rupture it. If the pouch is large enough to justify surgery, it is first washed out through a soft rubber catheter and penicillin solution instilled into the cavity. Parenteral antibiotics are also given.

**Operation.** The patient is placed in position as for thyroidectomy, and the pouch approached either by a collar incision or by an incision along

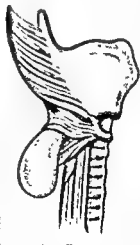


FIG. 322. The mucosal pouch extruded between the fibres of the inferior constrictor. (After Negus.)

the anterior border of the left sternomastoid muscle. The left lobe of the thyroid is exposed, and the middle thyroid veins and, if necessary, the inferior thyroid artery, are doubly ligated and divided. The carotid sheath is retracted laterally, and the left lobe of the thyroid medially, to expose the pharyngeal wall. The fundus of the pouch is easily found if a rubber tube is passed into it beforehand. The pouch is freed by blunt dissection until its neck can be accurately defined. As the pouch consists almost entirely of mucous membrane it must not be handled with toothed forceps. The tube is withdrawn from it and passed down the œsophagus, and the neck of the pouch is divided about half an inch from the œsophagus so that a sufficient cuff of tissue is left to avoid encroaching on the œsophageal lumen during suturing. The use of clamps should be avoided. The divided neck of the sac is closed with fine interrupted catgut sutures on atraumatic needles. The muscle layer is approximated to cover the suture line and a small drainage tube left close to it but not touching it. The wound is then closed. Restricted fluids are allowed after twelve hours and soft solids after a week. Antibiotic cover is continued for ten days.

## THE SURGICAL TREATMENT OF MITRAL STENOSIS

RHEUMATIC heart disease accounts for 25 per cent. of all cardiac invalidism and in a good half of these cases the predominating lesion is stenosis of the mitral orifice. (There are about a quarter of a million people in Great Britain with mitral stenosis.) In comparison, only 2.5 per cent. of cardiac patients have congenital defects such as Fallot's tetralogy. Operative relief of mitral stenosis therefore constitutes one of the major surgical advances of recent years and has opened the way to a whole new field of intracardiac surgery.

Successful cardiac surgery calls for the closest co-operation between the thoracic surgeon and a first-rate department of cardiology. The meretricious glamour with which the subject has unfortunately been imbued, and the apparent simplicity of technique required in easy cases, has tempted many a surgeon, untrained in chest surgery and unsupported by adequate cardiological facilities, to "try his hand," with resulting unacceptable mortality or invalidism.

The selection of suitable patients, as well as correct technique, is of vital importance, and although this is not the place to discuss fully the medical aspects of mitral stenosis, it is necessary to consider in some detail the favourable and the unfavourable signs which provide an answer to the crucial question "Will operation make this patient better or worse?" A stricture at the mitral valve may call for relief like obstruction elsewhere in the body, but the symptoms must result from the stenosis and not from regurgitation, other valvular defects, or myocardial degeneration. Furthermore, some mitral valves are so deformed by disease that valvotomy would produce little or no improvement. Various factors militate against good results but do not necessarily contraindicate surgery. It does not at present seem justifiable to operate on patients with the physical signs of mitral stenosis but who have no symptoms.

Mitral stenosis is about four times commoner in women than in men, and in nearly half the cases there is no precedent history of acute rheumatism. The average period of survival from the first symptoms, which may come on at any time, but are commonest in the early thirties, is eighteen years, and the average age at death forty-seven years. The incidence of auricular fibrillation increases steadily as time goes on, but is not of such grave prognostic significance as the onset of right-sided heart failure with sinus rhythm. About 60 per cent. of patients with the latter complication die within a year. An isolated hæmoptysis is not of serious import, but repeated ones are.

Patients suffering from mitral stenosis usually complain of slowly increasing dyspnoea on exertion, but testimony about this symptom differs widely with the temperament of patients. Women should be asked whether they can do all their housework and, if not, how much of it can they do? How many stairs can be climbed without halting? How far can they walk or run? Men are usually able to compare their ability to work with that of other men of their own age. Most patients have had one or more episodes of right-sided heart failure with peripheral oedema or hepatomegaly. Attacks of dyspnoea are associated with sudden increases of pulmonary venous congestion due to obstruction to the blood flow through the mitral valve. As soon as the pressure in the pulmonary capillaries reaches or exceeds 50 mm. of mercury oedema of the lungs occurs. Such alarming attacks are sometimes precipitated by emotion, exertion or sexual excitement, and are especially likely in pregnancy. They constitute a strong indication for valvotomy. Orthopnoea is a sign of pulmonary congestion and patients with mitral stenosis are often found to sleep comfortably only if they are propped up with pillows. They may wake with a sense of suffocation should they slip down from them. Although haemoptysis may be due to embolism, it is usually a sign of pulmonary venous hypertension and is therefore common in pregnancy, after which it may not recur. Repeated haemoptysis suggests serious and increasing pulmonary hypertension and calls urgently for surgical relief. "Tightness" across the chest and even retrosternal pain (to be distinguished from the pain of angina) are common. There may be a cough and attacks of "bronchitis," some of which are due to congestion, some to intercurrent infection. Embolic phenomena are unusual if the heart is in normal rhythm but common with auricular fibrillation. Cerebral embolism is so grave a catastrophe that the need for operative prevention must override other unfavourable factors. Frequently patients complain of coldness of the extremities, which may be cyanosed as a result of vasoconstriction and stagnation of blood.

On examination the pulse is characteristically small in volume due to decreased cardiac output. The apex beat has a slapping quality, like the blow of an open hand as compared with the blow of a clenched fist in left ventricular hypertrophy. A diastolic thrill is commonly felt. Fibrillation is present in about 40 per cent. of patients submitted for surgery. In some patients the fibrillation is intermittent, but almost invariably signals the imminent onset of permanent irregularity. The first heart sound at the apex is characteristically loud and snapping in quality. If this is not so it is doubtful if stenosis is the predominating lesion. Loudness and crispness of the first sound is a favourable sign, for it suggests that pliant valve cusps are coming smartly together. The classical rumbling presystolic and mid-diastolic murmurs of mitral stenosis are present. With the onset of fibrillation the presystolic murmur disappears. Absence of a diastolic murmur, or a faint one, suggests much pu

or very tight stenosis. Midway between the apex and the tricuspid area the so-called "opening" snap is audible immediately after the second heart sound, and when well marked is confirmatory evidence of predominant stenosis. Absence or diminution of this sound suggests either that the cusps are calcified or that a good deal of incompetence is present. Increase in intensity of the pulmonary second sound is generally indicative of pulmonary hypertension, but its quality varies much in the same individual with exercise or emotion: it is normally split, but if the splitting is very close, or absent altogether, pulmonary pressure is probably high.

Skilled radiological screening is indispensable. In general the lung fields show some congestion, with increase in the vascular markings, and occasionally evidence of infarction or of hemosiderosis. The heart at first appears generally enlarged, but it is important to tell if possible whether it is the right or left ventricle which is enlarged. Enlargement of the left ventricle usually indicates predominant regurgitation which contra-indicates surgery. The more normal the size of the left ventricle the better, generally speaking, is the outlook for surgery. The antero-posterior view of the left cardiac outline alone provides valuable information. Beginning at the top, the aortic knuckle is inconspicuous, either because it is actually smaller than usual (if the disease has been present from childhood), or because it is overshadowed by enlargement of the pulmonary artery just below it—a fairly constant sign reflecting, though not with accuracy, the degree of hypertension present. Below this again is the prominence of the left auricular appendix, the absence or diminution of which provides a warning of possible difficulty in surgical access to the atrium (*vide infra*). The left profile ends in the normally sized apex of the left ventricle. Both the left atrium and the right ventricle may be enlarged to a variable extent. They are studied best with patients in the right and left anterior oblique positions respectively. In particular it is necessary to look carefully at the left atrium for systolic expansion which suggests a considerable degree of mitral regurgitation. Calcification of the mitral cusps may be clearly visible on direct screening, but is more likely to be revealed by tomography.

Electrocardiography may show right ventricular preponderance in mitral stenosis, but the presence of left ventricular preponderance indicates predominant regurgitation or aortic disease and is a strong contraindication to surgery. Cardiac catheterisation is quite unnecessary in the great majority of patients except for the purpose of academic studies, and it is doubtful if the decision with regard to surgery should ever rest upon the evidence so provided. The best criteria are the clinical ones.

Whereas all stenotic mitral valves permit *some* regurgitation, valvotomy by restoring a great measure of valvular function, often diminishes its degree in selected cases. It is essential, however, to recognise when regurgitation predominates over obstruction, for then operation seldom



Patients suffering from mitral stenosis usually complain of slowly increasing dyspnoea on exertion, but testimony about this symptom differs widely with the temperament of patients. Women should be asked whether they can do all their housework and, if not, how much of it can they do? How many stairs can be climbed without halting? How far can they walk or run? Men are usually able to compare their ability to work with that of other men of their own age. Most patients have had one or more episodes of right-sided heart failure with peripheral oedema or hepatomegaly. Attacks of dyspnoea are associated with sudden increases of pulmonary venous congestion due to obstruction to the blood flow through the mitral valve. As soon as the pressure in the pulmonary capillaries reaches or exceeds 50 mm. of mercury oedema of the lungs occurs. Such alarming attacks are sometimes precipitated by emotion, exertion or sexual excitement, and are especially likely in pregnancy. They constitute a strong indication for valvotomy. Orthopnoea is a sign of pulmonary congestion and patients with mitral stenosis are often found to sleep comfortably only if they are propped up with pillows. They may wake with a sense of suffocation should they slip down from them. Although haemoptysis may be due to embolism, it is usually a sign of pulmonary venous hypertension and is therefore common in pregnancy, after which it may not recur. Repeated haemoptysis suggests serious and increasing pulmonary hypertension and calls urgently for surgical relief. "Tightness" across the chest and even retrosternal pain (to be distinguished from the pain of angina) are common. There may be a cough and attacks of "bronchitis," some of which are due to congestion, some to intercurrent infection. Embolic phenomena are unusual if the heart is in normal rhythm but common with auricular fibrillation. Cerebral embolism is so grave a catastrophe that the need for operative prevention must override other unfavourable factors. Frequently patients complain of coldness of the extremities, which may be cyanosed as a result of vasoconstriction and stagnation of blood.

On examination the pulse is characteristically small in volume due to decreased cardiac output. The apex beat has a slapping quality, like the blow of an open hand as compared with the blow of a clenched fist in left ventricular hypertrophy. A diastolic thrill is commonly felt. Fibrillation is present in about 40 per cent. of patients submitted for surgery. In some patients the fibrillation is intermittent, but almost invariably signals the imminent onset of permanent irregularity. The first heart sound at the apex is characteristically loud and snapping in quality. If this is not so it is doubtful if stenosis is the predominating lesion. Loudness and crispness of the first sound is a favourable sign, for it suggests that pliant valve cusps are coming smartly together. The classical rumbling presystolic and mid-diastolic murmurs of mitral stenosis are present. With the onset of fibrillation the presystolic murmur disappears. Absence of a diastolic murmur, or a faint one, suggests much pulmonary hypertension

The best results are obtained in patients with normal cardiac rhythm, between the ages of twenty-five and forty years, with slight or no cardiac enlargement, in whom the only detectable valvular lesion is a tight mitral stenosis, and in whom the valves remain pliable and membranous. The physical signs suggesting these ideals are a small volume pulse, a clapping apex beat, a normal sized left ventricle, a loud snapping first heart sound at the apex, and a crisp clear "opening snap."

Indications for early surgery are increasing dyspnoea, attacks of right-sided heart failure, pyrexio-mal pulmonary oedema, frequent haemoptysis, and emboli—even if some other factors are unfavourable. As pregnancy is particularly likely to cause pyrexio-mal attacks of dyspnoea, and the alternative may be termination of pregnancy and sterilisation, valvotomy should be done, if possible before the sixth month.

The mitral valve consists of two cusps, a larger and more important anteromedial one, and another smaller posterolateral one. They fuse together when inflamed, along a line which runs roughly from ten o'clock anteriorly to five o'clock posteriorly when the valve is viewed from the atrium. After fusion, the anterior commissure is divided from the posterior only by a stenotic orifice of variable size left in the middle between them. This orifice is usually 0.5 to 1 cm. in diameter according to the severity of the stenosis. The disease process is not, however, confined to the valve cusps themselves. Inserted on the undersurfaces of their edges, chiefly at the junction of their middle and outer thirds, are the chordae tendineae, originating on the papillary muscles which rise like stalagmites from the ventricular floor. All these structures may undergo scarring and contraction, and become matted to each other or to the myocardium. Fusion of the cusps first occurs where the chords are inserted. If the chords are not severely affected but the cusps are fused the valve will retain the character of a diaphragm. It is this type which is easiest to split completely with the finger and which is most amenable to surgery, for there is a better chance of restoring the function of the valve. But if the chords are much contracted, or even destroyed so that the papillary muscles are virtually fused directly to the cusps, the whole valve is drawn down into the ventricle as a cone of sclerotic tissue. The orifice is at the apex of the cone, and the valve cusps are no longer at right angles to the myocardium but closely contiguous and almost parallel to it (Fig. 323). The difficulty of the operation, and the prospects of its success, worsen with the degree of this deformity, but it is only by direct palpation that its extent can be estimated.

Preoperatively time is devoted to getting the patient into the best possible state for surgery. Cardiac catheterisation is not performed as a routine. Whether in sinus rhythm or fibrillation, most patients are digitalised, although no attempt is made to restore normal rhythm with quinidine. If there is any degree of heart failure, medical treatment, including if necessary mercurial diuretics and limitation of salt intake, is

does good and may do harm. The presence of an apical systolic murmur, with or without a systolic thrill, though it rouses strong suspicion of mitral incompetence, is not conclusive. Therefore other evidence must be sought, such as clinical or radiological signs of left ventricular enlargement, or left ventricular preponderance on the electrocardiograph. Whether these signs are due to mitral regurgitation or aortic valve disease, they forbid surgery. The systolic murmur of mitral incompetence is high pitched and blowing, and the more pronounced it is the more severe is the retrograde leak likely to be. On the other hand, the absence of such an apical murmur does not exclude a significant reflux. Calcification in the commissures abolishes or greatly diminishes the "opening snap," but if no calcification is present reduction in the intensity or absence of the first heart sound, or absence of the "opening snap" makes predominance of regurgitation over stenosis likely. Great enlargement of the left atrium or its expansion during systole should be similarly interpreted.

It is clearly undesirable to operate on a heart when rheumatism is still active. Therefore surgery is seldom advised under the age of twenty-five years, though if symptoms of stenosis are sufficiently severe it may have to be undertaken at an earlier age. At the other extreme, long-standing valvular disease is likely to have produced so much myocardial damage that operation would be too late for a satisfactory result. Therefore fifty years is regarded as the upper age limit for operation. Although there is evidence that long-standing pulmonary hypertension tends to produce irreversible changes, and results of surgery may then be disappointing, this cannot be foreseen, and the presence of raised pulmonary artery pressure is usually accepted as an indication for surgery.

To summarise briefly, therefore, the contraindications to mitral valvotomy are: (1) No disability; (2) clinical evidence of active rheumatism; (3) age over fifty years; (4) predominant mitral regurgitation or aortic valvular disease.

Although auricular fibrillation presages myocardial damage, and therefore a less satisfactory result, it is not a contraindication to surgery, especially as it is often associated with emboli. Normal rhythm is very seldom restored after successful valvotomy, and indeed a number of patients previously in sinus rhythm fibrillate after operation, though in many of these normal rhythm is restored by quinidine. Calcification of the cusps may also adversely affect the results of operation, both because of the increased scarring and therefore the difficulty in separation of the cusps likely to be found, and also because of added risk of embolic fragments being dislodged by commissurotomy. Often, however, calcified valves retain their diaphragmatic shape and can be separated easily, with excellent results. Sometimes some of the worst valves, with gross deformity and much cicatrisation, are not calcified and yet they are inoperable.

of the pericardium. The anterior pericardial flap is picked up with two sutures which are held in forceps to retract the pericardium away from the surgeon.

An examination of the heart is now conducted, but with a minimum of manipulation. The rhythm and force of the beat is observed together

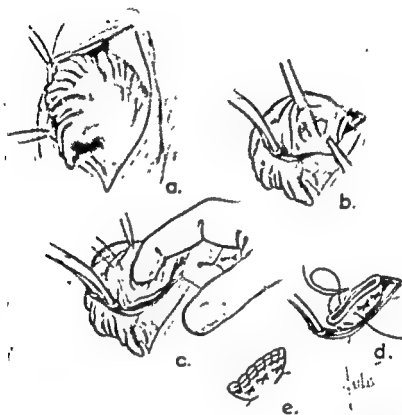


FIG. 324. (a) The auricular appendage exposed. (b) The appendage is opened after its base is clamped. Obstructing muscle bands are cut. (c) After stay sutures have been inserted the index finger is introduced as the clamp is removed. (d) The appendage is amputated and closed with mattress sutures. (e) Closure of the stump is completed with a continuous stitch.

with any systolic expansion of the atrium. The size of the pulmonary vessels is recorded. At the apex the characteristic diastolic thrill is felt. A systolic thrill on the atrium suggests significant regurgitation. With the left thumb in the transverse sinus and the index on the front of the aorta, the surgeon palpates the region of the aortic valve and forms an opinion of its condition, noting any thrill or post-stenotic dilatation. If necessary direct blood pressure recordings are taken from the aorta and from the left ventricle.

continued so long as it produces improvement. The patient's fluid intake and output are charted throughout. Anæmia if present is corrected. A few days before the operation penicillin is begun, and is continued subsequently for a week or ten days.

**The Operation.** The patient lies in the usual position for a left postero-lateral thoracotomy, but a chest rest is not employed. The line of the incision is carried well anteriorly in the line of the fifth rib. It is infiltrated with local anæsthetic and a paravertebral block is carried out. One-third per cent. Xylocain is a suitable solution, with 1 ml. of 1 : 1,000 adrenaline



FIG. 323. In the specimen the deformed mitral valve is drawn down into the left ventricle by the greatly thickened and shortened papillary muscle. Note how one valve cusp has come to lie almost in apposition with the ventricular wall.

added just before use. The chest is entered through the bed of the fifth rib, though the rib itself is not resected (the rib bed above or below will not do). The texture of the lung is palpated and an attempt made to grade the consistency of induration. If required, a biopsy can be taken from the lingula process for this purpose. The lung is then displaced posteriorly and covered with a wet warm pack, held in place by a malleable lung retractor. The pericardium is thus exposed. The operating table is now tipped laterally towards the surgeon.

The pericardium is picked up, incised posterior to the phrenic nerve, and opened widely from the pulmonary artery above to a point below the inferior pulmonary vein so that wide access is obtained to the side of the atrium and its appendage (Fig. 324a). The phrenic nerve must not be injured or interfered with during either the opening or subsequent closure

of the pericardium. The anterior pericardial flap is picked up with two sutures which are held in forceps to retract the pericardium away from the surgeon.

An examination of the heart is now conducted, but with a minimum of manipulation. The rhythm and force of the beat is observed together

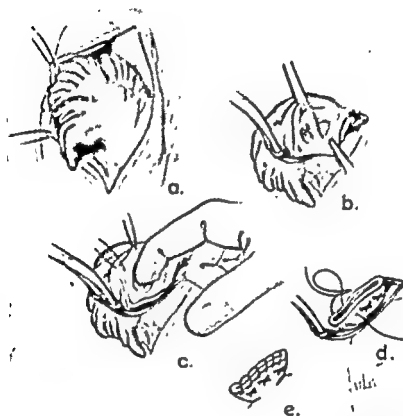


FIG. 324. (a) The auricular appendage exposed. (b) The appendage is opened after its base is clamped. Obstructing muscle bands are cut. (c) After stay sutures have been inserted the index finger is introduced as the clamp is removed. (d) The appendage is amputated and closed with mattress sutures. (e) Closure of the stump is completed with a continuous stitch.

with any systolic expansion of the atrium. The size of the pulmonary vessels is recorded. At the apex the characteristic diastolic thrill is felt. A systolic thrill on the atrium suggests significant regurgitation. With the left thumb in the transverse sinus and the index on the front of the aorta, the surgeon palpates the region of the aortic valve and forms an opinion of its condition, noting any thrill or post-stenotic dilatation. If necessary direct blood pressure recordings are taken from the aorta and from the left ventricle.

The atrial appendage (syn. auricle of the left atrium) affords the easiest site for access to the mitral valve. It varies greatly in size and shape and may be too small and narrow for use. Sometimes it is vestigial. In the great majority of cases, however, it is of suitable dimensions. The appendage must be gently felt for the presence of clot, which if found may later be evacuated through the incision in its wall. Usually a special fabric-covered clamp is applied to the base of the appendage (Fig. 324). Great care is taken to avoid the coronary vessels lying in close proximity medially. It is quite unnecessary, and indeed dangerous, to insert any kind of purse-string suture round the base of the appendage.

An incision just long enough to accommodate the surgeon's index finger is now made at a convenient point on the lateral wall of the atrial appendage and a stay suture inserted at each end of the upper leaf to give control to the assistant during application and removal of the clamp (Fig. 324). If too big an opening is made bleeding from the heart occurs and prevents proper deliberation. The interior is inspected for clot and any present evacuated. The clamp is taken off for a moment to allow a jet of blood to wash fragments of clots from the atrium. If the latter contains clot which cannot be washed out, the situation is dangerous, and the safest approach to the valve is then probably through the lumen of the superior pulmonary vein. A clamp is applied where this vessel enters the atrium, and its branches from the lung are controlled with tapes. The practice of temporarily occluding the carotid arteries, either by the anesthetist pressing them in the neck, or by clamping them after they have been dissected out in the mediastinum, to avoid possible cerebral emboli when clot is known to be present in the atrium, or the valve densely calcified, is debatable; experience has shown that it is more dangerous to do this, from the point of view of cerebral damage, than to accept the evident risk of emboli, for the resulting anoxia may produce the very brain damage which it is hoped to avoid. The incidence of damage to the brain is higher when such precautions are taken than when they are not.

If the appendage is too small to admit the index, it may be possible to perform the valvotomy with the little finger. Sometimes there is a constricting ring at the junction of the appendage with the atrium which has to be gently dilated. If neither finger can be introduced, or the appendage is wanting altogether, or is obstructed by organised clot, the approach to the valve is made directly through the atrial wall itself. In these circumstances, the area between the appendage and the superior pulmonary vein is perhaps the best. No. 40 linen thread is used to make an oval purse-string long enough to enclose the index finger. It is essential to take good deep bites of the myocardium, for otherwise the stitches may tear out. Their two ends are threaded into a Rumel snare which is held on the side away from the surgeon by his assistant. A cut is made in the muscle within the purse-string, and in its long axis, almost

down to the endocardium, the final rupture of which is made with the finger itself (Fig. 325). As the finger enters the atrium, the assistant exerts very gentle tension on the snare so that the finger is snugly held and no blood is lost. As the index is ultimately withdrawn the purse-string is pulled tight, and final closure of the myocardium made with a few deep interrupted sutures.

Whether the heart is entered via the appendage, or directly as just described, no unwanted bleeding should occur. When the appendage is used, as in the great majority of cases, the surgeon releases the clamp with his left hand as the right index passes into the lumen. When the finger approaches the mitral valve some regurgitant jet or flow can usually be felt, and its nature and extent must be carefully judged both before and after commissurotomy (valvotomy). Subsequent apparent diminution may be due only to a temporary fall in blood pressure. The valve itself is palpated, the size of the orifice is measured; calcification is felt for, and in particular the flexibility of the leaflets (as demonstrated by the ballooning of the anteromedial cusp on systole) is judged.

When the examination has been completed the finger tip is passed into the orifice, and the posterior commissure is split with the ulnar edge of the finger. If this proves easy, the finger is swept forward to complete the separation of the anterior commissure out to the valve ring. Such a process is only possible with a lightly adherent diaphragmatic type of valve, and considerable (but not excessive) force may be needed to effect the separation. The finger must not remain too long in the mitral orifice as it completely obstructs blood flow while it is there. Therefore if repeated efforts are needed, there must be short periods of rest during which the finger is withdrawn from the mitral orifice. It is better to attack the posterior commissure first while it is given some fixity by the rest of the adherent valve. The anterior commissure is split with the right index inside the heart while counterpressure is applied by the left palm on the ventricular wall outside, but posteriorly no such helping support can be given. When separation

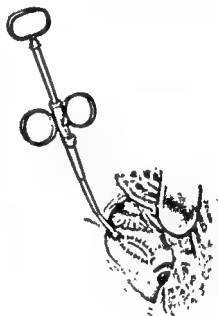


FIG. 325. As the appendage is vestigial a purse-string suture has been inserted between it and the origin of the superior pulmonary vein, and secured by a Rumel snare. The incision does not go all the way through the atrial wall.



is complete, the ventricular wall should be palpable at the end of the fissure. The finger is passed down through the orifice to separate adherent chordæ tendineæ and to restore their mobility and that of any of the papillary muscles which have become adherent to the myocardium. At the same time, the tip of the finger can sometimes be brought up on the ventricular aspect of the valve to complete an unsatisfactory division, and this is particularly important in cases in which the valve is retracted and

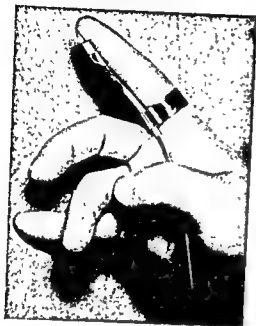


FIG. 326. The Brock probe-pointed type of valvulotome.

cone-like, for then the whole outer third of the cusp lies close against the ventricular wall. This portion may be difficult to distinguish, and more difficult to split, but it is vital that it should be dealt with, for upon its freedom the restoration of valvular function largely depends. Merely to enlarge the central orifice without carrying out this manoeuvre and the subvalvular exploration is to invite re-adherence of the cusps. At all times great care is taken not to damage the chords or rupture a cusp, for severe regurgitation will certainly ensue and may prove fatal. A particularly susceptible area for the latter accident is on the medial part of the posterior commissure where no chordæ are inserted. Therefore this commissure ought not as a rule to be split further

than about 1 cm. if it is not readily separable.

Although complete separation of the cusps may be accomplished with the finger alone, in many cases this cannot be done, for either the edge of the central orifice proves too tough to split, or separation occurs only as far out as the point of insertion of the thicker chords, and does not include the peripheral part of the commissure, which, in retracted conical valves, is closely in contact with the ventricular wall. In all such cases—and it is notable that their number increases with the experience of the surgeon—a valvulotome must be employed. Many varieties exist. Some have the form of guillotines. Some are in the form of a blade directly mounted on a ring. The one devised by Brock is generally satisfactory: it consists of a flexible strip held to the index by a slotted finger ring so that it can be advanced or withdrawn as required to allow further palpation or digital splitting. At its end there is a small probe-pointed blade (Fig. 326). The probe is engaged in the mitral orifice, and a little pressure on the blade cuts the commissure. It can also be hooked under the deep edge of a much retracted valve, and the portion lying parallel to the ventricular

wall fixed by drawing the knife edge upwards. The knife is not a substitute for the finger; it is complementary, and in most cases both are necessary. They are used alternatively until maximum separation and mobility of the cusps has been attained.

At the end of the operation the valve orifice should be capable of accommodating two fingers. As the index is finally withdrawn from the heart the clamp is slipped back into place on the base of the appendage, but before this is done it is well to be sure no accidental tearing of the appendage, or of the atrial wall, has occurred, for bleeding from a tear of this kind can be much more effectively controlled if sutures are inserted while a finger remains inside than after it is withdrawn. When the clamp is replaced the part of the appendage distal to it is cut away, except for a cuff of tissue which is closed by a few mattress sutures and a final continuous stitch along the cut edge. No. 40 linen thread is suitable for both (Fig. 324d and e). The amputated appendage is sent to be examined for histological evidence of active rheumatism. The clamp is now removed. If any bleeding still occurs from the stump of the appendage an additional mattress suture or two will control it. The pericardial sac is thoroughly irrigated with warm saline to wash out blood clot which may have collected in it, and only when the stream returns quite clear is the pericardium closed. A continuous fine linen stitch approximates the incision in the pericardium, but is not continued the whole way, a gap being left large enough to allow any effusion to drain into the pleural cavity, but not so large that there is risk of the apex of the heart dislocating through it and strangulating when the patient lies on his side. Care is taken not to pick up the phrenic nerve in the running stitch, and not to handle it with dissecting forceps. A water-sealed drain is placed dependently in the pleural cavity, the lung is fully re-expanded, and the chest closed in the usual manner.

The patient's limbs should be examined before he leaves the table to make sure that no arterial obstruction has been caused by an embolus, for it would require immediate operation. As soon as consciousness returns it is reassuring to hear the patient speak, move his arms and legs, and to see that he has no abnormal neurological signs indicating a cerebral embolus. The drainage tube may be withdrawn (usually after twenty-four hours) when radiography shows the lung to be fully expanded. Any subsequent pleural effusion is aspirated, although this is seldom necessary. If atelectasis occurs it is treated by prompt bronchoscopy. The onset of auricular fibrillation is not uncommon in the first two or three post-operative days and sometimes proves irreversible. Quinidine should always be tried, and is often effective in restoring normal rhythm, though not in those cases in which fibrillation existed before operation. Quinidine should not, however, be employed before a fortnight has elapsed and the heart has had time to settle down, for it is much less successful during the immediate post-operative period.

Pericardial effusion may give rise to a mild degree of cardiac tamponade, with breathlessness, cervical venous distension, and radiographic evidence of a distended pericardial sac. If symptoms are severe it may be necessary to aspirate. This is best performed with a fairly wide-bore needle, directed upwards, backwards, and slightly medially, in the angle formed by the xiphisternum and the left costal margin. The diaphragm and the pericardium above are pierced at a point well away from coronary vessels, and where injury to the heart is unlikely. This complication is rare, for adequate drainage holes are usually left in the pericardium. Similar symptoms are produced by the "post-valvotomy syndrome" which is now well recognised by most cardiac surgeons although its cause remains obscure. It affects about one in three patients and as a rule comes on towards the end of the first week or during the course of the second; sometimes it is longer delayed. There is often a rise of temperature and pulse rate accompanied by malaise and some breathlessness. A pericardial friction rub is usually audible at the onset, followed by a mild degree of cardiac enlargement. There is a rise in cervical venous pressure. The patient is kept at rest until the symptoms and venous distension subside spontaneously—usually a week to ten days. It is probable that the syndrome is post-traumatic in origin rather than due to reactivation of rheumatism, for the latter would hardly resolve so rapidly without specific treatment. On the other hand, it is certainly true that histological evidence of rheumatic activity is found in 30 to 40 per cent. of the amputated appendages.

If no complications occur and fibrillation, if present, is adequately controlled, patients can usually begin to get out of bed in the second week and start graduated activity. The extent of this progress is dependent largely on the length of time spent in bed before operation. Most patients at once comment on their warmer hands and feet; on the removal of a sense of oppression and constriction across the lower part of the chest; and on their ability to breathe more comfortably and to take deep satisfying inspirations. As they become ambulant, their increased exercise tolerance soon becomes apparent, but improvement continues over many months and a true assessment of the benefit brought by surgery should not be made until six months have elapsed.

## CHAPTER 46

### CONSTRICTIVE PERICARDITIS

In tuberculous pericarditis the resultant effusion is absorbed, leaving caseous material within the pericardium, the layers of which become adherent to each other and to the heart. As healing proceeds, the pericardium becomes greatly thickened and fibrotic, slowly contracting and compressing the heart so that not only are the ventricles unable to contract and shorten as they must do in systole, but they are also unable to expand and lengthen in diastole when they should receive blood from the auricles. The heart's intake as well as its output are thus diminished. The pulse is therefore of poor volume and may decrease on inspiration. The blood pressure is low. The vascular markings of the lungs, as seen in X-rays, are diminished. The cardiac output, normally some 4 litres a minute, is likely to be reduced in constrictive pericarditis and cannot be increased, so that although the patient is usually not breathless at rest, dyspnoea occurs on exertion. The velocity of the blood flow is also decreased (the normal arm-to-tongue time of sixteen to twenty seconds being increased to forty or even sixty seconds).

Because of the diminished intake of blood into the heart, the venous return is progressively dammed up, so that there is a rising venous pressure causing distension of the veins of the neck, marked enlargement of the liver, ascites, pleural effusion and later peripheral oedema—roughly in that order. Cyanosis is common but not constant. The cervical veins pulsate (they do not do so when distension is caused by mediastinal obstruction). The venous pressure is often found to be as high as 15 or 20 mm. of mercury. The blood volume becomes increased by 30 to 40 per cent. Usually the hepatic engorgement and ascites are disproportionately greater than the oedema of the legs and ankles. No moist sounds are to be heard at the pulmonary bases, and the heart is usually small and quiet—a picture quite unlike that of congestive failure. Furthermore, the heart sounds are normal, although a "third heart sound" may be distinguished immediately after the normal second. Partly because of frequent delay in diagnosis, auricular fibrillation is present in about a quarter of all cases, and tends to be associated with some degree of myocardial atrophy.

Radiographically the heart is usually small, especially in relation to the symptoms, but this is not always so, for the pericardial thickening and even residual caseous material in the sac may give a bigger outline than normal. The most striking sign of all, however, is the relative lack of pulsation demonstrated by screening and kymography, though this

sign varies in different parts of the heart, tending to be most conspicuous over the apex and the left ventricle. In long-standing cases, calcification of the pericardium is common and may completely ensheath the heart, or lie mostly in the dependent diaphragmatic part of the pericardium like a cup (Fig. 327). Its presence greatly simplifies diagnosis; it is found in a quarter to a third of cases. The electrocardiogram is consistently of low



FIG. 327. Densely calcified pericardium in a case of constrictive pericarditis.

voltage in all leads and the T waves are flattened or inverted. The amount of change in the T waves provides some measure of the myocardial involvement.

Some patients may already be known to suffer from pulmonary tuberculosis, or have radiological evidence of it. Sometimes there is a history of exposure to infection; otherwise the evolution of the disease may be followed from the onset of the tuberculous pericarditis, to the enlargement of the pericardial sac, with serous and later semi-purulent effusions, and eventually to the final stage of cardiac constriction. Surgery should not be undertaken during the active phase of tuberculous

infection, but it should not be delayed until hepatic damage has developed. Always, except when dense calcification has occurred, a full course of streptomycin and para-aminosalicylic acid (PAS) is given before operation. Paracentesis abdominis is often required, and the fluid intake is limited to 1,500 ml., and salt intake to about 2 gm. a day. If the plasma protein levels are low, a high protein diet is called for. Mercurial diuretics are given to help raise fluid output. Treatment is adjusted according to a fluid intake-output chart. Digitalis is usually contraindicated because it tends still further to diminish cardiac output in these cases, but when auricular fibrillation is a serious factor digitalization is essential. Secondary anemia requires correction, but blood transfusion is avoided if possible.

Surgery should be carried out without undue delay once the patient's condition is judged to be suitable. If there is clinical evidence of active tuberculosis, at least two month's preoperative antibiotic therapy is needed and the sedimentation rate should be declining.

**Pericardectomy.** The object of this operation is to free both ventricles as completely as possible from their coat of constricting fibrous tissue. Although controversy exists on the subject, it is unnecessary as well as dangerous to make any attempt to clear the auricles or the great vessels. It has been shown on many occasions—both experimentally and clinically, that the great vessels are capable of much greater narrowing than ever occurs in constrictive pericarditis without ill effect. Failure or unsatisfactory results following pericardectomy are due either to insufficient clearance of the ventricles, especially at the apex, or, in late cases, to secondary myocardial degeneration. The ventricles should be completely cleared of fibrous tissue as far as the auriculo-ventricular groove.

The heart is best approached for pericardectomy by an anterolateral incision, with subperiosteal resection of the anterior half of the left fifth rib and its cartilage up to the edge of the sternum. The pleura is almost always found densely adherent to the pericardium; no attempt should be made to perform the operation extrapleurally. When it has been freed the lung is packed away from the heart and the phrenic nerve identified as it courses down the lateral aspect of the pericardium. Throughout the operation every care must be taken not to injure this nerve. Thus bleeding from branches of the phrenic vessels are dealt with by ligation and not by coagulation.

When the pleura has been cleared from the pericardium the parietal layer of the latter is incised with a knife until the plane of cleavage between it and the visceral layer is reached. This represents what remains of the pericardial cavity, and in early cases often contains caseous material which is removed for examination for the presence of tubercle bacilli. The plane of cleavage is developed partly by blunt and partly by sharp dissection, until a satisfactory flap has been raised on both sides. The edges of the flaps are useful for subsequent retraction. At this stage the

ventricles still appear relatively immobile, for the true constricting coat is the visceral pericardium which must now be incised down to the heart muscle at a convenient point over the right ventricle.

When this new plane has been established its dissection (much less easy than dissection of the first) is carried out, in postage stamp amounts, over the surface of the left ventricle, which is freed first, for pulmonary congestion might ensue if the right ventricle were released first. As each patch of visceral pericardium is raised, the heart muscle bulges outward as it escapes from the strait-jacket. If this does not happen, the correct plane of dissection has not been reached. Running down the interventricular sulcus is the descending branch of the left coronary artery, and great care must be taken not to damage it or any of its branches. If the artery is closely involved in fibrous tissue, the tissue over it should be left alone and a new start made on its left side. Inadvertent injury and bleeding from this vessel calls for the application of a muscle patch and gentle pressure. It must never be under-run with sutures. Bleeding from elsewhere on the myocardium is slight, and soon stops, but there may be a brisk ooze from the cut pericardial edges. Really severe bleeding results from perforation of the heart. It is much more likely to follow encroachment on the auricles than to come from the ventricles. Such hæmorrhage is controlled by digital pressure until a few deep silk sutures can be passed through the heart and a flap of the pericardium sewn in place over the wound.

The dissection is carried round the left border of the ventricle and down over the apex. It is most important to free the apex, for otherwise the heart cannot shorten on its long axis during systole. The already raised pericardial flaps are used as retractors, and as much as possible of the diaphragmatic and posterior surfaces of the heart are liberated. Finally, attention is returned to the surface of the right ventricle, which is similarly treated. The dissection of both ventricles always stops just below the A-V groove. Cardiac irritability is tempered throughout by intravenous procaine given in the anæsthetist's drip—not applied locally. As a rule it is manifested by extrasystoles, and if these are persistent a short rest is indicated until normal rhythm returns. In the past there has been a tendency to desist from further clearance if there is much bulging of the myocardium, and to complete the operation at a later stage for fear of the occurrence of cardiac dilatation. There seems little justification for this policy in young patients, but in long-standing cases in which myocardial atrophy is suspected, and fibrillation present, it is probably a wise precaution.

When clearance is complete, particular care is taken to stop all bleeding from the cut pericardial edges and to see that the lung is completely re-expanded. The chest is closed as usual and a water-sealed drain left in the pleural cavity for twenty-four hours to remove any subsequent effusion.

The first sign of recovery is usually a sharp rise in urinary output, but this is often delayed for several days, or even some weeks, during which time the patient remains in bed. Once it begins, subjective improvement is rapid, but it may take some months for the ascites to disappear, for the liver edge to recede, and for the high venous pressure to subside. Failure to improve is almost always due to inadequate decortication of the ventricles and calls for further intervention.



ventricles still appear relatively immobile, for the true constricting coat is the visceral pericardium which must now be incised down to the heart muscle at a convenient point over the right ventricle.

When this new plane has been established its dissection (much less easy than dissection of the first) is carried out, in postage stamp amounts, over the surface of the left ventricle, which is freed first, for pulmonary congestion might ensue if the right ventricle were released first. As each patch of visceral pericardium is raised, the heart muscle bulges outward as it escapes from the strait-jacket. If this does not happen, the correct plane of dissection has not been reached. Running down the interventricular sulcus is the descending branch of the left coronary artery, and great care must be taken not to damage it or any of its branches. If the artery is closely involved in fibrous tissue, the tissue over it should be left alone and a new start made on its left side. Inadvertent injury and bleeding from this vessel calls for the application of a muscle patch and gentle pressure. It must never be under-run with sutures. Bleeding from elsewhere on the myocardium is slight, and soon stops, but there may be a brisk ooze from the cut pericardial edges. Really severe bleeding results from perforation of the heart. It is much more likely to follow encroachment on the auricles than to come from the ventricles. Such hæmorrhage is controlled by digital pressure until a few deep silk sutures can be passed through the heart and a flap of the pericardium sewn in place over the wound.

The dissection is carried round the left border of the ventricle and down over the apex. It is most important to free the apex, for otherwise the heart cannot shorten on its long axis during systole. The already raised pericardial flaps are used as retractors, and as much as possible of the diaphragmatic and posterior surfaces of the heart are liberated. Finally, attention is returned to the surface of the right ventricle, which is similarly treated. The dissection of both ventricles always stops just below the A-V groove. Cardiac irritability is tempered throughout by intravenous procaine given in the anæsthetist's drip—not applied locally. As a rule it is manifested by extrasystoles, and if these are persistent a short rest is indicated until normal rhythm returns. In the past there has been a tendency to desist from further clearance if there is much bulging of the myocardium, and to complete the operation at a later stage for fear of the occurrence of cardiac dilatation. There seems little justification for this policy in young patients, but in long-standing cases in which myocardial atrophy is suspected, and fibrillation present, it is probably a wise precaution.

When clearance is complete, particular care is taken to stop all bleeding from the cut pericardial edges and to see that the lung is completely re-expanded. The chest is closed as usual and a water-sealed drain left in the pleural cavity for twenty-four hours to remove any subsequent effusion.

usually be corrected by surgery. This is best determined at radioscopy (screening). When the flow is increased the hilar shadows are seen to be enlarged and pulsatile, and the lung fields engorged, whereas when the flow is decreased the pulmonary vessels look small, their branches are inconspicuous or absent, and the lungs clear. The distinction is crucial and generally straightforward. Only when it is in doubt is cardiac catheterisation required. The object of surgery in the cyanotic group is to relieve symptoms by increasing the blood flow to the lungs, either by creating an artificial ductus arteriosus, as in the operations of Potts or Blalock, or by a direct attack upon the pulmonary stenosis as advocated by Brock (*vide infra*).

In the acyanotic group surgery is directed to relieving the obstruction in pulmonary stenosis, aortic stenosis or coarctation, and in closing the left to right shunt accompanying auricular and ventricular septal defects, and patent ductus arteriosus.

**Fallot's Tetralogy.** Most children with central cyanosis have this congenital defect. It consists of a high interventricular septal defect; an aorta which overrides (lies astride) both ventricles; pulmonary stenosis, usually of the infundibular type; and right ventricular hypertrophy (Fig. 328). Cyanosis is consequently present from birth or develops very shortly afterwards. Clubbing of the fingers and toes is usual and the nail beds are seen to be blue even when the fingers are warm. Dyspnoea becomes more obvious as the child gets older and begins to move about. About four-fifths of them characteristically squat to regain their breath after walking short distances. This feature is much less common in patients with other cyanotic conditions which cannot be relieved surgically. The children are usually intelligent and often spoilt, but occasionally they are mentally retarded. A tendency for a child to regress in play and associate with younger children is a sign that the condition is worsening and therefore that early operation is indicated. In spite of their severe disability, patients do not succumb more readily than others to intercurrent infections, but they are often poorly developed physically and slow to gain weight.

On auscultation a systolic murmur, sometimes accompanied by a thrill, may be heard in the pulmonary area, and the pulmonary second sound is single and diminished in intensity, indicating a lowered pressure in the partially stenosed artery. Taussig says that in Fallot's tetralogy the intensity of the murmur is inversely proportional to the severity of the stenosis. No other murmurs are audible and the heart itself is not usually much enlarged. The hæmoglobin is raised and the red cell count increased to seven or eight million. Radiologically, the normal prominence of the pulmonary artery shadow on the left border of the heart is wanting. Thus this border looks concave and gives rise to the "sabot-shaped" heart (seen in about half the cases). In the left oblique view there is also an abnormally clear "pulmonary window" (i.e. the area

## CHAPTER 47

### CONGENITAL HEART DISEASE

SUFFERERS from congenital defects of the heart account for only 2 to 3 per cent. of all cardiac patients. The problem they pose, therefore, is not comparable in magnitude to that of rheumatic or degenerative heart disease, but the afflicted are the very young, some of whose lesions respond remarkably to well-planned operation. Congenital defects fall naturally into two groups, those causing central cyanosis and those which do not. Of the former, *Fallot's tetralogy* is much the most important, accounting for 60 per cent. of those producing cyanosis. Others are pulmonary or tricuspid atresia, and more complicated and less treatable anomalies such as Eisenmenger's complex, transposition of the great vessels and the various defects associated with dextro- or isolated lævo-cardia.

Simple pulmonary stenosis may belong to either group, for it does not cause cyanosis unless an atrial defect or patent foramen ovale is present through which blood can be shunted, when the pressure on the right side of the heart rises sufficiently. In the absence of such an opening the patients remain acyanotic.

The other congenital lesions which are not associated with cyanosis are patent ductus arteriosus, which is much the commonest, coarctation of the aorta, auricular and ventricular septal defects and aortic stenosis.

Central cyanosis indicates a shunt of unoxygenated blood from the right to the left side of the heart. It follows, therefore, that an abnormal communication of some sort must exist between the two sides, and also that there is some mechanism which raises the pressure on the right side of the heart above that on the left and thus causes flow to occur from right to left. This rise in pressure is usually produced by some type of pulmonary stenosis, which can be relieved by surgery, but sometimes pulmonary hypertension is responsible, and this cannot be treated surgically. It is therefore important to differentiate the two conditions. Central cyanosis is distinguished from peripheral cyanosis (which affects the extremities, the nose, ears and lips in cold weather or in heart failure) by its being present in the warm buccal mucous membranes and the conjunctivæ, and by polycythæmia out of proportion to the increased hæmoglobin accompanying it. Central cyanosis is usually obvious at rest and becomes worse on exertion, when it is associated with marked dyspnoea. The fingers and toes are often *clubbed*.

In patients with central cyanosis the first thing to decide is whether the blood flow to the lungs is increased or decreased, for a deficiency can

cent. of patients, if untreated, die before the age of seven years, and less than 10 per cent. survive to twenty-one years. Nevertheless, the optimum age for surgery lies between five and seven years. Over twenty years the risks again increase and the chances of a satisfactory result diminish.

Four operations are at present available for the relief of Fallot's tetralogy: Blalock's operation and Potts' operation, which increase the blood flow to the lung by creating an arteriovenous anastomosis; instrumental valvotomy, and a direct open operation. The choice of procedure should be left until the heart and its vessels are directly examined at operation, for the type of lesion dictates the decision. It is true that to make an artificial ductus arteriosus (as is done in the first two operations) is presumably to court those complications to which congenital patent ductus is prone, namely, eventual heart failure or the risk of bacterial endocarditis, and, furthermore, the right ventricle continues to labour as before against the pulmonary stenosis, which tends to increase as fibrin and platelets are deposited upon it. Some limitation in the size of the fistula created is imposed in Blalock's operation by the small size of the systemic artery employed (*vide infra*), but whereas in Potts' operation an opening of any size can be made, if it is made too large right-sided heart failure is likely to be swift. None the less these operations have so far yielded excellent results and in the presence of tricuspid atresia or pulmonary atresia—provided an arterial lumen exists beyond the atresia—there is at present no alternative to them. All these operations have a mortality of between 5 to 10 per cent., and it is three times greater in children less than three years old.

One of the fundamental principles of surgery is that where obstruction exists it should if possible be relieved; this is one of the attractions of the direct attack on the stenosis under hypothermia. Pulmonary stenosis may occur at the valve, or more proximally in the infundibulum, or in both situations. In the valvular type, whether associated with Fallot's tetralogy or occurring as an uncomplicated lesion, there is no doubt that pulmonary valvotomy is the best operation, for it goes some way, or most of the way, towards restoring normal anatomy, but in Fallot's tetralogy the obstruction may involve the whole outflow tract, and is sometimes at both levels. This renders adequate relief of the stenosis more difficult, and the results of direct attack not so satisfactory as that of indirect operations. It must also be remembered that the stenosis is not the only abnormality present. Deficiency of the septum and overriding of the aorta remain to burden the right ventricle and probably cause its eventual failure. Therefore definitive surgery of Fallot's tetralogy must await further development of new methods permitting direct relief of the pulmonary stenosis at whatever level it may be, and closure of the septal defect. Some operations of this nature have already been performed with success, employing mechanical heart-lung pumps. In 20 per cent. of patients pulmonary atresia is present so that direct valvotomy is

below the aortic arch, behind the pulmonary conus seen in a left antero-oblique view). These signs are really of less importance than the recognition of diminished vascularity of the lung fields (small hilar shadows and absent pulsation). In 25 per cent. of cases the aorta is seen to be right sided. Nothing more of importance is likely to be elicited by elaborate angiocardiographic and catheterisation studies, and these, which are not without risks, should be reserved for cases in which the



FIG. 328. The heart in Fallot's tetralogy. The hypertrophied right ventricular wall is well shown. The probe is passed through the interventricular septal defect, and just in front of this can be seen the stenosed pulmonary artery. The aorta, on the left, receives blood from both ventricles. (Dr. S. Lockett's patient.)

exact diagnosis is in doubt. Even the distinction between *valvular* and *infundibular* pulmonary stenosis (which must be made if a direct operation is contemplated) can be decided with more accuracy by direct palpation and direct pressure measurements at the time of operation.

Operation, of whatever kind, should be deferred if the child's condition permits, until it is at least three years old, for before this age the smallness of the structures adds seriously to the technical difficulties: the anastomosis is small, and the risk of thrombosis at the suture line proportionately greater. On the other hand, if the infant's state is clearly deteriorating operation may have to be undertaken earlier. Moreover, about 50 per

and ventilation of the lungs as well as for resting the heart. A marked deterioration in the condition of the patient is a signal to press on and complete the operation, for neither cardiac massage nor the injection of drugs is as likely to restore cardiac function as is relief of the obstruction in the pulmonary artery. This applies even if collapse occurs, as it may do, during induction of anaesthesia; the blood pressure may then drop owing to vasodilatation and lead to cardiac ischaemia and arrest, for the heart is unable to increase its output. Continuous electrocardiographic tracings are made throughout operation and they are carefully scrutinised for such danger signals of anoxia as S-T depression or branch bundle block.

**Blalock's Operation.** Although Blalock himself originally described and preferred a right-sided approach, most surgeons now employ a left thoracotomy because it gives better access to the heart and thus a more complete diagnosis can be made, especially regarding the nature of the pulmonary stenosis. There is greater flexibility through this approach in regard to the choice of the operation best suited to the circumstances revealed by direct examination. Furthermore, in the 25 per cent. who have right-sided aortas, the subclavian branch of the innominate artery, which then lies on the left, is very suitable for anastomosis.

The chest is entered through the whole length of the bed of the 4th left rib and the lung retracted downwards. The pericardium is opened and the anatomy of the heart and its great vessels carefully examined. Among other observations, the type of pulmonary stenosis is determined, and if necessary direct blood pressure readings are made by passing a cannula into the right ventricle and the pulmonary artery. The aorta and its branches are also examined. If Blalock's operation is to be performed, the subclavian artery must be of sufficient length after division to reach the pulmonary artery, and the pulmonary artery must be of sufficient calibre for an anastomosis to be feasible. It is at this stage that the final choice of operation is made.

The mediastinal pleura is divided. Anastomotic vessels underlying it are carefully tied and cut, and the left pulmonary artery is exposed. A fine areolar sheath which covers the vessel is dissected clear so that the artery is absolutely clean from its origin to a little beyond its first division (Fig. 329a); the vessel is then occluded for a few moments to see whether the opposite pulmonary artery can maintain sufficient oxygenation. If all is well, the subclavian artery is identified as it leaves the aortic arch and is dissected free from mediastinal tissue. When a right-sided aorta exists, the left subclavian is a branch of the innominate artery, and especial care must be taken not to injure the recurrent laryngeal nerve where it hooks round the artery to ascend posteriorly into the neck. The superior intercostal vein crosses the subclavian artery superficially and must be divided between ligatures. The subclavian artery is freed up to the point where it gives off its vertebral and internal mammary branches, and the operator must be satisfied that a sufficient length exists

impracticable and no adequate length of pulmonary artery may exist for anastomosis to a systemic vessel.

**Pulmonary and Tricuspid Atresia.** Both cause cyanosis, and have to be distinguished from Fallot's tetralogy, which they closely resemble clinically, for in them the chances of successful operative treatment are at least halved and the operative mortality is greater. Therefore much more severe disability must be present than in Fallot's tetralogy before surgery is justified.

In pulmonary atresia blood reaches the lungs only through enlarged bronchial arteries, or sometimes a patent ductus arteriosus which gives rise to a characteristic continuous "machinery" murmur. The aortic second sound is heard alone in the *pulmonary* area, and on radioscopy there is no sign of the pulmonary artery, although numerous dilated anastomotic bronchial vessels may be visible. As a rule, the pulmonary artery possesses a lumen beyond the atresia, but angiocardiology is imperative to show that there is a sufficient length of patent pulmonary artery for an anastomotic operation to be carried out. Tricuspid atresia presents a similar syndrome to that of Fallot's tetralogy except that the left ventricle is hypertrophied and the right is small. Sometimes blood reaches the right ventricle from the left through a septal defect; sometimes the aorta and pulmonary artery both arise from the left ventricle. In those with a septal defect, blood flow to the lungs is diminished and can be increased by an anastomotic operation, otherwise the flow is increased and surgery has nothing to offer. The distinction is made on the radioscopic appearance of the lung fields, but both cardiac catheterisation and angiocardiology are needed to confirm this, as well as the diagnosis itself.

**Preoperative Care.** Preoperatively, the child is acclimatised to the ward, and to the nurses and physiotherapists who will subsequently care for it. It is made familiar with the oxygen tent or cot in which it will be nursed for the first few post-operative days. Particular attention is paid to adequate fluid intake in polycythæmia, for there is greatly increased risk of thrombosis. Children submitted for surgery not infrequently have some respiratory infection. This must be eliminated as far as possible, but if it does not respond to antibiotics in a reasonable time the operation should nevertheless be performed. Penicillin therapy is always begun a week preoperatively. Digitalis is not required before operation but it may be needed afterwards. Two or three pints of blood are made available to replace any sudden loss that may occur during operation, but transfusion should be circumspect because it adds to the risk of thrombosis. Four per cent. dextrose in 1/5 isotonic saline is given during operation; 0.4 per cent. procaine hydrochloride is added to the solution in doses of up to 500 mg. an hour in a child and 1 gm. in an adult to diminish cardiac irritability.

It is of paramount importance throughout operation to maintain good oxygenation, and pauses are made at convenient moments for reinflation

to turn down to form the union with the left pulmonary artery without tension or sharp angulation. A little extra length may sometimes be gained by flexing the child's head on to its left shoulder. It is preferable to divide the subclavian artery below its branches so that the anastomotic channels to the arm are not disturbed, but very often it is necessary to ligate the branches (Fig. 329a) and divide the subclavian at their point of origin in order to obtain a slightly wider funnel-shaped end for the line of anastomosis (Fig. 329b). Before division, a small bull-dog clamp is applied at the aortic end of the artery. After division the cut end of the subclavian artery is also scrupulously cleaned of all arterial tissue.

A ligature is now tied round each end of the proximally placed bull-dog clamp (Fig. 329b). The ends, which are left long, are passed deep to the left pulmonary artery and used by an assistant to bring the subclavian artery down into close apposition with it. Blalock's clamps are placed across the pulmonary artery as far proximally and distally as possible to leave the maximum length of the vessel clear for suturing (Fig. 329b). These clamps are kept straight and steady by an assistant.

An incision to match the available lumen of the subclavian artery is made in the wall of the left pulmonary artery. A stay-suture is placed at each extremity of the incision and left temporarily untied (Fig. 329b). A posterior everting 5/0 arterial suture on an atraumatic needle is then inserted beginning at the left stay suture. The suture is passed from the outer to the inner side of the subclavian, thence to the inside of the pulmonary artery, back to the outside of the subclavian, and so on (Fig. 329c). When the row is completed, the suture is gently pulled tight approximating the two vessels, and the ends are tied to the stay-sutures which are tied in turn. All knots are tied externally, and each bite is 1 to 2 mm. long. The anterior edges are similarly sutured, or, if desired, a continuous everting mattress stitch may be employed instead. At convenient intervals during the operation it is wise to pause and allow the retracted lung to be re-inflated for a time, but any serious deterioration in the child's condition is an indication to complete the anastomosis as rapidly as possible rather than to procrastinate or abandon the operation. When the anastomosis is completed, the distal clamp on the pulmonary artery is released first, and any oozing at the suture line dealt with by gentle pressure with a swab, or, if necessary, by inserting extra interrupted stitches where required. The proximal pulmonary clamp is then taken off, and finally the bull-dog clamp on the subclavian artery. The anastomosis is inspected on each new accretion of pressure. As soon as a free flow is established through the junction a thrill becomes palpable.

If the pulmonary artery is of unusually small calibre, though not actually atresic, or if it is found that undue tension is needed to anastomose the subclavian to its side, the left pulmonary artery may be tied and divided as it emerges from the pericardium and an end-to-end junction made between its distal part and the subclavian artery.



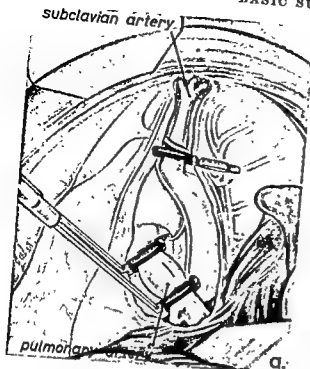
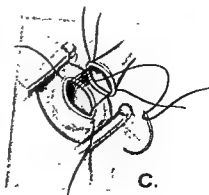
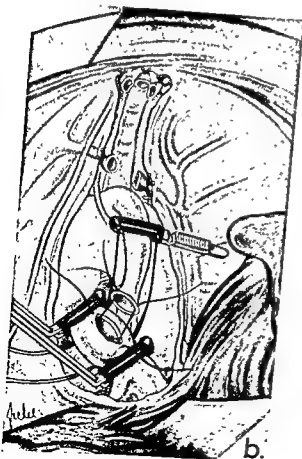


FIG. 320. (a) The left subclavian and left pulmonary arteries ready for anastomosis. The branches of the former have been ligated to afford more room. (b) The subclavian has been drawn down to the pulmonary artery by ligatures around the bulldog clamp, and stay sutures have been inserted. (c) The posterior sutures of the anastomosis being inserted.



through the wall of the aortic lip held by the clamp and the corresponding part of the pulmonary artery (Fig. 230b). If this length is exceeded the resulting fistula will be too great and will almost inevitably lead to heart failure. Atraumatic sutures (5/0 silk on No. 9 needles) are then used. The anastomosis is begun at the upper angle and the suture is carried inward through the wall of the pulmonary artery from outside, across through the aortic wall from inside out, and tied so that the knot is external. This method carries the cut edges. The posterior edges of the vessels are sutured together by a continuous over and-over stitch with bites about 1 mm. apart, and when the lower angle is reached the suture is locked, or tied, and the anterior layer of the anastomosis similarly completed (Fig. 230c). First the distal and then the proximal large ligatures (including the left pulmonary artery) are cut and slipped off, and the anastomosis is tested for leaks under the increasing pressure. Its posterior aspect is inaccessible, therefore the greatest care must be taken with its suturing in the first place. Finally the Potts' clamp is taken off the aorta. A marked thrill is at once felt over the pulmonary artery as the systemic blood courses into it.

**Pulmonary Stenosis and Pulmonary Valvotomy.** Pulmonary stenosis as an isolated lesion or in association with other congenital cardiac defects occurs as commonly as patent ductus arteriosus, but in a great many patients the stenosis is mild, the prognosis is excellent, and surgery is not required. The advantages of valvotomy over anastomotic operations have already been discussed. The operation was devised and developed by Brock.

In simple stenosis, i.e. not forming part of Fallot's tetralogy, central cyanosis does not usually appear before the age of three years, and then only if a patent foramen exists between the right and left atrium through which blood can be shunted as pressure on the right side of the heart rises; this may not occur until much later than three years. If the septum is intact, the pressure in some cases may soon rise and cause increasing right ventricular enlargement which goes on to failure. In Fallot's tetralogy, the overriding aorta provides a safety mechanism whereby the pressure is prevented from rising so high, therefore the problem is one of anoxia rather than failure, and the heart is not enlarged as it becomes in stenosis alone. Although the electrocardiogram does show right ventricular preponderance in Fallot's tetralogy, it is never so severe as in stenosis occurring apart from the tetralogy, and in stenosis, even if the heart has not begun to enlarge, the region of the pulmonary conus as seen in X-rays is full and not concave. As the heart enlarges, the harsh systolic murmur audible on the left of the sternum may cease, and the pulmonary second sound, already weaker than that heard in the tetralogy, also diminishes.

Surgery is more dangerous and the results less successful, when cardiac enlargement and cyanosis have begun. Therefore the decision to operate

**Potts' Operation.** Provided the aorta is on the left side, an aorto-pulmonary artery anastomosis can be performed as an alternative to Blalock's operation. The left pulmonary artery is dissected free and the ability of the child to survive its temporary occlusion tested as in Blalock's operation. A pair of fairly thick untied silk ligatures are placed as proximally and distally as possible on the main trunk of the left pulmonary artery (i.e. one close to its origin and one close to its first division), so that by pulling upon them blood is prevented from flowing into the intervening segment (Fig. 330a). The part of the descending aorta to which the pulmonary artery can most easily be approximated is

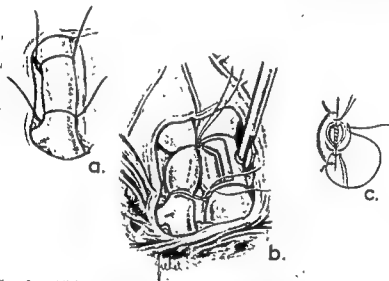


FIG. 330. (a) The left pulmonary artery encircled by ligatures. (b) The Potts' clamp has been applied to the aorta and the pulmonary artery approximated to it. (c) The posterior layer of the anastomosis has been completed and the anterior begun.

then chosen, and after the pleura and fascia overlying it have been stripped off it is mobilised by division of two or three pairs of intercostal arteries at this level. A Potts' clamp of suitable size is used to isolate a part of the wall of the aorta without interrupting the blood flow through the main part of the aorta (Fig. 330b). If necessary the part between the jaws of the clamp is pulled out with forceps to give more room for the anastomosis. When the clamp is closed, a thrill is felt peripheral to it confirming that the blood flow continues, and this is most important, for if the aorta is completely occluded the spinal cord is likely to be deprived of its blood supply.

The ligatures round the pulmonary artery are tightened and tied firmly to each end of the Potts' clamp, thus both occluding the left pulmonary artery and bringing it into close contact with the aorta (Fig. 330b). Incisions of not more than 4 mm. are cut squarely with a very sharp knife

either with instruments or by pressure. The probe is withdrawn and replaced by a catheter catheter, and pressure readings are recorded (on an electromanometer) from the ventricle and the artery beyond the stenosis. The pressure in the former is high, about 80 mm. of mercury or more during systole, whereas the pressure in the latter is much lower and the difference between them confirms the site of the obstruction. As the

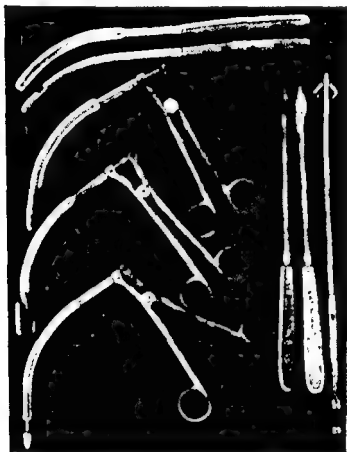


FIG. 331. Brock's dilators and valvulotomes used in pulmonary valvotomy. The cutting instruments are on the right.

catheter is withdrawn, it is possible to determine the presence or absence of additional stenosis in the infundibulum. When pressure readings are complete, a wider probe (4 mm. in diameter) is substituted for the catheter and passed up to give an accurate idea of the position of the stenosis (Fig. 331). It is then followed by the Brock valvulotome itself, which is probe pointed. It has sharp shoulders for splitting the valve from side to side in one direction. The valve is cut by applying a little pressure to the valvulotome after the probe has engaged in the valve orifice. The instrument is made in several sizes ranging from 0.7 to 1.25 cm. in

should be made early rather than late. Cardiac catheterisation is of value in detecting the rise in right ventricular pressure before it is clinically manifest. A pressure of 80 to 100 mm. of mercury, or electrocardiographic evidence of increasing right-sided strain, is an indication for surgery. Valvotomy should reduce the ventricular pressure by more than half. The chief risks of the operation are sudden cardiac arrest or failure, and these are more likely to occur in patients who have reached their teens or twenties, and in whom there is cardiac enlargement, than in younger ones in whom disability and cyanosis are still slight.

*Valvotomy.* The patient lies supine on the operating table with the left arm abducted laterally on a splint to expose the axilla. The chest is entered through a curved inframammary incision extending from the mid-axilla to the edge of the sternum at the level of the third interspace. The incision is deepened to expose the fourth rib by detachment of the pectoralis insertions. The periosteum of the rib is stripped off its upper border, and the rib bed is incised (without removing the rib) throughout its whole length to open the pleural cavity. For a wider exposure the cartilage of the fourth rib may be cut, or, if necessary, a transverse division of the sternum may be carried out at the level of the third intercostal space.

The thymus and all areolar tissue are dissected from the pericardium which is then widely opened by a vertical incision parallel to and about 1 cm. medial to the phrenic nerve. A generous rectangular flap is made by two further incisions at right-angles to, and running medially from, each end of the first incision. Stay sutures are attached to each corner of this flap so that an assistant can, by pulling upon them, slightly rotate the heart laterally and at the same time hold it firm. This manoeuvre is carried out only during actual instrumentation. At all other times the traction sutures are left slack as any torsion of the heart causes cardiac embarrassment.

The diagnosis of pulmonary stenosis is confirmed by direct inspection and palpation. Post-stenotic dilatation of the pulmonary artery is seen, and the valve cone itself is felt to harden and rise during systole and transmit a jet of blood into the thin-walled chamber beyond. A point is selected on the anterior wall of the right ventricle, well away from any coronary branches, and about 5 cm. proximal to the pulmonary valve itself, through which to pass the valvotomy instrument.

No controlling sutures are required at the point selected for cardiomy, for any bleeding is readily controlled by digital pressure and tension on the pericardial flap helps to stabilise the heart. A vertical incision 1 cm. long is then made almost through the myocardium, which is finally pierced by a curved probe, the end of which can be felt between the left index and thumb as it enters the stenosed valve. As succeeding instruments are passed they are similarly guided from outside by the fingers, but care must be taken at all times not to occlude the blood flow in the artery

either with instruments or by pressure. The probe is withdrawn and replaced by a cathiac catheter, and pressure readings are recorded (on an electromanometer) from the ventricle and the artery beyond the stenosis. The pressure in the former is high, about 80 mm. of mercury or more during systole, whereas the pressure in the latter is much lower and the difference between them confirms the site of the obstruction. As the

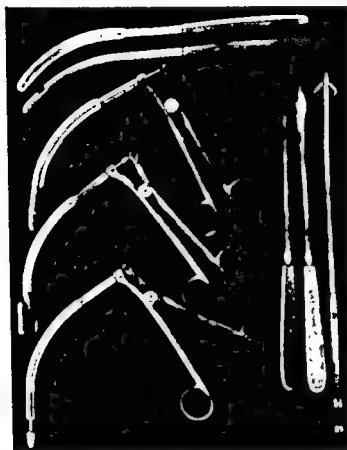


FIG. 331. Brock's dilators and valvulotomes used in pulmonary valvotomy. The cutting instruments are on the right.

catheter is withdrawn, it is possible to determine the presence or absence of additional stenosis in the infundibulum. When pressure readings are complete, a wider probe (4 mm. in diameter) is substituted for the catheter and passed up to give an accurate idea of the position of the stenosis (Fig. 331). It is then followed by the Brock valvulotome itself, which is probe pointed. It has sharp shoulders for splitting the valve from side to side in one direction. The valve is cut by applying a little pressure to the valvulotome after the probe has engaged in the valve orifice. The instrument is made in several sizes ranging from 0.7 to 1.25 cm. in

diameter. The largest which can pass safely through the valve ring is selected. As the valvulotome is withdrawn, bleeding from the heart is controlled by digital pressure on the incision in the myocardium. Finally, an expanding valve dilator is introduced and opened in two directions at the site of the stenosis which is thus split completely across to a diameter of 2.5 to 3.5 cm. according to the size of the vessel. Two complete cusps result *which do not permit any regurgitant flow between them*. All the instruments have to turn sharply through almost a right angle to enter the region of the valve. If the turn made is excessive the myocardium may be torn; if insufficient, the interventricular septum (down which runs the Bundle of His) may be injured. It is important, therefore, that none of the manoeuvres should be hurried and that the fingers of the left hand outside the heart should check the position of instruments and help to guide them.

The myocardium is closed with three 2/0 silk sutures on a curved No. 6 atraumatic needle. They are passed through the muscle while the left index finger controls bleeding from the wound. Two of them are tied, and through the smaller remaining orifice the cardiac catheter is again passed to make final pressure recordings in the artery and the right ventricle. Relief of the obstruction is shown by a rise in the pulmonary arterial pressure, a fall in ventricular pressure, and the appearance of a pulse wave in the artery where none was visible before. However, the increase in blood flow is more important than the pressure changes. If the stenosis is not adequately relieved, the dilating instruments must be re-introduced. The pressures are repeated until relief of the stenosis is indicated. The catheter is then withdrawn and the final suture in the myocardium tied. The pericardial sac is thoroughly irrigated with normal saline to wash out blood clot that may have accumulated, and when the washings are quite clear the sac itself is sutured, leaving an adequate dependent opening for drainage. The left thoracotomy wound is closed in the usual manner, and a water-sealed drain left in the pleural cavity.

**Open Exploration under Hypothermia.** In some 40 per cent. of cases of Fallot's tetralogy the stenosis is in the infundibulum between the ventricle and pulmonary artery, and in another 20 per cent. is in both positions. Instrumental valvotomy is unsatisfactory in such instances which call for open operation with deliberate resection of the obstructing muscle. As this usually involves interruption of the blood-flow for periods longer than the three minutes cerebral cells can survive anoxia, hypothermia is necessary; for at 30° C. the oxygen requirements of the cerebral cells are roughly halved, and a safe operating time of six or seven minutes is afforded. Below 30° C. there is serious risk of ventricular fibrillation. Because of its obvious advantages open exploration is now replacing indirect operations in all cases suitable for it.

The anaesthetised patient is cooled by semi-immersion in an ice-water bath, or by means of chilled glycol circulating in rubber coils arranged

on the body, and oesophageal temperatures (which approximate to those of the brain) are recorded. At 32° C. cooling is stopped, and the operation begun, for the temperature continues to drop 2° or 3° thereafter. Rewarming then begins, and as soon as the operation is complete the temperature is raised to 35° C. before the patient is returned to bed.

On the operating table both arms are extended on splints, and through curved bilateral inframammary incisions reaching from axilla to axilla, both pleural cavities are opened, the left through the third intercostal space, the right through the fourth, and the sternum cut transversely between them. A large rectangular pericardial flap is turned back to the right and stitched to the wound edge. The heart is then widely exposed and the nature of the stenosis (i.e. whether valvular, infundibular, or both) determined. Stay sutures are inserted on the right ventricular wall below the infundibulum and on the wall of the pulmonary artery beyond the valve. Both venæ cavae are secured by tapes, and after some ten further beats have emptied the heart, the aorta and pulmonary artery are clamped in the transverse sinus. The wall of the pulmonary artery and infundibulum is incised between the stay sutures, the valve cut (if necessary) along its commissure, and the myocardium stenosing the infundibulum cut away with a small bone rongeur. The outflow tract is then flooded with saline, trepanned, and the clamps released.

**Post-operative Care.** After pulmonary valvotomy, as well as after Blalock's and Potts' operations, water-sealed drains are left in the pleural cavity for twenty-four to forty-eight hours, both to ensure full re-expansion of the lung and to enable any serious bleeding to be recognised promptly. All patients are returned to bed in an oxygen tent and nursed flat until consciousness is fully restored. The pulse rate and blood pressure are recorded quarter-hourly, especially as any sudden fall in blood pressure predisposes to thrombosis, either cerebral, or at the suture line in anastomotic operations. If necessary, small doses of suitable vaso-pressor drugs such as noradrenaline or methedrine are given to help sustain the blood pressure. Handling the child tends to cause a fall in blood pressure and is to be discouraged. If the anastomosis is too small, as it tends to be in infants under the age of three or four years who have had Blalock's operation performed, there is a particularly serious risk of thrombosis at the suture line leading to complete obstruction; nevertheless, the use of heparin in the early post-operative period is probably unwise, for it may cause reactionary hæmorrhage.

Adequate oxygenation is of prime importance at all times, so the airway must constantly be kept clear and every effort made to secure re-expansion of the lung by removal of any air or effusion impeding it. After operation, there is usually prompt improvement in the patient's colour, and the hæmoglobin percentage and red cell count fall sharply. Radiologically, increased vascularity of the lung fields is seen, and sometimes it is excessive, particularly after Potts' operation. There may



even be severe pulmonary congestion and some degree of cardiac enlargement or failure, calling for prolonged rest. In all cases it is of great importance when convalescence is reached to put a brake on activity, especially in older children, for the new freedom from crippling dyspnoea naturally encourages exuberant mobility which may throw too great a strain upon the heart. Any progressive enlargement of the latter is a signal for prolonged bed rest.

**Septal Defects.** *Ventricular septal defects* occur both as isolated lesions (about 8 per cent. of all cases of congenital heart disease) and in conjunction with other deformities such as Fallot's tetralogy. Many produce a left-to-right shunt of blood with a rising pressure on the right side of the heart and pulmonary congestion, but as the pressure increases on the right, this shunt may become reversed with resulting cyanosis. No surgical treatment is as yet *established* for the closure of these defects, but must await the development of a satisfactory technique for operating within the open heart. Some closures have been performed with the aid of a mechanical heart pump, hypothermia (to reduce cerebral metabolism), or donor cross-circulation; all existing methods have serious drawbacks and the procedures remain temporarily in the realm of experimental surgery.

*Atrial defects* are much commoner than ventricular defects, being indeed the commonest congenital cardiac abnormality, but as a rule the outlook is good until middle age, when patients begin to suffer increasing disability due to shunting from the left to the right atrium, and they eventually develop congestive failure. The right side of the heart and the pulmonary artery are conspicuously large, whereas the left side of the heart and the aorta are small. Right bundle-branch block is shown in the E.C.G.

Most interatrial defects lie posteriorly on the septal wall (ostium secundum), but some are anterior and involve the valve ring (ostium primum). Hence the latter are often associated with a mitral or tricuspid lesion and cannot be closed without still further impairing valve function. It is important therefore to distinguish them clinically and exclude them from surgery. Cardiac catheterisation is essential. Because of the poor ultimate prognosis many attempts have been made, some of them successful, to close interatrial defects indirectly by encircling the septum with a purse-string or mattress suture from outside, or by invaginating the atrial appendages or the atrial wall itself into the foramen and anchoring them there by sutures. All these procedures are essentially blind, and it is obvious that direct closure in the dry and open heart with the aid of hypothermia is vastly preferable.

The child is cooled to 30° to 32° C. The heart is widely exposed by dividing the sternum transversely and opening both fourth intercostal spaces, and the two venae cavae are encircled with tapes to control the blood flow through them. The nature of the defect is confirmed by

digital exploration. After a few beats to empty the pulmonary circulation, the aorta and pulmonary trunk are clamped. The right atrium is opened, the defect is closed by interrupted sutures, the operation field is flooded with normal saline to avoid air embolism, the atrial wall is clamped, and the flow through both arteries and veins resumed before the atrium is sewn up and the chest shut.

**Aortic Stenosis.** Brock, Bailey, Cleland, Glover, Logan and a number of other surgeons, encouraged by the success of direct operations on the mitral and pulmonary valves, have all done pioneering work on the surgical relief of aortic stenosis. Usually a dilator, similar to that used by Brock after pulmonary valvotomy, is passed up through the left ventricle and opened in the valve orifice. However, many factors combine to limit success: there is a much greater risk of producing aortic incompetence, the results of which are even more serious than when regurgitation is produced in operations upon the mitral valve; the aortic valve is frequently calcified, thus increasing the difficulty of operation and the risk of emboli; the disease process often affects the ascending part of the aorta from which the coronary vessels arise, so that there is a chance of cardiac ischemia; the hypertrophied left ventricular myocardium tolerates incision and suturing badly, and bleeding from it may be difficult to control. For the latter reason in particular, some surgeons approach the aortic valve from above through an artificial operating tunnel sewn on to the aortic arch. It must be borne in mind that the majority of patients operated upon are those already suffering from the effects of their lesion, and that no other form of treatment but valvotomy could relieve them. The operative mortality in various series published by those most experienced in this field has ranged from 10 to 40 per cent. Further technical advances must be awaited before patients with aortic stenosis can confidently be submitted to surgery, but direct approach to the valve under hypothermia probably offers the most satisfactory prospect.

**Aortic Incompetence.** So far the only operation that has had any success in this disorder is that of Hufnagel of Washington who divides the descending aorta and interposes into it a plastic ball valve. Most of his eighty patients were already severely ill; 50 per cent. of them were in severe heart failure, and none were accepted for surgery who did not have signs of decompensation. The operative mortality was 20 per cent., and 20 per cent. of those surviving died within two years. In the rest the operation was successful, but insufficient time has elapsed for the ultimate fate of the prosthesis to be known.

even be severe pulmonary congestion and some degree of cardiac enlargement or failure, calling for prolonged rest. In all cases it is of great importance when convalescence is reached to put a brake on activity, especially in older children, for the new freedom from crippling dyspnoea naturally encourages exuberant mobility which may throw too great a strain upon the heart. Any progressive enlargement of the latter is a signal for prolonged bed rest.

**Septal Defects.** *Ventricular septal defects* occur both as isolated lesions (about 8 per cent. of all cases of congenital heart disease) and in conjunction with other deformities such as Fallot's tetralogy. Many produce a left-to-right shunt of blood with a rising pressure on the right side of the heart and pulmonary congestion, but as the pressure increases on the right, this shunt may become reversed with resulting cyanosis. No surgical treatment is as yet established for the closure of these defects, but must await the development of a satisfactory technique for operating within the open heart. Some closures have been performed with the aid of a mechanical heart pump, hypothermia (to reduce cerebral metabolism), or donor cross-circulation; all existing methods have serious drawbacks and the procedures remain temporarily in the realm of experimental surgery.

*Atrial defects* are much commoner than ventricular defects, being indeed the commonest congenital cardiac abnormality, but as a rule the outlook is good until middle age, when patients begin to suffer increasing disability due to shunting from the left to the right atrium, and they eventually develop congestive failure. The right side of the heart and the pulmonary artery are conspicuously large, whereas the left side of the heart and the aorta are small. Right bundle-branch block is shown in the E.C.G.

Most interatrial defects lie posteriorly on the septal wall (ostium secundum), but some are anterior and involve the valve ring (ostium primum). Hence the latter are often associated with a mitral or tricuspid lesion and cannot be closed without still further impairing valve function. It is important therefore to distinguish them clinically and exclude them from surgery. Cardiac catheterisation is essential. Because of the poor ultimate prognosis many attempts have been made, some of them successful, to close interatrial defects indirectly by encircling the septum with a purse-string or mattress suture from outside, or by invaginating the atrial appendages or the atrial wall itself into the foramen and anchoring them there by sutures. All these procedures are essentially blind, and it is obvious that direct closure in the dry and open heart with the aid of hypothermia is vastly preferable.

The child is cooled to 30° to 32° C. The heart is widely exposed by dividing the sternum transversely and opening both fourth intercostal spaces, and the two venae cavae are encircled with tapes to control the blood flow through them. The nature of the defect is confirmed by

digital exploration. After a few beats to empty the pulmonary circulation, the aorta and pulmonary trunk are clamped. The right atrium is opened, the defect is closed by interrupted sutures, the operation field is flooded with normal saline to avoid air embolism, the atrial wall is clamped, and the flow through both arteries and veins resumed before the atrium is sewn up and the chest shut.

**Aortic Stenosis.** Brock, Bailey, Cleland, Glover, Logan and a number of other surgeons, encouraged by the success of direct operations on the mitral and pulmonary valves, have all done pioneering work on the surgical relief of aortic stenosis. Usually a dilator, similar to that used by Brock after pulmonary valvotomy, is passed up through the left ventricle and opened in the valve orifice. However, many factors combine to limit success: there is a much greater risk of producing aortic incompetence, the results of which are even more serious than when regurgitation is produced in operations upon the mitral valve; the aortic valve is frequently calcified, thus increasing the difficulty of operation and the risk of emboli; the disease process often affects the ascending part of the aorta from which the coronary vessels arise, so that there is a chance of cardiac ischemia; the hypertrophied left ventricular myocardium tolerates incision and suturing badly, and bleeding from it may be difficult to control. For the latter reason in particular, some surgeons approach the aortic valve from above through an artificial operating tunnel sewn on to the aortic arch. It must be borne in mind that the majority of patients operated upon are those already suffering from the effects of their lesion, and that no other form of treatment but valvotomy could relieve them. The operative mortality in various series published by those most experienced in this field has ranged from 10 to 40 per cent. Further technical advances must be awaited before patients with aortic stenosis can confidently be submitted to surgery, but direct approach to the valve under hypothermia probably offers the most satisfactory prospect.

**Aortic Incompetence.** So far the only operation that has had any success in this disorder is that of Hufnagel of Washington who divides the descending aorta and interposes into it a plastic ball valve. Most of his eighty patients were already severely ill; 50 per cent. of them were in severe heart failure, and none were accepted for surgery who did not have signs of decompensation. The operative mortality was 20 per cent., and 20 per cent. of those surviving died within two years. In the rest the operation was successful, but insufficient time has elapsed for the ultimate fate of the prosthesis to be known.

even be severe pulmonary congestion and some degree of cardiac enlargement or failure, calling for prolonged rest. In all cases it is of great importance when convalescence is reached to put a brake on activity, especially in older children, for the new freedom from crippling dyspnoea naturally encourages exuberant mobility which may throw too great a strain upon the heart. Any progressive enlargement of the latter is a signal for prolonged bed rest.

**Septal Defects.** *Ventricular septal defects* occur both as isolated lesions (about 8 per cent. of all cases of congenital heart disease) and in conjunction with other deformities such as Fallot's tetralogy. Many produce a left-to-right shunt of blood with a rising pressure on the right side of the heart and pulmonary congestion, but as the pressure increases on the right, this shunt may become reversed with resulting cyanosis. No surgical treatment is as yet established for the closure of these defects, but must await the development of a satisfactory technique for operating within the open heart. Some closures have been performed with the aid of a mechanical heart pump, hypothermia (to reduce cerebral metabolism), or donor cross-circulation; all existing methods have serious drawbacks and the procedures remain temporarily in the realm of experimental surgery.

*Atrial defects* are much commoner than ventricular defects, being indeed the commonest congenital cardiac abnormality, but as a rule the outlook is good until middle age, when patients begin to suffer increasing disability due to shunting from the left to the right atrium, and they eventually develop congestive failure. The right side of the heart and the pulmonary artery are conspicuously large, whereas the left side of the heart and the aorta are small. Right bundle-branch block is shown in the E.C.G.

Most interatrial defects lie posteriorly on the septal wall (ostium secundum), but some are anterior and involve the valve ring (ostium primum). Hence the latter are often associated with a mitral or tricuspid lesion and cannot be closed without still further impairing valve function. It is important therefore to distinguish them clinically and exclude them from surgery. Cardiac catheterisation is essential. Because of the poor ultimate prognosis many attempts have been made, some of them successful, to close interatrial defects indirectly by encircling the septum with a purse-string or mattress suture from outside, or by invaginating the atrial appendages or the atrial wall itself into the foramen and anchoring them there by sutures. All these procedures are essentially blind, and it is obvious that direct closure in the dry and open heart with the aid of hypothermia is vastly preferable.

The child is cooled to 30° to 32° C. The heart is widely exposed by dividing the sternum transversely and opening both fourth intercostal spaces, and the two venae cavae are encircled with tapes to control the blood flow through them. The nature of the defect is confirmed by

or not the murmur heard is continuous. In such cases a phonocardiograph record usually resolves the doubt, but very rarely the murmur may be atypical and apparently confined to systole, so that cardiac catheterisation is needed to prove the diagnosis, although it is quite unnecessary in the vast majority of cases.

Cyanosis is never present unless blood flows not from the aorta to the pulmonary artery but in the reverse direction. Un-oxygenated blood thereby enters the systemic system. A continuous murmur is not present in these cases. This reversal and the accompanying cyanosis are exceedingly rare and call for rigorous reassessment of the diagnosis. Cyanosis may result from pulmonary hypertension, itself secondary to the fistula, or the ductus may be associated with other congenital cardiac anomalies. In either case, closure is contraindicated, especially in the latter circumstances, when the ductus is in all probability a life-saving channel through which blood can reach the lungs (the operations of Blalock or Potts for Fallot's tetralogy simply create artificially such a fistula).

The onset of infective endocarditis in a patient with a patent ductus is unusual before puberty but the risk increases thereafter; infants are, however, not entirely immune. Insidious at first, the infection is accompanied by increasing weakness, pallor, anaemia and loss of weight. The pulse becomes faster; there is irregular fever generally rising in the evening and associated with a minor rigor. It is then that a sample of blood should be taken for culture. The infecting organism is *Streptococcus viridans*. Later embolic manifestations occur: there are blood cells in the urine, petechiae and transient painful nodules. A hæmoptysis may occur. The spleen is sometimes palpable. If untreated, the disease progresses over about twelve months to a fatal termination. It can, however, be controlled by massive penicillin therapy, which should always precede surgery. Although bacterial endocarditis substantially increases the risk of operation, for the ductus wall may be much more friable, it is an urgent indication for operating on the fistula. Clearly, however, prevention of infection should be the aim. Therefore the patent ductus ought to be closed early in childhood when the risk is at a minimum and complications have not occurred.

**The Operation.** The left side of the chest is opened by a standard posterolateral incision through the fourth intercostal space without removing the rib. The lung is held downwards away from the superior mediastinum and covered with a warm moist towel. The region of the ductus is inspected and palpated for the maximal thrill. This point is usually not over the ductus itself but over the base of the adjacent pulmonary artery. The mediastinal pleura in the triangle formed by the diverging phrenic and vagus nerves above the lung root is incised from the hilum below to a point near the apex of the triangle (Fig. 332a), and the fatty areolar tissue thus exposed is cleaned away. A lymphatic gland is constantly present, lying superficial to the ductus itself. This gland is

## CHAPTER 48

### PATENT DUCTUS ARTERIOSUS

THE ductus arteriosus which connects the pulmonary artery with the aorta, and through which blood from the right side of the heart reaches the systemic circulation during foetal life, normally closes as soon as the lungs expand and take up the pulmonary flow; the channel then scleroses to become the ligamentum arteriosum. For reasons that are not understood the channel sometimes persists, most often alone, but occasionally with associated cardiac abnormalities. It constitutes an arteriovenous fistula which leads almost inevitably to death from left-sided heart failure within twenty-five to thirty years. The speed with which this ensues depends on the degree of patency and the corresponding leak of blood from the aorta back into pulmonary circulation. There is steadily increasing cardiac dilatation, decompensation and eventual death, if meanwhile the patient has not already succumbed to subacute bacterial endocarditis (which overtakes about a third of the patients). Once any abnormal cardiac murmur has been detected in a child it is often condemned to a life of cardiac invalidism, being kept from play and often from schooling as well, and, furthermore, a large vascular shunt may actually result in delayed physical and mental development. The longer surgery is postponed the greater the likelihood of cardiac damage and the more dangerous the operation. The risk of surgery in infancy is negligible, but in the adult considerable. When the diagnosis of patent ductus arteriosus has been made there is no excuse for procrastination. Operative closure should be undertaken at the first possible opportunity.

A patent ductus in infancy produces as a rule no symptoms, except that the child may become breathless more quickly than others if the communication is large. Therefore it is usually on routine examination that the condition is first detected. There is a thrill felt, maximal over the second left intercostal space, and it is accompanied by a machinery-like murmur continuous throughout systole and diastole. Such findings are in themselves virtually diagnostic, but in addition the pulse pressure is greater than normal—the diastolic pressure being low whereas the systolic pressure is about normal. The heart is usually not enlarged at first, but if the shunt is great or the diagnosis delayed to adult life, it may be much enlarged. Radiographically the lung fields appear congested, the pulmonary artery shadow is prominent, the hilar dance is increased. Both these latter signs also accompany an atrial septal defect. The electrocardiograph shows either no abnormality or slight left axis deviation. It is sometimes difficult on auscultation alone to determine whether

they can be moved quite freely round the back of the ductus. The effect of occlusion with the finger is again tested, and the child's colour and condition observed in order to assure that the fistula does not compensate in any way for an undetected cardiac lesion. A skein of floss silk is then threaded round the ductus with the aid of the forceps and tied firmly with a surgeon's knot as close to its aortic end as possible without interfering in any way with the recurrent laryngeal nerve (Fig. 332b). The thrill and cardiac turbulence should immediately be completely abolished. The persistence of a thrill means that the knot has been tied insufficiently tightly or that some other lesion is present. A second skein is passed and tied similarly at the end nearest the pulmonary artery.

Some surgeons prefer, on the grounds of possible recanalisation, always to divide the ductus completely and sew up the ends. This would, of course, have to be done if the ductus were damaged during dissection prior to intended ligation. Therefore, suitable clamps for the purpose should always be at hand. But compared with the simple and relatively easy operation already described, division and suture are more complex and dangerous.

If damage to the ductus occurs, its medial wall is most likely to be the one torn, and bleeding from this can be controlled by firm digital pressure from the pleural side. While this is maintained by an assistant, the aortic arch is mobilised sufficiently for the application of a Potts' clamp which closes the aortic end of the ductus without interrupting the main systemic flow. The base of the pulmonary artery is next freed of pericardium and a similar clamp used to control it. The ductus is divided in the middle to leave a sufficient cuff for direct suture with a fine atraumatic vascular needle on 4/0 silk. Alternatively, special finely toothed clamps devised by Gross may be applied directly to each end of the ductus leaving a sufficient flange between for division and suture of the ends. The mediastinal pleura is now closed with a continuous stitch of fine linen and the lung is re-expanded. Care must be taken to ensure that all parts of the lung are well aerated before the chest is closed. No drain is necessary.

On the first post-operative day an X-ray should show the lung fully expanded with perhaps a minimal effusion in the left costophrenic angle. Atelectasis calls for prompt bronchoscopy and much effusion for aspiration, but very seldom is either necessary. Usually the child gets out of bed on the day after operation. The pulse pressure returns to a normal figure, for the diastolic pressure rises after the ligation.

As a rule no murmur remains, but sometimes a short residual systolic murmur persists in the second interspace. It is of no clinical significance and it must be impressed on all concerned that the child is now normal in all respects and can henceforth lead an active and unrestricted life.



dissected clear and the small blood vessels entering it ligated. Care must be taken not to damage the vagus nerve as it is still accompanied by its recurrent laryngeal branch; coagulation in its vicinity is therefore avoided. The recurrent laryngeal nerve is defined as it leaves the vagus and curves round the lower margin of the ductus to ascend behind it and the arch of the aorta to reach the neck. The nerve should not be picked up, or held back by a stay suture, but should merely be recognised and avoided. As dissection proceeds, the dimensions and direction of the ductus become obvious, though it at first seems shorter and wider than it really is. This is partly due to a little diverticulum of the pericardial sac which envelops its lateral and superior margins but can be dissected back from them (Fig. 332a). Definition of the inferior margin of the ductus is easy,

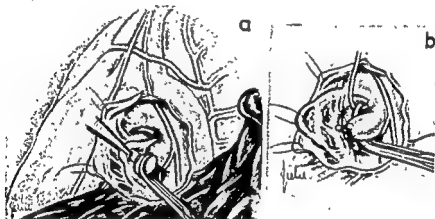


FIG. 332. (a) The pericardial extension is being peeled back off the patent ductus. Note the relation of the recurrent laryngeal nerve. (b) An O'Shaughnessy forceps has been passed behind the ductus to secure the second floss silk ligature; the first has already been tied at the pulmonary artery end.

for only loose areolar tissue intervenes between it and the pulmonary artery below. The course of the recurrent laryngeal nerve is followed by blunt dissection until it disappears on the medial surface of the ductus. Superiorly a fairly tough condensation of the pericardium extends from the ductus up on to the aortic arch. This tissue is picked up and cut with scissors until the upper margin of the ductus is quite clear. Then the ductus is seen to be longer and narrower than it appeared to be at first. A right-angled O'Shaughnessy forceps is introduced beneath the inferior margin (Fig. 332b) while the left index finger rests lightly on the surface of the ductus, and by very gentle blunt dissection the forceps are gradually passed up behind. Pressure between the finger and the forceps abolishes the thrill and diminishes the heart's turbulence, which may previously have been considerable. The blunt dissection is continued until the point of the forceps emerges in the superior gap (Fig. 332b). Sometimes it is necessary to cut a few strands of fibrous tissue overlying the forceps before

and the results are good in the great majority of survivors. About 25 per cent. also have bicuspid aortic valves which may be partially incompetent, and another 10 per cent. have intracranial aneurysms which may subsequently rupture. Both these conditions are associated congenital anomalies which prejudice the patient's future.

The diagnosis of coarctation of the aorta is made by the absence of femoral and popliteal pulses, a blood pressure much lower than normal in the legs and higher than normal in the arms. This hypertension in the upper extremities may be mild or very pronounced, and should it differ by more than 30 or 40 mm. of mercury in the two arms the coarctation is proximal to the left subclavian artery. Although a rare variant, it virtually spells inoperability. Exercise at once produces a disproportionate rise in blood pressure, often of 100 mm. or more. The pulsation of collateral arteries around the scapula, above and below the clavicles, and down each border of the sternum, can be seen and felt. Murmurs are often audible over these vessels, particularly in the area between the scapulae. The systolic murmur over the base of the heart is inconstant. Although symptoms are often absent, headache and oppressive throbbing in the neck, or shortness of breath may draw attention to the anomaly. Poor circulation in the legs sometimes leads to delayed healing of injuries, or causes numbness and coldness of the feet.

Radiography sometimes shows the heart to be enlarged even in infancy, but it is usually small. The aortic knuckle is inconspicuous, although the ascending aorta is visible to the right of the sternum and extends abnormally far up in the mediastinum. Typical notching of the inferior borders of the ribs by the enlarged intercostal arteries (acting as anastomotic channels) can be seen.

Aortography is a desirable but not essential step in investigation, and is performed by introducing a catheter into a radial artery and passing it up until it reaches the proximal part of the aorta. A contrast medium such as diodone is then rapidly injected, radiographs are taken, and an accurate delineation of the character and anatomical position of the stenosis is thereby obtained. In the rare instances in which there is a long occluded segment, or in which an aneurysm has developed proximal to the coarctation, it is clear that end-to-end anastomosis is out of the question and therefore grafting is required. The operation should never be undertaken if suitable grafts are not available. Similarly all other provision should be made for reducing the difficulties and dangers of a taxing and formidable operation. These hazards are primarily those concerned with hæmorrhage from the tortuous, enormously distended, and often very friable vessels of the collateral circulation; any measure reducing the pressure and blood flow in them contributes greatly to the ease and safety of operating, so that induced hypothermia or hypotension is desirable. An advantage of hypothermia is that it permits longer and safer occlusion of the aorta. At least 6 pints of suitable blood must be

## CHAPTER 49

### COARCTATION OF THE AORTA

THE cause of coarctation is unknown, but it seems to be connected with the closure and sclerosis of the ductus arteriosus soon after birth, for the stenosis in the "adult" type of coarctation is closely related to the point where the ductus joins the aorta. Presumably the fibrosis proceeds too far, involving the aortic lumen as well as the ductus, or it is misdirected, for the ductus itself remains patent in 10 per cent. of cases of coarctation. Coarctation is said to occur about once in 7,000 births and is four or five times commoner in boys than in girls. Most patients develop symptoms by the age of twenty-five years; the average age at death is thirty-two, and 90 per cent. are dead before fifty. About 25 per cent. of the patients live a reasonably long life with little or no disability, but another 25 per cent. succumb to bacterial endocarditis or aortitis, mostly in the third decade. A further 25 per cent. die from rupture of the aorta, and the remainder from the effects of hypertension—most from cardiac failure, and some from cerebral hæmorrhage. Rupture of the aorta and cerebral hæmorrhage are commonest in the second and third decades, whereas death from congestive failure tends to occur later. Without operation the prognosis is bad in 75 per cent. of cases, and as there are no means of determining the prognosis in a given case in infancy the arguments in favour of early surgical treatment are very strong. The circulation cannot sustain for long the hypertension proximal to the coarctation. Arteriosclerotic changes develop in the proximal aorta and in the cerebral vessels and have been observed as early as three-and-half years; furthermore, the longer hypertension exists, the less the chance that surgery will reverse it; and, finally, both for these reasons and because of the ever-increasing distension of the vessels of the collateral circulation, the operation becomes more and more dangerous, and less likely to succeed. The optimum time for surgery is between the ages of ten and fifteen years. Below five the infant thorax is inconveniently small; below ten there is doubt whether the lumen of the anastomosis will increase sufficiently with age, and after adolescence risks and difficulties begin to mount for the reasons given above. Thirty-five years probably represents the extreme upper age limit for operation, but it is better to avoid intervening in patients over thirty years. At any age, resection and anastomosis of the aorta is a lengthy, dangerous and difficult operation which should certainly never be undertaken except under the very best conditions and by very experienced and skilful thoracic surgeons. In these circumstances a mortality rate of about 7 per cent. is to be expected

pairs, below the coarctation to secure sufficient mobilisation of the distal segment. In addition a bronchial artery arising on the medial side must be tied and cut. Whereas it is desirable not to interfere with more of the collateral channels than is strictly necessary, it is a mistake to clutter up the operative field with bulldog clips on the intercostals for temporary occlusion, especially as it is assumed that a more adequate circulation is about to be established.

The aorta still remains fixed in the region of the coarctation where fibrosis is maximal. Tapes are placed round the aorta proximally and distally to this point and gentle traction on them helps to define the

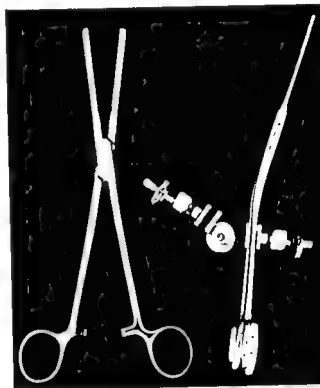


FIG. 333. Potts' coarctation clamp. The very fine teeth engage, but do not pierce the aortic wall; they, however, prevent slipping. The adjustable bracket is of great value in fixing the positions of upper and lower clamps.

stricture. The ligamentum arteriosum is joined very closely to the stricture, usually just proximal to it, and must be divided between ligatures. The coarcted area then becomes much more mobile. Dissection behind it should be kept close to the aorta to avoid damage to the thoracic duct, and the recurrent laryngeal nerve must also be protected on the medial aspect of the aorta.

As soon as adequate mobility has been secured Potts' clamps are placed on the aorta as far away as possible on each side of the constriction (Fig. 333). The latter is then excised for a length just exceeding 2 cm., leaving ends of approximately equal diameter and providing an anastomosis with a lumen not less than 60 per cent. of that of the normal aorta. As soon as they are cut the ends tend to retract towards the clamps, so it

at hand for transfusion. Blood loss throughout the operation is checked by weighing swabs and measuring all blood removed by suction. Thus accurate and prompt replacement is assured.

Homologous grafts previously obtained from suitable subjects, such as young victims of road accidents, and preserved by a freeze-drying technique, or prostheses of plastic cloth, Orlon or Vinyon, must be readily available even if there is reasonable assurance that the coarctation can be treated by resection and direct anastomosis. Apart from the element of doubt which remains until the cut ends are actually in apposition, it is always possible that a tear may occur in one or other segment and prove disastrous in the absence of an available graft.

The patient is placed in position for a left posterolateral thoracotomy and the incision, following the course of the fifth rib, is made in short segments at a time so that bleeding can be quickly controlled. As soon as the muscle layers are reached it is helpful to free the whole of serratus anterior as it presents in the wound so that it can be divided between light intestinal clamps placed round it above and below the line of the incision. These are released in turn so that the enlarged blood vessels in the muscles are secured and tied without excessive bleeding. The latissimus dorsi and other muscles are similarly treated and superficial blood loss is thereby greatly reduced. The pleural cavity is entered through the bed of the fifth rib which is resected from costotransverse joint behind to its cartilage in front; if a wider exposure is needed the posterior end of the rib above or below may also be divided. It is well for the surgeon to be comfortable and if possible seated, with the operating table tilted towards him so that he has unimpeded access to the interior of the thorax. The tortuous and distended collateral vessels are everywhere apparent, especially in the intercostal spaces. The mediastinal pleura is dissected back to lay bare the aortic arch and its branches. The marked pulsation of the aorta proximal to the coarctation is a striking contrast to the lack of pulsation in the distal segment which is, however, sometimes dilated and thin walled. The actual point of stenosis may be difficult to see at first, but can readily be palpated, usually just distal to the left subclavian artery which is often not much smaller than the aorta. Mobilisation is begun around the subclavian artery and carried upward and also behind the aortic arch. As a rule no intercostal branches arise from the arch, though if the point of coarctation is more distal than usual a pair of intercostal arteries may have to be isolated, tied and cut. The dissection of the intercostals call for special care, for not only are they enormously distended and tortuous but they are also very friable, especially close to the aorta itself from which they may be torn. Such an accident may be disastrous and the bleeding from it is severe. Blood rapidly fills the thorax and is very difficult to control. It is better, therefore, to dissect out these intercostals with care and deliberation at some distance from the aorta. Usually it is necessary to ligate and cut at least one, and sometimes two

**SECTION V**  
**NEUROSURGERY**  
by  
**LESLIE OLIVER**

**CHAPTER 50**  
**HEAD INJURIES**

**HEAD injuries** may be caused by violence applied directly to the head or indirectly through the spine by falls on the feet. Occasionally the brain is damaged by an acute rise in venous pressure in crush injuries of the chest. The significance of head injuries depends on the damage suffered by the brain, the complications which arise, and sometimes the unsightly deformities produced.

**Concussion.** The syndrome of concussion is characterised by sudden unconsciousness, varying from brief amnesia to prolonged coma, produced by violence applied directly or indirectly to the head, especially when rapid acceleration or retardation of the head occurs. Immediate examination of the unconscious patient reveals dilated and inactive pupils, flaccid paralysis of all voluntary muscles, absence of reflexes, pallor, a feeble pulse, a subnormal temperature and shallow respiration. During recovery, vomiting usually occurs and there is a brief pyrexia (99° to 100° F.). All neurological signs disappear when consciousness returns. There is usually loss of memory for the accident and a variable time preceding it (*retrograde amnesia*). After apparent recovery of consciousness there is also a period of loss of memory (*post-traumatic amnesia*), during which the patient is in a state of *post-traumatic automatism*. The time taken for complete restoration of consciousness is approximately proportional to the severity of the head injury and should therefore always be recorded. Diminished power of concentration, emotional instability, insomnia, bad dreams, headache and dizziness commonly follow concussion (*post-concussion syndrome*).

**Contusion of the Brain.** Contusion is characterised by multiple petechial hæmorrhages, damaged brain cells and œdema. It may be found at the site of injury and/or on the opposite side (*contre-coup injury*).

**Laceration of the Brain.** There is macroscopical destruction of tissue with surrounding contusion. Again, the lesion may be at the site of

is desirable to leave as wide a margin as possible for suturing. In most cases the ends can now be brought together with minimal tension and a direct anastomosis performed. This method is best, and is nearly always possible in children, but if the gap is too wide, or undue tension is likely, a graft or a prosthesis must be used. The exact fate of these is uncertain, but many surgeons have now employed them over considerable periods with excellent results. Gross found that a graft or prosthesis was required in 23 per cent. of patients over the age of twenty-one. During the anastomosis one assistant controls the two clamps, so that the cut ends of the aorta are in constant apposition; this is made easier by the use of a bracket which fixes the clamps to one another by screw grips (Fig. 333). The anastomosis itself is carried out with silk (3/0 to 4/0), beginning medially with a knot on the outside and continuing as an everting mattress suture, interrupted and tied at three or four points round the circumference. The posterior part is done from inside the lumen, left loose, and pulled tight when it is complete. It is a little more difficult to do this with a mattress than with an ordinary running suture such as some surgeons prefer, but the latter is more apt to cut when the edges are approximated. Tubbs has pointed out the advantages of placing the clamps vertically instead of horizontally so that the whole of the suturing can be done externally and each stitch pulled tight as it is made. When the anastomosis is complete, both proximal and distal clamps are removed together and the suture line is inspected. Any oozing soon stops if firm pressure with a swab is applied, but an actual leakage calls for the insertion of a further mattress stitch or two. The chest is closed with a water-sealed drain after re-inflation of the lung.

Post-operatively, the brachial blood pressure falls and that in the legs promptly rises. The femoral and popliteal pulses appear. In most older patients, however, some degree of hypertension may persist in the upper part of the body, perhaps due to long-standing changes in the vessels themselves, particularly the terminal arterioles. No special post-operative care is necessary, but anticoagulants, which might promote bleeding from the suture line, or needling of the chest which involves the risk of puncturing one of the large anastomotic vessels must be avoided.

the larynx with the aid of a laryngoscope, and the fluid aspirated. Collapse of the lung calls for immediate bronchoscopy and unplugging of the affected bronchus. There should be no hesitation in performing tracheotomy if frequently repeated bronchial aspiration is necessary, cyanosis is present, or pulmonary infection is established. Bronchopneumonia will often subside soon after tracheotomy. Indwelling endotracheal tubes are unsatisfactory for they become blocked with secretions, and cedema or ulceration of the pharynx and larynx tends to occur. When the best possible airway is established oxygen is administered. Penicillin (one million units in twenty-four hours) is injected intramuscularly as a further prophylaxis against pulmonary infection. *It should be remembered that pulmonary oedema may be caused by compression of the brain-stem.*

It is sometimes forgotten that unconscious patients have to be adequately fed. If unconsciousness persists for more than twelve hours, and swallowing (tested with a swab soaked in water) is absent, a medium-size well-lubricated stomach tube is passed through the patient's mouth, which is held open with a gag. The tube is guided into the back of the pharynx by the nurse's index and middle fingers, and then passed into the stomach. A Ryle's tube is unsatisfactory for deeply comatose patients as it curls up in the pharynx. *Before feeds are given, air is injected into the tube with the aid of a syringe, and the arrival of air bubbles in the stomach is detected by a stethoscope placed on the upper abdomen. No liquid of any kind is injected until the above test has proved the tube to be in the stomach. All other tests are unreliable and therefore dangerous.* A qualified nurse must carry out or supervise the above procedures. The medium-size stomach tube required for deeply comatose patients has to be passed for each feed on account of its size. Before each feed, the temperature of the liquid is taken and the stomach is aspirated to see that gastric retention is not occurring. When swallowing returns, the tube is no longer required.

Two litres of fluid and 2,500 calories are required in twenty-four hours, and vitamins should also be given. Nowadays a balanced diet in semi-liquid form can be given through a gastric tube by preparing the food in a mixing machine with a mincing and liquidiser attachment (Fig. 334). Constipation should be treated with enemas.

**Metabolism in Traumatic Coma.** Often there is an increase in the blood urea. The plasma protein tends to rise with recovery. A transient glycosuria (usually of low renal threshold type) occurs in about a third of patients who remain unconscious for twelve hours or more, but hyperglycaemia is rare.

**Hyperchloraemia** with hypochloruria may occur. It tends to be related to damage to the undersurface of the frontal lobes (ap. Lewin). The kidneys fail to excrete sodium and chloride ions. Sodium chloride should be restricted until normal plasma levels are obtained. *Hypochloraemia* with hyperchloraemia occurs only in elderly patients about a week after the



impact and/or on the opposite side. Either contusion or laceration may give rise to localising neurological signs, depending on the part of the brain involved. Laceration and contusion of the frontal and temporal poles are frequently observed in fatal cases. Localised damage to the brain is usually, although not necessarily, associated with concussion.

**Clinical Examination.** Both for medical and legal reasons a thorough examination should be carried out and the observations carefully recorded without delay. All bruises and wounds should be described and measured, and if there is any doubt about their cause they should be photographed before their appearance is altered by time or surgery. Bruises are particularly important in road accidents in helping to reconstruct the mode of injury. The size of a bruise depends on several factors other than the violence of impact (age, sex, texture of skin and presence or absence of abnormalities of the blood—e.g. hæmophilia). Therefore the extent of a bruise may not be proportional to the injuring force. Wounds vary according to the objects producing them. Thus blunt objects and hard surfaces cause splitting of the skin and contusion. Rounded objects cause stellate wounds. Sharp edges cause clean cut wounds without contusion. Choppers cause incised wounds with contusion. The presence or absence of dust, dirt, or sand in a wound should be recorded.

A neurological examination commensurate with the patient's degree of consciousness should be carried out immediately. This original examination is the standard for comparison if suspicion of complications arises. The suprapubic region should always be palpated for bladder distension, which is a common cause of restlessness. A thorough search should be made for associated injuries. Inability to move a limb is sometimes caused by a fracture and not by paralysis. Occasionally long tract signs are caused by associated spinal injury. Injuries of abdominal or thoracic viscera should be looked for although they occur somewhat rarely with head injuries in peace-time.

**Management of the Unconscious Patient.** During unconsciousness the patient is nursed in the lateral position, and is turned from one side to the other every two hours. The lateral position allows blood and secretions to flow from the mouth, and turning ensures that each lung is drained alternately. It is dangerous to raise the head and shoulders during unconsciousness if the cough reflex is absent, for blood, secretions and vomit may obstruct the respiratory passages. It is also dangerous to nurse patients supine, for in this position the tongue obstructs the airway. When coughing and swallowing return, patients are nursed in Fowler's position to discourage intracranial venous bleeding and œdema of the brain. The brain has remarkable powers of recovery after injury provided a good airway is maintained and hypostatic pneumonia prevented.

A trained nurse must be present all the time to suck out the pharynx. Separate catheters are used for the nose and the mouth. If fluid collects in the pulmonary tree despite these measures, a catheter is passed through

compensated for by periodic hypopnœa (Cheyne-Stokes' breathing). Otherwise tetany develops.

*Renal uræmia* sometimes occurs although at autopsy the kidneys are apparently normal. Protein should be withdrawn from the diet and caloric requirements maintained with peanut oil (peanut oil 100 gm. and glucose 400 gm. emulsified in a litre of water, given every twenty-four hours, provide 2,500 calories).

The lesson to be learnt from these metabolic changes is that, although appropriate treatment (see also under Reactions to Injury) should be given, considerable circumspection is required in the use of intravenous electrolytes, plasma substitutes, and dehydration therapy. The latter is better avoided in most head injuries, for cerebral œdema cannot be diagnosed by clinical examination.

**Extradural Hæmatoma** (Syn. Middle Meningeal Hæmorrhage). The hæmorrhage may originate from the middle meningeal vessels, the venous sinuses, or from diploic veins. It is usually, but not always, associated with a fracture of the skull, or diastasis (separation of sutures). There is nearly always a visible scalp lesion overlying an extradural hæmatoma. The classical syndrome is as follows. After an initial improvement, the level of consciousness declines, the pulse rate increases, then it may fall below normal (bradycardia), and finally it becomes fast and weak. The classical slow pulse, although of considerable diagnostic importance, is found less often than a fast one. Occasionally, in the early stages of bleeding, the homolateral pupil is constricted. In the *late* stages of compression, whether initially constricted or not, the pupil dilates and the direct and consensual light reflexes are lost. The term *Hutchinson's pupil* applies to these phenomena. By the time dilatation of the pupil has occurred there is an advanced degree of compression. *Therefore the diagnosis should be made before this sign appears.* Sometimes when the patient regains consciousness after operation, dilatation of the pupil is found to be accompanied by ptosis and paralysis of the external ocular muscles supplied by the oculomotor nerve. The complete oculomotor palsy is caused by pressure on the nerve by herniation of the medial border of the temporal lobe between the tentorium cerebelli and the brain stem. Bilateral dilatation of the pupils is a sign of imminent death. Unilateral or bilateral abducens palsy may result from raised intracranial pressure (Fig. 335).

Pyramidal signs may be found on the opposite side to the hæmatoma. *Homolateral* pyramidal signs are caused by displacement of the brain stem away from the side of the hæmatoma and consequent indentation of the contralateral cerebral peduncle by the edge of the tentorium. The pyramidal fibres of the affected peduncle cross below in the decussation of the pyramids to the same side as the lesion. If signs of an extradural hæmatoma are delayed for a few days some *papillædema* may be found.

*Only about half the patients with extradural hæmorrhage develop the*

injury. The tongue is dry, the cheeks are sunken and intra-ocular tension is low. The syndrome resembles that of Addison's disease, but there is no pigmentation and the plasma potassium level is normal. The sodium chloride intake should be increased, sometimes to 15 gm. a day (in capsules), and deoxycorticosterone acetate, adrenal cortical extract

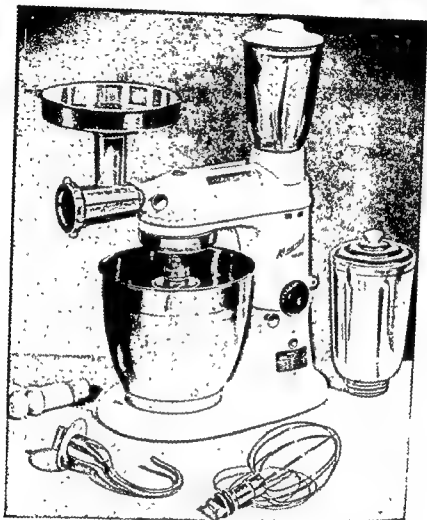


FIG. 334. The Kenwood mixing machine.

("Eucortone"), and cortisone may be tried. These measures do not, however, usually restore the sodium and chloride levels to normal.

*Water deficiency*, which is aggravated by pyrexia or hyperpncea, is shown by dryness of the mouth and tongue, loss of normal texture of the skin and reduced intra-ocular pressure. Both plasma and urinary levels of sodium and chloride are raised. The condition may be due to neglect, difficulty in administering fluids, or "therapeutic" dehydration.

*Respiratory alkalosis* occurs with prolonged hyperpncea. The plasma bicarbonate is lowered and the urine is alkaline. Alkalosis is usually

slow pulse, rising blood pressure and slowing of respiration) tend to occur without any previous recovery of consciousness and therefore without cerebellar signs. A fracture across the groove of the transverse or sigmoid sinus may be the only indication of a hæmatoma in the posterior fossa. The acute type of posterior fossa hæmatoma tends to be associated with severe brain damage; hence the mortality is high. The *subacute form* shows itself several days after injury. Manifestations of medullary failure (*vide supra*) follow a period of improvement in the state of consciousness and, in conscious patients, cerebellar signs may be found. The *chronic form* shows itself some months after injury and is usually mistaken for a posterior fossa tumour. Persistent headache, vomiting, papilloedema and cerebellar signs occur. Ventriculography shows hydrocephalus and displacement of the fourth ventricle away from the side of the lesion.

The mortality of extradural hæmorrhage is still 50 per cent. ! This deplorable state of affairs is mostly due to lack of recognition of its rapidly lethal nature. Any decline in level of consciousness or development of new neurological signs should lead to immediate action. If a patient in traumatic coma does not rapidly improve a neurosurgeon should be consulted without delay. *Lumbar puncture* contributes nothing to the diagnosis and *may kill the patient* by causing a temporal or cerebellar pressure cone (the lumbar cerebrospinal fluid pressure is often raised in head injuries when there is no space-occupying lesion, and, conversely, as the result of cerebellar coning, the pressure may be normal or even subnormal in the presence of a large hæmatoma).

*Operation.* Extradural hæmorrhage is one of the most rapidly lethal conditions in surgery. Mere suspicion of it should lead to diagnostic burr-holes without delay. The anaesthetist is responsible for maintaining a good airway. Opiates and barbiturates are forbidden, for the former depress the already threatened or affected vital centres, and the latter depress still more the level of consciousness. Many patients are too restless for local anaesthesia alone. Sometimes coma is so deep that no anaesthetic of any kind is required.

A burr-hole is made over the suspected site of the hæmatoma (the precise measurements given in older textbooks of surgery have no value). When the diagnosis is correct, blood is seen as soon as the inner table of the skull is perforated. As much of the hæmatoma as possible is removed by suction and the extent of the dural stripping ascertained with the aid of a malleable probe. A bone flap is then planned accordingly (Fig. 336). Bleeding from the middle meningeal vessels is controlled by electro-coagulation, silk sutures, metal clips or occasionally by plugging the foramen spinosum with the pointed end cut from a sharpened match-stick. When metal clips are used, a small incision is made in the dura parallel to the vessel so that the clips can be applied. Bleeding from dural sinuses or veins is arrested by application of muscle "stamps" or

*classical syndrome described above. Thus in many cases there is no recovery of consciousness before the onset of cerebral compression, and in childhood an initial period of coma seldom occurs; more often children are momentarily dazed and later become drowsy and finally comatose. They sometimes have convulsions. Also, wide separation of the edges of the fracture or sutures, together with the thin elastic scalp, sometimes allows much of the blood to escape under the pericranium or galea to form*



FIG. 335. Bilateral abducens paralysis (convergent squint) associated with a left-sided extradural haematoma. Photograph taken a few days after evacuation of the haematoma.

a large external haematoma. Thus spontaneous decompression may occur. In infants, blood lost in the formation of an extradural haematoma may cause severe anaemia and profound shock.

About a third of all extradural haematomas occur other than at the classical temporal site (e.g. frontal, parietal and cerebellar). The location of extradural haematomas is often revealed by bruising of the scalp, the position of the fracture and neurological signs.

*Posterior fossa extradural haematomas* are likely to be missed because of their rarity. They are caused by lacerations of the transverse or sigmoid sinuses. In the acute form, occurring within twenty-four hours of the injury, signs of compression of the medulla oblongata (repeated vomiting,

removal of bone with rongeurs. In this way the site of the hæmorrhage—usually a lacerated transverse or sigmoid sinus—can be exposed (Fig. 384).

**Subdural Hæmatoma.** There are two types of hæmatoma occurring between the dura and arachnoid—acute and chronic. An *acute subdural hæmatoma* is frequently found in severe head injuries. The collection of blood is usually small and is associated with other more serious lesions, e.g. laceration or contusion. In these circumstances it contributes little or nothing to the clinical picture, but if discovered when exploratory burr-holes are made the blood should be removed. Rarely a *large* subdural hæmatoma arises from torn meningeal vessels which have bled under the dura mater instead of over its outer surface, or a subdural hæmatoma of considerable size may be caused by spontaneous hæmorrhage from an intracranial aneurysm. In both cases, the blood is aspirated through a rubber catheter passed into the subdural space through a burr-hole, and the causal lesion appropriately treated.

A *chronic subdural hæmatoma* may follow a minor head injury occurring a few weeks previously, although not infrequently there is no history of injury. The cause is thought to be tearing of veins passing between the cerebral cortex and the venous sinuses. The hæmatoma becomes surrounded by a capsule. In adults, chronic subdural hæmatoma is sometimes bilateral whereas in early childhood it is *almost always* bilateral. The diagnosis is made when a patient develops evidence of raised intracranial pressure, and sometimes also lateralising signs, a few weeks after a head injury. Occasionally the course is fulminating or there may be merely a vague mental disturbance reminiscent of the dementia caused by a frontal tumour. In fact, intracranial tumour is the most likely diagnosis when there is no history of head injury. When the lesion has been overlooked, the capsule tends to be thick, and indeed the hæmatoma may become completely organised into a fibrous mass with patches of calcification.

In early childhood, birth trauma is held to be a major factor in the ætiology. A history of other types of head injury is more often lacking in children than in adults, and there is no characteristic clinical picture in the majority of cases. Thus *infants* may show nothing more than restlessness and bad temper, or there may be generalised convulsions (*the commonest symptom of the lesion in early life*) and vomiting. There is pyrexia in more than half the cases and bulging of the fontanelle in somewhat less than half. The circumference of the head may be 2 or 3 inches more than average. Retinal hæmorrhages are quite common but papilloedema is rare. The tendon reflexes may be exaggerated, but paresis of limbs is found in only a small proportion of cases. The manifestations in early life are thus vague, and therefore subdural hæmatoma should be one of the conditions considered when a child is not thriving.

**Investigations.** Plain radiography usually gives no evidence of a chronic

gelatin sponge, together with elevation of the patient's head. Bleeding from bone is controlled with bone-wax.

When an extradural hæmatoma is not revealed by the first burr-hole the dura mater is opened. If an acute *subdural* hæmorrhage is found, a small rubber catheter is passed beneath the dura mater and the blood aspirated. Some of it may have to be washed out with normal saline injected through the catheter. If neither an extradural nor a subdural hæmatoma is found, but the brain is bulging, a small stab is made in an avascular part of the exposed cortex with a pointed tenotome, and a fine

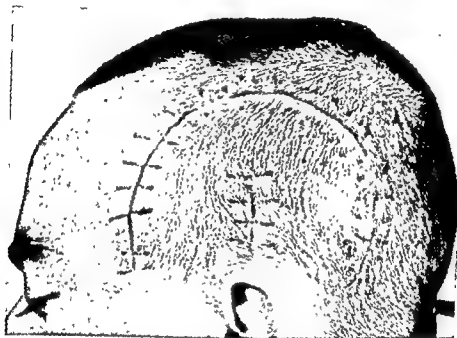


FIG. 336. Incision for an extradural hæmatoma, at the classical site in the temporal region. The diagnosis had first been confirmed by means of a burr-hole made through the small incision just above the pinna.

brain cannula passed into it. An intracerebral clot may thus be discovered. After elevation of a bone flap, the surface of the brain is incised in a relatively silent area and the clot evacuated by means of low-pressure suction.

If the first burr-hole reveals no evidence of an extradural, subdural or intracerebral hæmatoma, burr-holes are made at other sites on the same side. Thus if the first burr-hole was made in the temporal region, others are made in the frontal and parietal regions. Failure to find a hæmatoma on one side should always lead to burr-holes on the other side. When there is occipital bruising and a fracture overlying the transverse or sigmoid sinuses there is the possibility of an extradural hæmatoma in the posterior fossa, burr-holes should therefore be made in this region. The exposure can be extended by lengthening the burr-hole incision and

clear or yellow fluid of high protein content may occur, with or without a history of head injury (Fig. 337). They cause increased intracranial pressure. Some cases follow meningitis, especially that caused by *H. influenza*. The traumatic hygromas are assumed to follow tears in the arachnoid. Hygromas, whether traumatic or inflammatory, are frequently bilateral. Treatment is the same as for subdural hæmatomas, including removal of any capsule which may have formed.

**Subarachnoid Hæmorrhage.** Red cells are commonly found in the



FIG. 337. A giant left-sided hygroma demonstrated radiologically after displacement of some of the fluid by air.

cerebrospinal fluid after head injuries, but severe hæmorrhage revealed to the naked eye by lumbar puncture is rare in adults. There are usually the classical signs of meningeal irritation, viz. neck stiffness and Kernig's sign (other causes of neck stiffness are *meningitis*, raised intracranial pressure, fracture of the occipital bone and fracture or dislocation of cervical vertebræ). *Tentorial tears*, due to difficult labour, commonly cause subarachnoid hæmorrhage. The fontanelle is tense and there is neck stiffness. White asphyxia is common. *In early life*, subarachnoid hæmorrhage calls for daily lumbar puncture and aspiration of cerebrospinal fluid to reduce intracranial pressure and to prevent the formation of meningeal adhesions and subsequent external hydrocephalus.



subdural hæmatoma, although in long-standing hæmatomas in children there may be enlargement of the calvarium on the affected side in unilateral cases, and also elevation of the lesser wing of the sphenoid (as seen through the orbit in an anteroposterior view of the skull). There may also be separation of the sutures (diastasis). Occasionally calcification of the hæmatoma is shown radiologically. Electroencephalography shows electrical "silence" or low potentials over the hæmatoma. When the diagnosis of subdural hæmatoma is made the appropriate investigation is exploration by means of burr-holes. In doubtful cases carotid angiography is sometimes carried out. In the presence of a hæmatoma, the vascular tree is seen to be displaced to the opposite side. A frontal hæmatoma causes displacement of the corresponding anterior cerebral artery towards the opposite side and downward deflection of the middle cerebral artery and its branches. Ventriculography is not performed deliberately when a subdural hæmatoma has been diagnosed, but it may reveal the presence of one when the investigation is carried out for a suspected intracranial tumour.

*Treatment.* In adults a chronic subdural hæmatoma is drained through burr-holes. The dura mater is opened and the outer membrane of the hæmatoma is punctured (the surgeon is careful not to mistake a large cortical vein for a hæmatoma). A rubber catheter is passed into the hæmatoma. If the altered blood is in the fluid state, much of it will flow out. The remainder is evacuated by saline irrigation through the catheter. Burr-holes are then made on the opposite side, for subdural hæmatomas are frequently bilateral. Further irrigation of the cavity may be required in a few days if the patient's condition does not improve, or deteriorates. If the hæmatoma is composed of clotted blood it is necessary to elevate a bone flap to remove it.

In children, especially during infancy, the management is different, for sudden evacuation of large subdural hæmatomas has a high mortality. Treatment begins with aspiration of not more than 10 ml. of blood daily on alternate sides. (N.B. Subdural hæmatomas are bilateral in children in the majority of cases.) Aspirations are carried out through the coronal sutures at a different point each day and well away from the midline. After about a week when the patient's condition is sufficiently improved, burr-holes are made to see whether or not a membrane has formed. It is particularly important in children to remove all the membrane of subdural hæmatomas to prevent cortical atrophy. Removal is carried out after elevation of a large osteoplastic flap. It is of the utmost importance immediately to replace all blood lost during operation. After operation, repeated aspiration of the subdural space is often necessary. During the early years of life, when the brain is growing rapidly, it is vitally important to avoid missing a subdural hæmatoma which, if untreated, causes marked dementia.

**Subdural Hygroma (Syn. Subdural Hydroma).** Subdural collections of

sometimes occurs on the outside of the skull under the galea when a fracture communicates with the subarachnoid space. If the meningocele does not subside spontaneously, the dural defect should be repaired. Elevation of a bone flap is necessary.

**Cephalhæmatoma.** A large *subperiosteal* hæmatoma may be caused by trauma at birth irrespective of the use of forceps (Fig. 338). It is observed two to three days after birth. The commonest site is the parietal region. There is usually no fracture, for the hæmatoma is caused by stripping of the periosteum (pericranium) from the bone. The extent of the hæmatoma is limited by the skull sutures. It fluctuates, and palpation gives the *false* impression of a depressed fracture. A cephalhæmatoma is easily distinguished from a caput succedaneum, for the latter is present at birth; is not limited by the sutures; and does not fluctuate. Cephalhæmatomas usually resolve spontaneously in a few weeks. Aspiration is therefore unnecessary when there is no doubt about the diagnosis, and, furthermore, aspiration, if asepsis is imperfect, may cause *intractable* infection of the hæmatoma.

**Subgaleal Hæmatoma.** Linear fractures, especially those affecting the parietal bone in infancy, frequently cause large subgaleal hæmatomas. They are not limited by the attachment of the pericranium at the sutures. These hæmatomas may be regarded as spontaneously decompressed extradural hæmorrhages. Many of them subside, but some need aspiration. Craniotomy may sometimes be necessary to deal with the bleeding vessels.

**Traumatic Arteriovenous Aneurysm.** See under aneurysms.

**Types of Fracture.** Fractures may be either open (compound) or closed. The aim is always to convert open fractures into closed ones *as soon as possible* to prevent entry of organisms. *Fissure (linear) fractures* are caused by impact of the head against a flat surface. They are single, multiple or branching, and may involve the vault or base of the skull, or both. *Separation of sutures* (diastasis) is a common injury in children, although not confined to them. It may be continuous with linear fractures. *Stellate fractures* are caused by blows with blunt rounded objects and therefore affect the vault. *Depressed fractures* arise in many ways. Thus they can be caused by the impact of a flat surface, a protruding object, or by a missile. In early life, a depressed fracture may be caused by obstetric forceps. This is not to be confused with the occasional impression made in the skull of the foetus by the promontory of the mother's sacrum (Fig. 339). The indications for operating on depressed fractures are : (a) When they are *open*. Bone fragments in the brain are removed but surface fragments are retained, and the normal contours restored as much as possible. Hair and foreign bodies, which are liable to be trapped under and between fragments, are removed. The wound is closed, if necessary, by mobilising a scalp flap. (b) When in a *closed* injury the radiograph shows that fragments of bone must be in the brain

**Traumatic Intracerebral Hæmorrhage.** This lesion has already been mentioned under *extradural hæmatoma*. It is rare in closed head injuries but is common in missile wounds. It may be discovered when burr-holes are made in search of an *extradural hæmatoma*, or may show itself quite suddenly after an interval (late traumatic apoplexy). The clot is evacuated after elevation of an osteoplastic flap and incision of the cerebral cortex.

**Traumatic Porencephaly.** An area of cerebral softening following contusion may cause *cavitation of the brain* (porencephaly). The cavity may be cystic or may communicate with one of the lateral ventricles or the subarachnoid space. A traumatic cyst may gradually expand and cause



FIG. 338. A typical cephalohæmatoma in the left parietal region.

raised intracranial pressure; treatment consists in establishing a communication with the corresponding lateral ventricle. Care should be taken to avoid confusion with gliomatous cysts.

**The Pseudohæmatoma Syndrome.** Diagnostic burr-holes are often made in cases in which there is a classical syndrome of *extradural hæmatoma*, but no hæmatoma is discovered. Some patients recover spontaneously but many die. At autopsy one or more of the following lesions may be found: (a) Severe laceration and contusion of the cerebral hemispheres; (b) cerebral oedema; or (c) hæmorrhage in the brain stem. The cause of the initial improvement in these patients is obscure. When exploratory burr-holes do not reveal an *extradural*, *subdural* or *intracerebral hæmatoma*, a *subtemporal decompression* should be done if the brain is swollen (i.e. oedematous).

**Traumatic Meningocele.** A cystic collection of cerebrospinal fluid

## OPEN HEAD INJURIES

The opening may be through the scalp or through the mucosa of the nasal sinuses or the middle ear. Penetration of the dura mater greatly increases the chances of intracranial infection. Open head injuries are commonly associated with concussion but sometimes occur without it.

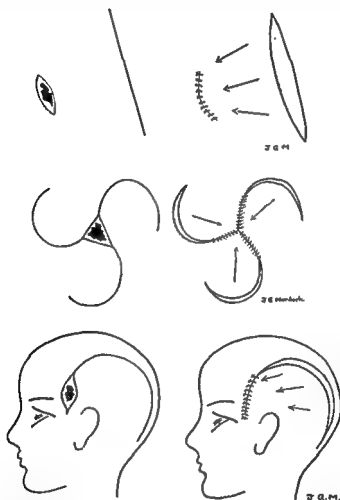


FIG. 340. Methods of covering open fractures of the skull when the overlying scalp is deficient—converting an open fracture into a closed one.

**Treatment.** Part of the scalp may be missing, or so badly damaged that some sacrifice of tissue cannot be avoided, although the aim should always be to conserve as much scalp as possible. Open (compound) fractures must be closed by one of the methods of scalp-suture illustrated in Fig. 340 to shut out infection from the brain and meninges. An essential part of the technique is to increase the mobility of the scalp by wide undercutting between it and the underlying pericranium. If scalp has to be

and are therefore likely to cause gliosis and subsequent epilepsy. (c) When abnormal neurological signs correspond with the site of the depression, e.g. paresis of an upper extremity or aphasia. (d) When in a closed injury the patient demands operation for cosmetic reasons, as in depressed frontal fractures. (e) In infants when the depression does not show signs of gradual spontaneous restitution.

A large number of closed depressed fractures which do not cause neurological signs need not be elevated. Indeed it may only lead to troublesome hæmorrhage, especially if the fracture involves the course of

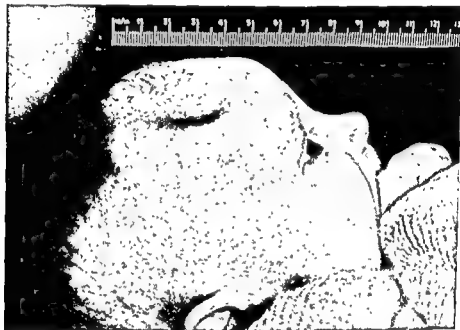


FIG. 330. A frontal impression (not a fracture) in a new-born infant. The mother had a deformed pelvis resulting from tuberculosis of the left sacro-iliac joint. After twelve months the impression had almost disappeared without treatment.

the middle meningeal vessels or the venous sinuses. Depressed fractures compress the brain immediately beneath them. Therefore if signs of generalised brain compression develop in the presence of a depressed fracture they are caused by an associated extradural or subdural hæmatoma or cerebral oedema.

Elevation of depressed fractures can be difficult. It is accomplished by making burr-holes close to the margin of the fracture for the passage of elevators, but often bone has to be nibbled away before the fragments can be mobilised.

**Tangential Wounds.** Severe neurological disturbances may occur in missile wounds of the scalp without penetration of the bone or dura mater. Operation may reveal cerebral contusion, cerebral laceration, extradural hæmatomas, subdural hæmatomas or subcortical hæmatomas.

## OPEN HEAD INJURIES

The opening may be through the scalp or through the mucosa of the nasal sinuses or the middle ear. Penetration of the dura mater greatly increases the chances of intracranial infection. Open head injuries are commonly associated with concussion but sometimes occur without it.

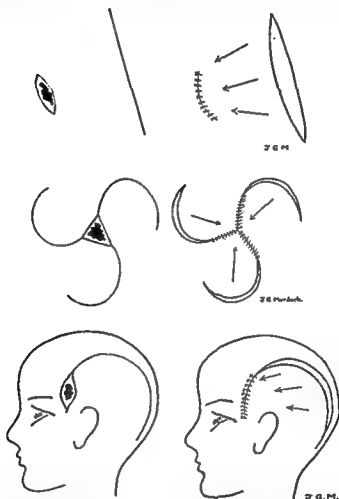


FIG. 340. Methods of covering open fractures of the skull when the overlying scalp is deficient—converting an open fracture into a closed one.

**Treatment.** Part of the scalp may be missing, or so badly damaged that some sacrifice of tissue cannot be avoided, although the aim should always be to conserve as much scalp as possible. Open (compound) fractures must be closed by one of the methods of scalp-suture illustrated in Fig. 340 to shut out infection from the brain and meninges. An essential part of the technique is to increase the mobility of the scalp by wide undercutting between it and the underlying pericranium. If scalp has to be

moved to cover an open fracture, it follows that a bare area of *intact* bone must be left somewhere, although most textbook diagrams give the opposite impression. The planning of relieving incisions and flaps demands careful thought, for it is easy to underestimate the requirements. Straight incisions radiating from the defect are valueless.

Accessible bone fragments and foreign bodies which have been driven into the brain must be removed, otherwise a brain abscess will develop. Radiography should be used before closure of the wound to check their

removal. Missiles are removed only if easily accessible, for they do not readily cause infection. Disintegrated brain is removed by means of low-pressure suction, and strict hæmostasis is attained by neuro-surgical methods. The dura mater is sutured. Dural defects are repaired with grafts of temporal fascia, pericranium, galea or fascia lata. Wounds which have been contaminated with dirt are sprinkled with penicillin-sulphadiazine powder, but care must be taken not to administer an excessive dose of the latter drug by this means. Penicillin should also be given by intramuscular injection.

**Small Penetrating Wounds.** At the site of entry of a *bullet* the inner table of the skull is more fragmented than the outer table, whereas at the site of exit the outer table is more

fragmented than the inner table. Small penetrating wounds produced by sharp objects tend to be neglected, and thus patients may not be sent to hospital until a brain abscess or meningitis has developed (Fig. 341).

**Cerebrospinal Fluid Rhinorrhœa.** The diagnosis of cerebrospinal fluid rhinorrhœa, when copious, is usually easy. However, it may occasionally be confused with allergic rhinitis. Confirmation of the diagnosis is obtained by a positive test for sugar in the fluid, an increase in rhinorrhœa on jugular compression, and the radiological demonstration of a fracture involving the frontal, ethmoid or sphenoid sinuses. Usually rhinorrhœa begins within forty-eight hours of the injury, but the onset may be delayed for weeks, months or years. Plain radiography sometimes reveals air in the subdural space, in the cerebrum (aerocoele), or in the ventricles. Anosmia is commonly associated with cerebrospinal fluid rhinorrhœa, and is very suggestive of a fracture of the ethmoid region. The fluid usually, but not always, flows from the nostril on the side of the fistula. Sometimes



FIG. 341. Abscess at the site of entry of a pencil into the orbit. The pencil entered the brain through the roof of the orbit and caused an abscess in the frontal lobe. The cerebral abscess was successfully treated by the aspiration method described in Chap. 51.

instead of rhinorrhœa there is effusion of fluid into the orbit causing a fluctuating swelling above and medial to the eye. According to *Calvert*, half the patients with cerebrospinal fluid rhinorrhœa develop meningitis.

*Treatment.* Patients are instructed not to blow their noses, and everyone approaching the patient wears an impervious mask. Penicillin given intramuscularly does not pass from the blood into the cerebrospinal fluid, and therefore is useless as prophylaxis against meningitis. Sulphadiazine is therefore given. In most cases, careful radiography shows the site of the fracture. The incidence of meningitis is so great that an attempt should be made to close the fistula in all cases, including those in which rhinorrhœa ceases spontaneously. A bifrontal intradural approach, with division of the superior longitudinal sinus at its anterior end, gives the best approach to what may be a difficult exploration. One or both olfactory tracts may have to be sacrificed. When the fracture involves the sphenoid sinus, the opening may be in the upper part of the anterior wall of the sella turcica or under cover of one of the optic nerves. When the fistula is found, it is covered with a piece of fascia lata. No sutures are required to fix the graft.

**Cerebrospinal Fluid Otorrhœa.** Intracranial infection may follow fractures of the petrous bone involving the middle ear. Operative repair of the fistula is rarely undertaken, for it is not known whether the internal opening is in the middle or posterior fossa. The same precautions against infection are taken as for cerebrospinal fluid rhinorrhœa. When otorrhœa is copious it is futile to instil antibiotics into the external meatus, for they are rapidly washed away. A large wad of sterile cotton wool is placed over the pinna with aseptic technique, and changed as required. The meatus must not be plugged.

**Injuries of Venous Sinuses.** A venous sinus may be torn by indriven bone fragments, missiles, the force causing a linear fracture, or transmitted violence in tangential wounds (with or without a fracture). In closed head injuries, laceration of a venous sinus is one of the causes of extradural hæmatoma.

The *superior longitudinal sinus* is the one most often injured. Except when the tear is well anterior, spastic paralysis of the lower limbs is commonly found. Sometimes the upper limbs are also involved. Parietal lobe sensory loss is common on one or both sides (astereognosis, loss of tactile discrimination, and loss of postural sense).

*Treatment.* Bleeding is reduced by gradual elevation of the patient's head, but great care is taken to avoid a negative pressure in the sinus. Depressed fractures which involve venous sinuses without causing hæmorrhage, neurological signs, or cerebral compression are left undisturbed, for interference is apt to cause torrential bleeding. When operation is indicated, bone fragments involving a sinus are removed last, if at all. Wounds of the prefrontal part of the superior longitudinal sinus or of one transverse or sigmoid sinus may be treated by double ligation.



Injuries in other situations are sutured and covered with grafts taken from the temporal muscle. It is an advantage to use muscle with temporal fascia attached. The graft is placed over the tear with the muscle facing inward, and the fascia on the outer side is sutured to the surrounding dura mater. The sutures of the graft should not be placed through the sinus itself. The patient's head is kept elevated during operation and also post-operatively, provided the cough and swallowing reflexes are intact.

**Cerebral Fungus.** A cerebral fungus is a protrusion of part of the

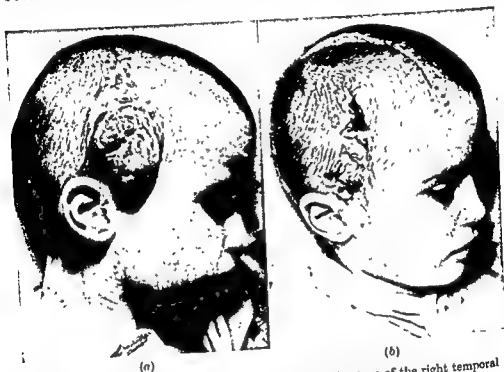


FIG. 342. (a) A cerebral fungus following an open fracture of the right temporal region. The dura mater had not been repaired and the scalp had been sutured in one layer only. (b) After operation. The fungus did not respond to conservative treatment. Therefore it was gently separated from the surrounding scalp (its principal attachment); the gap in the dura mater was closed with temporalis fascia, and the scalp closed by mobilising a large flap (Fig. 340).

cerebrum through an opening in the skull and its coverings, usually caused by trauma (Fig. 342). The injury is responsible for the opening, and resultant localised infection of the cerebrum, together with constriction by the adherent skin causes and maintains the protrusion. Sometimes bone fragments or foreign bodies prevent resolution of the infection. Raised intracranial pressure is not usually an aetiological factor, for in most cases the pressure is normal. However, occasionally there is a subjacent abscess causing or contributing to the protrusion. Cerebral fungus is commonly seen in warfare but is rare in peacetime. It results from delay and faulty technique in treatment of open head injuries. A cerebral fungus takes about a week to develop. At first it looks pale

and is covered with flakes of "lymph" and superficial sloughs. Granulations are seen at the junction of the scalp with the fungus. After a few weeks red granulations form over the surface of the fungus, and epithelialisation begins at the skin margin. Finally, after many weeks, if conservative treatment succeeds, the surface becomes covered with scar tissue.

*Treatment* consists of: (a) Administration of antibiotics to combat the localised encephalitis; (b) daily withdrawal of cerebrospinal fluid by lumbar puncture until the fungus is indrawn; (c) *careful separation of soft adhesions between the fungus and the surrounding skin*; (d) elevation of patient's head and trunk on pillows; and (e) restriction of fluids to 1,500 ml. in twenty-four hours. If these measures fail the fungus is gently separated from the skin and any deep attachments. Then the dura is repaired with fascia lata and the gap in the scalp covered by the methods previously described (Fig. 340). After successful treatment there is a strong tendency for neurological signs produced by protrusion of the cerebrum to clear up. With the use of antibiotics, cerebral fungus is rarely complicated by ventriculitis or meningitis, although there is often a pleocytosis (mostly lymphocytes) and a rise in protein level in the cerebrospinal fluid. Epilepsy occurs with the same frequency as with other types of open head injuries.

**Other Complications of Head Injuries.** *Epilepsy* (see under epilepsy, Chap. 57). *Diplopia*.—This is a fairly common complication. It is usually due to an abducens palsy which has a strong tendency to spontaneous recovery except when caused by a space-occupying lesion (Fig. 335). The exact mechanism of the ophthalmoplegia is unknown in most cases, although sometimes it is caused by displacement of the eye when fractures involve the orbit. *Facial Paralysis*.—This may occur with or without a demonstrable fracture of the petrous bone. Recovery occurs in most cases. When paralysis is incomplete, or delayed in onset, recovery is almost certain. Facial paralysis of the new-born infant, caused by forceps, always recovers. *Auditory Nerve Deafness*.—This may occur with or without a demonstrable fracture of the petrous bone. Permanent deafness usually follows. *Middle-ear Deafness*.—When conductive deafness follows a head injury there is usually blood in the middle ear. There is, however, no indication for myringotomy. Unless recovery occurs in a few weeks, deafness is usually permanent. *Anosmia*.—Loss of sense of smell occurs most often with frontal or occipital injuries—especially in association with fractures of the ethmoid. Thus it is commonly found with cerebrospinal fluid rhinorrhœa. *Injuries of the Optic Nerves and Chiasm*.—Damage to the optic nerves or chiasm may occur in closed or open head injuries. In the latter the lesions are caused by missiles or other penetrating objects. The injury usually affects one optic nerve, causing unilateral blindness, or affects the chiasm, causing bilateral blindness or bitemporal hemianopia. No recovery is to be

expected. *Visual field defects* follow penetrating injuries of the optic radiation in its course through the temporal and occipital lobes. *Hemiplegia*.—When hemiplegia is caused by contusion, extradural hæmatoma, subdural hæmatoma, or cerebral fungus, there is a strong tendency for recovery to occur after treatment, but when caused by penetrating injuries or intracerebral hæmorrhage, considerable spastic paralysis may remain. *Dysphasia or Aphasia*.—Partial or complete loss of speech may be caused by the same mechanisms responsible for traumatic hemiplegia. After closed head injuries, recovery is often complete, but after open injuries there may be permanent dysphasia or aphasia. Considerable benefit can be obtained from *speech therapy*. *Hypothalamic Disorders*.—Hypersomnia, insomnia, hyperthermia, diabetes incipidus, and acute peptic ulceration may follow head injury. Recovery tends to occur. *Psychosis*.—Personality changes are not uncommon after head injuries, but psychosis is very rarely attributable to injury.

## CHAPTER 51

### INTRACRANIAL SUPPURATION

**Extradural Abscess.** An abscess may form between the skull and the dura mater as a complication of mastoiditis, osteomyelitis of the skull (Fig. 343), or fracture of the skull (open to the exterior, or into an infected mastoid process or nasal sinus). The dura mater forming the inner wall of the abscess is inflamed and usually covered with granulations corresponding to the extent of the abscess (pachymeningitis). There is severe

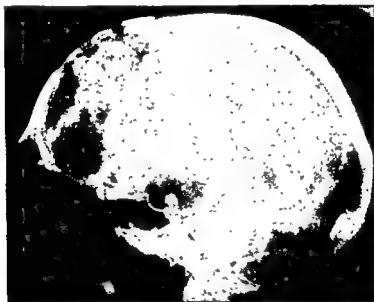


FIG. 343. Osteomyelitis of the frontal bone. Typical moth-eaten appearance. (Radiograph: Dr. J. M. Stewart.)

toxæmia and pyrexia. Localising neurological signs are sometimes found and depend on the site of the abscess. Rarely, osteomyelitis, blood-borne or arising from an overlying carbuncle, affects the suboccipital region causing an extradural abscess with cerebellar signs. The cerebrospinal fluid shows a moderate increase in white cells but contains no organisms; its pressure may be raised.

**Treatment.** When an extradural abscess is caused by osteomyelitis or mastoiditis, treatment of these diseases establishes the necessary drainage. When, however, an extradural abscess follows fracture of the skull, a burr-hole is made over the site of the abscess and a drainage tube inserted. Appropriate antibiotics are administered.

**Subdural Empyema and Abscess.** The synonym *purulent pachymeningitis* is a bad term, for it equally well applies to extradural abscess which is commonly associated with pachymeningitis secondary to overlying osteomyelitis.

*Subdural empyema* means a *widespread* collection of pus between the *dura* and *arachnoid* mater. It is an uncommon but serious condition which before the days of antibiotics was invariably fatal. The commonest cause is *osteomyelitis* of the skull following an acute exacerbation of, or operative interference in, inflammation of the nasal air sinuses. It may also be caused by blood-borne osteomyelitis of the skull, mastoiditis, or fractures of the skull which open to the exterior or into septic nasal sinuses or mastoid cells. There is marked toxæmia and pyrexia, with signs of raised intracranial pressure (headache, vomiting and papilloedema). The infection spreads widely in the *subdural* space producing contralateral hemiparesis, cortical sensory loss and hemianopia. If the infection involves the dominant cerebral hemisphere, *dysphasia* or aphasia may be found. Patients sometimes present with *grand mal* or Jacksonian epilepsy. Empyemas involve the subdural space diffusely and sometimes occur bilaterally.

A *subdural abscess* is a *localised* collection of pus; it may form during the treatment of subdural empyema, or the infection may be localised from the beginning. Subdural abscesses tend to form along the falx or between the occipital lobe and the tentorium. A subdural abscess produces a limited neurological deficit. Thus an abscess on the medial aspect of the hemisphere involving the motor cortex causes paralysis of the contralateral leg with sparing of the arm and face. Lumbar puncture in subdural infection shows a raised pressure, and the cerebrospinal fluid contains an increase in the number of white cells (100 to 300 per cu. mm.) but no organisms. Although a variety of organisms may be grown from the primary focus, the commonest one found in the subdural pus is the non-hæmolytic streptococcus.

**Treatment.** Burr-holes, planned according to the neurological signs, are made to confirm the diagnosis and to enable drainage to be carried out. In subdural *empyema* small rubber catheters are insinuated between the *dura* and *arachnoid* for the instillation of *penicillin*, to which the organisms of subdural infection are usually sensitive. The strength of penicillin employed is 500 units per 1 ml. of isotonic saline. Although systemic penicillin passes into the *subdural* space, the greater concentration of the drug obtained by the catheter method is desirable. Subdural abscesses are located with the aid of a brain cannula which is passed unhesitatingly through relatively silent parts of the brain, but it is particularly important to avoid the motor cortex and the speech zones. Diodone (e.g. Pyelosil) is injected into the subdural empyema or abscess cavity to demonstrate its extent radiologically. A subdural abscess, unlike a brain abscess, is shown to be superficial to the brain. The help of

ventriculography and angiography may be required to localise a subdural abscess. From the beginning antibiotics are given. When penicillin is indicated 20,000 units in 5 ml. of isotonic saline are given by lumbar puncture to prevent meningitis arising after needling the brain (penicillin given by intramuscular injection does not reach the cerebrospinal fluid). Subdural abscess is treated by intermittent aspiration and the instillation of the appropriate antibiotic (usually penicillin).

**Lateral Sinus Thrombosis.** Septic thrombosis may complicate either acute or chronic middle ear infection, for the sigmoid sinus is intimately related to the mastoid process. The manifestations are headache, *rigors*, swinging temperature and a moderate degree of papilloedema. Blood culture may be positive (septicæmia), and metastatic abscesses may form, especially in the lungs (pyæmia). Lumbar puncture may show a raised cerebrospinal fluid pressure if the thrombosis extends beyond the transverse sinus, and compression of the internal jugular vein *on the side* of the thrombosis fails to cause a rise in cerebrospinal fluid pressure (positive *Ayer's test*). The number of white cells in the fluid is moderately increased. Appropriate antibiotics are administered. Mastoidectomy is performed, and the sigmoid sinus exposed and incised. The clot is extracted until both ends bleed freely. The hæmorrhage is then controlled by gauze packing. If no bleeding occurs from the lower end of the sinus, the common facial and internal jugular veins are ligated to stop the spread of infection through the blood stream.

**Cavernous Sinus Thrombosis.** Septic thrombosis of the cavernous sinus may arise from : (a) Boils or carbuncles of the nose or upper lip, especially after surgical interference ; (b) erysipelas ; (c) dental sepsis in the upper jaw, especially after extractions ; or (d) middle-ear infection. In (a), (b) and (c) infection spreads to the sinus through the angular and ophthalmic veins ; in (d) it spreads through the superior petrosal sinus. The onset occurs with headache, *rigors* and a remittent temperature. The eyelids become swollen, the conjunctivæ œdematous (*chemosis*), the eyes fixed and proptosed, and the pupils dilated and inactive. If the cornea is clear, retinal hæmorrhages and papilloedema may be observed.

Septic sinus thrombosis is rare in countries where antibiotics are freely available for the treatment of the causal lesions. Formerly, the condition was fatal ; patients died from septicæmia with metastatic infection of the lungs, or from meningitis ; nowadays the disease is usually cured with antibiotics. Nevertheless, septic lesions of the nose or upper lip—the "danger area"—should not be squeezed or incised. There is no indication for ligation of the angular veins in cavernous sinus infection, as formerly recommended, when the primary lesion is in the danger area.

**Meningitis.** Unless otherwise stated, the term meningitis refers to leptomeningitis (i.e. inflammation of the arachnoid and pia mater—leptos Greek for fine, small or *thin*). Infection may reach the meninges directly from the exterior : (a) By erosion of the skin over a cranial or spinal

congenital defect (e.g. meningocele) ; (b) as the result of open injuries of the head or spine ; (c) following cranial or spinal operations or lumbar puncture when aseptic technique has been defective. Infection may reach the meninges from the following adjacent inflammatory diseases : (a) Otitis media ; (b) osteomyelitis of the skull ; (c) an infected dermoid sinus of the occiput or spine ; (d) a cerebral abscess which ruptures into the ventricles or subarachnoid space. Lastly, infection may occur through the blood stream ; tuberculous and meningococcal meningitis arise in this way whereas pneumococcal meningitis can occur either as a blood-borne infection from pneumonia, or directly from otitis media and mastoiditis (by far the commonest cause) or nasal sinusitis. In many cases of pneumococcal meningitis the source of infection cannot be traced.

*Diagnosis of Meningitis.* The symptoms are frontal headache and sometimes occipital headache, vomiting and in some cases photophobia. Retention of urine may occur. The physical signs are neck-stiffness, positive Kernig's sign and sometimes a positive Brudzinski's sign.\* These signs of *meningeal irritation* are accompanied by pyrexia and toxæmia. With the onset of coma the signs of meningeal irritation may disappear, and in infants and young children they may be inconspicuous or absent from the beginning. In *meningococcal meningitis*, which may occur sporadically or in epidemics, a petechial rash may be present on the trunk ("spotted fever"). In tuberculous meningitis, the syndrome of meningeal irritation may be absent, and thus the "diagnosis" of "P.U.O." (pyrexia of unknown origin) may be made.

In all forms of *leptomeningitis*, the cerebrospinal fluid pressure is raised. In *pyogenic meningitis* the fluid is opalescent because of the presence of a large number of polymorphs (usual range 1,000 to 10,000 per cu. mm.). The protein is increased (usual range 100 to 500 mg. per 100 ml.) while the sugar is reduced below 40 mg. per 100 ml. The chlorides are moderately reduced.†

In *tuberculous meningitis*, the cerebrospinal fluid appears clear or slightly hazy, and after standing a few hours a fine web-like fibrin clot forms. The cell count is increased. In the majority of cases there are between 50 and 500 cells per cu. mm. Lymphocytes and mononuclears predominate. The protein level is raised with an average of 200 mg. per 100 ml. If in tuberculous meningitis the protein exceeds 500 mg. per 100 ml. there is almost certain to be an arachnoid block. The sugar content is lowered but not to the same extent as in pyogenic meningitis. The chloride is usually reduced below 650 mg. per 100 ml. Although formerly thought to be a reliable test for the presence or absence of tuberculous meningitis, the chloride test is now regarded as significant

\* Flexion of the hips and knees occurs when the patient's head is flexed.

† Normal cerebrospinal fluid : Pressure up to 170 mm. of cerebrospinal fluid, cells not more than 3 per cu. mm., protein 15 to 45 mg. per 100 ml., sugar 50 to 80 mg. per 100 ml., and chloride (as NaCl) 700 to 750 mg. per 100 ml.

only when positive. Under the microscope, tubercle bacilli can often be seen in the fluid or can be cultured from it.

*Treatment of Meningitis.* In pyogenic meningitis, the organism should be identified as soon as possible and its antibiotic sensitivity determined. In the meantime, wide spectrum antibiotics should be given. If the organism is sensitive to penicillin, the drug should be given intrathecally as well as intramuscularly, for an adequate concentration of penicillin does not pass into the cerebrospinal fluid from the blood stream. The dose injected into the cerebrospinal fluid must *not* exceed 20,000 units daily and the dose should be dissolved in not less than 5 ml. of isotonic saline. Cases are on record in which 500,000 units were given in error and the patients developed generalised convulsions and died within twenty-four hours. Pneumococcal meningitis is usually treated with penicillin and a sulphonamide, whereas meningococcal meningitis requires only a sulphonamide in the majority of cases. *Hæmophilus influenzae* meningitis usually responds to chloramphenicol alone although there is a tendency to give it with sulphadiazine. In pyogenic meningitis, the focus of origin of the infection may be known and can be treated surgically, e.g. mastoiditis and osteomyelitis of the skull. In tuberculous meningitis, a course of *streptomycin* is given lasting several months. The drug is given intramuscularly and intrathecally. *Neurosurgeons* are often called upon to make burr-holes for injection of the drug into the lateral ventricles when adhesions have produced a subarachnoid block and thus interfere with the circulation of the drug (the intrathecal or intraventricular dose of streptomycin is 50 to 100 mg. in 5 ml. normal saline). Burr-holes may also be required in pneumococcal meningitis when a block occurs. When *satisfactory progress is not being made by a patient suffering from pyogenic meningitis*, the possibility of a coexistent brain abscess, subdural abscess or subdural hygroma should be considered.

### BRAIN ABSCESS

The causes of brain abscess are as follows : *Otitis media and mastoiditis* may give rise to an abscess in either the temporal lobe or the cerebellum. Abscess of the brain is much more likely to be caused by *chronic infection* of the middle ear, especially in the presence of cholesteatoma, than at the time of the original acute ear infection. *Inflammatory diseases of the lungs*, especially following their surgical treatment, are liable to cause a metastatic brain abscess ; *single abscesses occur much more frequently than multiple ones*. Degeneration of a secondary carcinoma, when there is no evident primary growth, may cause confusion in diagnosis when the lesion in the brain is aspirated. *Nasal sinusitis*, with or without osteomyelitis, may cause a frontal lobe abscess. *Open fractures of the skull*, when the dura mater has been opened and the brain penetrated, have a strong tendency to cause intracranial suppuration. A brain abscess is almost certain to form if fragments of bone, hair or hat-material have been



left in the brain. Fractures into the nasal air sinuses, with or without cerebrospinal fluid rhinorrhœa, frequently cause meningitis and sometimes a cerebral abscess. The presence of a missile is much less likely to cause an abscess. Therefore only the most easily accessible missiles should be removed. There is no justification for inflicting further damage on the brain for the purpose of removing a missile which may never cause suppuration. *Osteomyelitis of the skull*, caused either by infection of the nasal air sinuses or by blood-borne organisms, is liable to cause a brain abscess. Occasionally an inflammatory lesion of the scalp produces a cerebral abscess without evidence of osteomyelitis. Rarely, infection of an operation wound causes an abscess of the brain. *Peripheral sepsis* is responsible for an occasional brain abscess. Examples of such causal lesions are carbuncle, whitlow, tonsillitis, infected dental root (especially after extraction), septic wounds and osteomyelitis. Sometimes an abscess occurs without an evident focus of infection and may be encountered unexpectedly. Cryptogenic brain abscess is particularly liable to occur in the presence of congenital heart disease, especially when there is cyanosis. More than 50 per cent. of such abscesses have been found in association with *Fallot's tetralogy* and some have followed operations for cardiac lesions. They are not necessarily caused by paradoxical emboli, for the shunt is not always from right to left.

**Bacteriology.** Organisms isolated from abscesses of the brain include *Staphylococcus aureus* or *albus*; *Streptococcus pyogenes*, *viridans* or *pneumoniæ*; *Pseudomonas pyocyanea*; *Proteus vulgaris*, *actinomyces*, *diphtheroids*; *Hæmophilus influenzae*; and *Entamoeba histolytica* (rarely). Not infrequently a mixed growth of organisms is obtained. The nature of the responsible organisms and their sensitivity to the various antibiotics are ascertained. Often, however, the pus is sterile although the infection may still be active in the abscess capsule.

**Diagnosis.** An abscess of the brain behaves like a tumour, giving rise to increased intracranial pressure and localising signs. Thus there are headache, vomiting, and frequently, but *not always*, a mild degree of *papillædema*. A metastatic abscess may begin with a "shower" of epileptic attacks, which, if of the Jacksonian type, are of vital localising value. When the infection arises in the skull or its air-containing cavities, the cerebral abscess is usually immediately subjacent to the primary lesion, but occasionally it may be far removed from the area of osteomyelitis. A careful neurological examination is therefore necessary. The commonest site for osteomyelitis is in the frontal region, although no part of the skull is exempt. Of particular importance in temporal lobe abscesses is the examination of the visual fields which commonly show an upper quadrant homonymous defect, or, if the abscess is extensive, a complete hemianopia. Cerebellar abscesses produce minimal or moderate cerebellar signs (unilateral hypotonia; inco-ordination of the upper extremity of the same side; *nystagmus*, which is more marked and of

greater excursion when the patient fixes his gaze towards the side of the abscess ; and a tendency for the patient to deviate towards the side of the lesion when walking). A metastatic abscess may occur anywhere in the brain. As much information as possible about its location is obtained from the history and neurological examination, as in the diagnosis of intracranial tumours (*vide* Chap. 53), and help obtained from the investigations mentioned below. The temperature is most likely to be *slightly* raised (99° to 100° F.) or subnormal. A high temperature is not found with an uncomplicated brain abscess. The pulse is often bounding and may show an increased rate or the classical slowing of increased intracranial pressure.

**Investigations.** *Lumbar puncture* is avoided when possible for the reasons discussed in Chap. 53. The cerebrospinal fluid is clear in uncomplicated cases, although there is usually a moderate increase in white cells. There should be no organisms, either on direct examination of the fluid or after culture. When there is doubt about the presence or location of an abscess, *ventriculography* or *arteriography* should be carried out. The latter investigation is particularly useful in demonstrating an abscess in the temporal lobe, because it may be difficult or impossible to fill the temporal horn with air in such cases. *Electroencephalography* is likely to show slow waves of marked amplitude, and phase-reversal in the region of a supratentorial abscess. When a brain abscess has been localised, its extent can be demonstrated by positive contrast radiography. Diodone (e.g. "Pyclosil"), 2 to 3 ml. is injected into the abscess cavity after *some* of the pus has been aspirated. The skull is then X-rayed. Many neurosurgeons recommend Thorotrast (thorium dioxide), for it has the advantage of remaining *in situ* and thus the progress of the abscess can be followed radiologically. But there is experimental evidence that Thorotrast, acting as a foreign body, stimulates capsule formation. Therefore if resolution of the abscess is desired, the rapidly absorbable contrast medium, diodone, should be employed. Furthermore, it is a violation of surgical principles to leave an unabsorbable foreign body in a septic place.

#### TREATMENT OF BRAIN ABSCESS

**A. Aspiration.** A burr-hole is made over the site of the abscess, unless it is secondary to osteomyelitis of the skull when the dura mater is exposed by removal of infected bone after a suitable scalp flap has been reflected. A small incision, a few millimetres long, is made in the dura mater with the aid of a sharp hook and tenotome. An avascular point on the surface of the brain is punctured with a pointed tenotome, and a blunt-ended brain needle is passed into the abscess cavity. The pus is aspirated and replaced by 2 to 3 ml. penicillin solution (100,000 units per ml.). A small volume of fluid is injected to avoid rupture of the capsule which may be extremely thin. Sometimes an abscess heals after one penicillin replacement, but the procedure may have to be repeated several times. The

cannula (brain needle) is then passed through the original burr-hole between the sutures. Considerable judgement is needed in spacing aspirations. Needling should be repeated if the patient's general and neurological state do not improve; it should not be delayed until deterioration occurs. At first, aspiration and penicillin replacement may be needed at intervals of twenty-four to forty-eight hours, but when progress is satisfactory, the intervals become longer and longer until no more pus can be aspirated. Many abscesses can be completely cured by this technique, but if a patient's condition does not improve or deteriorates

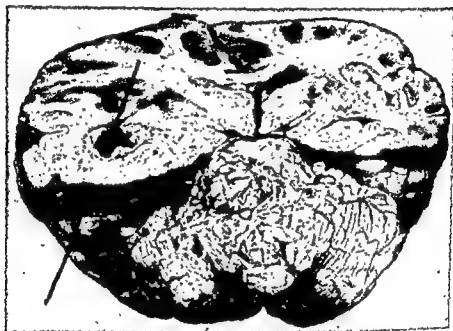


FIG. 344. This temporal lobe abscess communicated directly with the mastoid antrum through a large opening. The abscess thus remained active and failed to respond to repeated aspiration.

the possible causes are: (1) *Persistent infection in the capsule associated with brain swelling.* The abscess should then be excised (*vide infra*, method B) and decompression performed. The oedema may also be reduced by the intravenous injection of 100 ml. 50 per cent. sucrose solution which may be repeated once after six hours. (2) *Loculation*, so that aspiration only drains part of the abscess. A *loculus* may be detected and drained by needling in several new directions, or ventriculography may be needed. (3) The presence of *multiple abscesses* which may occur when the primary infection is in the lungs. Neurological examination alone may not reveal the location of additional abscesses, but they may be demonstrated by ventriculography. (4) A *sinus* which may become established with the tympanic antrum in otogenic brain abscesses (Fig. 344). Such abscesses will not heal unless excised. (5) The presence of a foreign body

in the abscess, e.g. bone fragments in post-traumatic cases. The aspiration technique is particularly applicable to abscesses which involve the motor cortex, the speech zones or the optic radiations when other methods would cause severe neurological deficits. Furthermore, aspiration may be life-saving for patients who are too ill to undergo a major operation.

**B. Secondary Excision.** Excision of the abscess capsule is carried out when aspiration alone fails. Some neurosurgeons, however, excise the abscess capsule as a routine as soon as aspiration is unproductive. Before enucleation, penicillin (20,000 units in 5 ml. of normal saline) is injected into the ventricles, and it is also given systemically, to "cover" the operation and the post-operative period. After excision of the abscess capsule, the wound is closed without drainage.

**C. Primary Excision.** This is the most recent method of treatment, and is made possible by the discovery of antibiotics. The advantage is that the anxious period of observation required by the aspiration technique is eliminated. Primary excision of the abscess is particularly indicated when it is situated in a relatively silent part of the brain, e.g. the frontal or cerebellar lobes where a neurological deficit is unlikely to be caused by excision. It is also the best method of treating an abscess following an open head injury, for any indriven fragments of bone or foreign bodies are automatically removed and the infection is thus brought to an end. A brain abscess occasionally follows a very slow course, and the onset of increased intracranial pressure and localising signs are long delayed. Such an abscess develops into a solid mass of fibrous tissue closely resembling a tumour, although there may be a small quantity of pus in the centre. Primary excision is the only possible treatment in such a case.

The above methods of treatment apply to both *cerebral* and *cerebellar* abscess, but the management of the latter requires further discussion because of its close relationship to the medulla oblongata. Compression of this structure by the increasing volume of the abscess and the surrounding oedema of the cerebellum (Fig. 345) demands immediate decompression by all means at our disposal. Therefore the patient is first given 100 ml. of 50 per cent. sucrose intravenously, and then a full exposure of the posterior fossa is carried out through the time-saving longitudinal midline incision (Fig. 384). The abscess is aspirated with a brain cannula through a small puncture made in the dura mater on the side of the abscess, and a small quantity of penicillin (100,000 units dissolved in 1 ml. of isotonic saline) is instilled into the cavity. The dura mater is opened over both cerebellar lobes, and then the abscess is removed by gentle dissection, when there is a well-defined capsule, or by low-pressure suction when there is little or no visible capsule. The wound is closed without drainage, and 20,000 units of penicillin in 5 ml. of normal saline are given by lumbar puncture. If the patient is very ill, the abscess is aspirated and its removal carried out a few days later. Alternatively, repeated

aspiration and penicillin replacement may be employed; a small additional incision is made for this purpose through the skin and muscles on the appropriate side, but a cerebellar abscess is not easy to reach with a cannula through the depth of the occipital muscles.

### THE FOCAL LESIONS

**Osteomyelitis of the Skull (Fig. 343).** This is caused by direct spread of infection from nasal sinusitis, or by blood-borne organisms (usually *Staphylococcus aureus*) from a distant focus such as a boil, carbuncle or septic dental root. In *pansinusitis*, the skull, sometimes including the

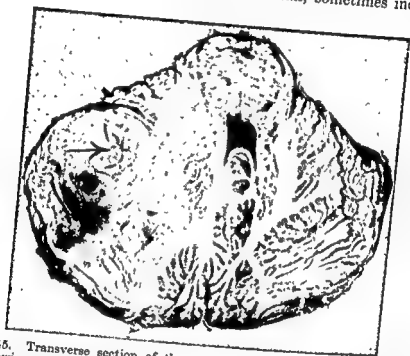


FIG. 345. Transverse section of the pons and cerebellum, seen from above, showing a small abscess (arrow) which caused fatal swelling of the left half of the cerebellum.

facial bones, is most likely to become infected during an acute exacerbation of chronic inflammation, or after operations for sinusitis. Osteomyelitis of the skull occasionally follows *typhoid fever*. *Pott's puffy tumour* is a classical term applied to marked localised oedema overlying an area of osteomyelitis and an extradural abscess. However, in osteomyelitis the scalp is oedematous, but localised oedema sufficient to produce a swelling worthy of the name of tumour is uncommon. Radiological evidence of osteomyelitis tends to lag behind clinical evidence of the disease. Sometimes bone containing pus has a normal radiological appearance. Antibiotics are administered, but craniectomy should be carried out if the response to conservative treatment is not immediately satisfactory. Patients suffering from osteomyelitis of the skull are very toxic and usually have a marked anaemia.

**Otitis Media and Mastoiditis.** *Acute otitis media* is usually preceded by tonsillitis, rhinitis, influenza or measles. The causal organisms are *Streptococcus pyogenes*, *Pneumococcus* or *Staphylococcus aureus*. The symptoms are severe pain, deafness and tinnitus. In the fully developed attack, the drum is red and bulging, and the handle of the malleus is obscured. Hæmorrhagic blebs may be seen on the surface of the drum in influenzal cases. In infants, the tympanic membrane is normally thick, and although it bulges in acute otitis media it may not appear inflamed. Sulphonamides are often sufficient to cure acute otitis media. The organisms are usually sensitive to penicillin which is therefore given if the response to sulphonamides is not satisfactory. Most cases respond to conservative treatment, but if the drum continues to bulge and pain persists, the drum is incised vertically in its posterior half down to its inferior margin (*myringotomy*). The perforation usually heals and hearing is restored. Mastoiditis is nowadays a rare complication of acute otitis media.

*Chronic otitis media* is usually the result of failure of resolution of acute infection. The symptoms are *deafness* and *chronic otorrhœa*. A central or marginal perforation is present. The latter usually indicates adjacent caries of bone. Some cases of chronic otitis media arise insidiously and show a perforation of Shrapnell's membrane (attic perforation). Chronic aural discharges should be examined for the tubercle bacillus which is sometimes the causal organism, although a mixed infection commonly exists. Continuous exfoliation of the epithelium lining the attic may cause a mass known as a *cholesteatoma* (epithelial debris, pus and cholesterol crystals). It is the lesion which is most often the cause of intracranial complications in otitis media. Radical mastoidectomy is the treatment for chronic otitis media except when the infection is tuberculous.

**Inflammatory Disease of the Lungs.** When the inflammatory focus is in the lungs, it should be treated surgically, when possible, as soon as the patient's condition permits, for there is a tendency for further metastatic lesions to occur in the brain.

## CHAPTER 52

### THE CRANIAL NERVES

#### THE OLFACTORY NERVES

THE causes of anosmia (absence of smell) are : (a) *Congenital*.—This is rare and the mechanism is unknown. (b) *Traumatic*.—In head injuries, with or without fractures of the skull, anosmia may be produced by avulsion of the olfactory filaments from the cribriform plate. Unilateral anosmia follows upward retraction of the frontal lobe during intracranial operations, e.g. during removal of a pituitary tumour. (c) *Inflammatory*.—Infection of the nose and paranasal sinuses frequently causes anosmia. Loss of sense of smell occasionally occurs in *meningitis* and *tabes dorsalis*. (d) *Neoplastic*.—Anosmia, unilateral or bilateral, is a constant feature of *olfactory groove meningiomas*, and it is very occasionally caused by the pressure of frontal gliomas.

#### THE OPTIC NERVES

Blindness in one eye of gradual onset is sometimes not noticed by patients until something happens to the sound eye (e.g. the entry of a foreign body into the conjunctival sac) ; the history of sudden blindness then obtained is misleading. Patients with bitemporal hemianopia may have no symptoms until the visual acuity declines, for each eye partially compensates for the field defect of the other (Fig. 346). On the other hand, patients with homonymous hemianopia are often well aware of the defect, especially when the complete half-fields are affected.

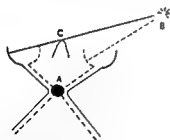


FIG. 346. The lesion A (e.g. pituitary tumour) causes blindness of the nasal half of each retina, but the object B may still be picked up on the normal temporal half of the left retina provided the light rays are not obstructed by a prominent nose C.

The importance of ophthalmoscopy cannot be overstressed. Patients should be examined in the dark after the pupils have been dilated with a mydriatic (*homatropine* 2 per cent. and *cocaine* 2 per cent. in aqueous solution, instilled into the conjunctival sac, and followed after the examination by  $\frac{1}{4}$  per cent. *eserine* to guard against glaucoma). A normal fundus is shown in Fig. 347.

**Primary Optic Atrophy.** Optic atrophy not caused by papilloedema or lesions of the retina is termed *primary optic atrophy* (Fig. 348). After an acute lesion of the optic nerve, atrophy begins in two weeks



FIG. 347. Painting of a normal right fundus

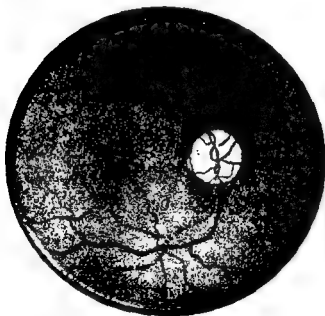


FIG. 348. Primary optic atrophy. In this case caused by a glioma of the optic nerves and chiasm. Note the pallor of the disc and its sharp outline.





and is complete in six weeks. When optic atrophy is well advanced, the pupil is dilated and does not react to direct light, although there is a consensual reflex and a reaction to accommodation. In incomplete atrophy, as when the causal disease is slowly progressive, the only pupillary abnormality may be failure of the pupil to sustain contraction to direct light.

**Intrinsic Causes of Optic Atrophy.** *Disseminated Sclerosis.*—The diagnosis depends on evidence of multiple lesions, careful investigations to exclude other diseases, and a fluctuating clinical course. The visual fields may show transient or permanent central or paracentral scotomas. (N.B.—Contrary to what might be expected compression of the optic nerves also produces central or paracentral scotomas.) *Tabes Dorsalis.*—Other evidence of the disease is present, including Argyll-Robertson pupils and a positive Wassermann reaction. The visual fields tend to be constricted in this disease. (Note: Optic atrophy is rare in acquired G.P.I.) *Occlusion of the Central Artery of the Retina.*—There is a sudden onset of unilateral blindness. A zone of pallor is observed around the fovea which is prominent as a "cherry-red" spot. *Amaurotic Family Idiocy (Tay Sachs' Disease).*—This is a very rare disease characterised by widespread lipoid degeneration of ganglion cells, including those of the retina. The disease usually begins in the first year of life, and is a familial disease almost entirely confined to Jews. Blindness is accompanied by dementia. Examination of the fundi shows optic atrophy and pallor around a "cherry-red" fovea. *Deric's Disease (Syn. Neuro-myelitis Optica).*—In this rare disease there is rapid impairment of vision followed by optic atrophy, which may be primary or sometimes secondary to papilloedema. There are bilateral central scotomas. A coincident or consecutive transverse lesion of the spinal cord occurs. About half the patients succumb to the disease. *Chronic Glaucoma.*—Optic atrophy is associated with cupping of the whole disc. The tension of the globe is raised, and the fields are constricted, especially on the nasal side. An arcuate paracentral scotoma is typical of the early stages of the disease. *Poisons.*—Optic atrophy may be caused by methyl alcohol, lead or quinine. *Temporal Arteritis.*—The superficial temporal arteries and their branches are very swollen and tender. In some cases, loss of vision occurs as the result of involvement of the nutrient vessels supplying the optic nerves (occlusion of the central artery of the retina is rare). The optic discs are pale; sometimes there is slight papilloedema. *Glioma of the Optic Nerve and Chiasm.*—There is slowly progressive blindness in both eyes, but one side is usually affected more than the other. Some cases are associated with von Recklinghausen's neurofibromatosis. Plain radiography shows enlargement of one or both optic foramina (Fig. 349). The chiasmal part of the tumour tends to grow upwards and backwards indenting the floor of the third ventricle (i.e. the hypothalamus).

**Extrinsic Causes of Optic Atrophy.** *Head Injuries.*—The injury is

usually severe, and a fracture involves the walls of the optic canal. Injury of the chiasm may cause bitemporal hemianopia or total blindness. In warfare, the optic nerves or chiasm may be injured by missiles. Injuries of the optic nerves produce permanent blindness on the affected



FIG. 349. Radiographs taken to show the optic foramina. In the upper view the enlarged right optic foramen is shown at "9 o'clock," and in the lower view the normal left optic foramen is shown at "3 o'clock." The patient had a glioma of the chiasm and right optic nerve proven by transfrontal exploration and biopsy. Removal of the tumour was not done, for total blindness would have been produced, and, furthermore, the tumour, being benign, was not an immediate threat to life.

side. *Diseases of Bone.*—Paget's disease, leontiasis ossea and metastatic tumours sometimes compress the optic nerves. De-compression of the optic nerve should be carried out to save remaining vision. *Intracranial Tumours.*—Meningiomas of the medial end of the lesser wing of the sphenoid bone compress the optic nerve causing optic atrophy. Sometimes there is optic atrophy on the side of the tumour with papillœdema on the opposite side (Foster - Kennedy syndrome). Meningiomas arising from the tuberculum sellæ (syn. suprasellar meningiomas) displace the optic nerves and chiasm upwards, and thus cause optic atrophy and bitemporal hemianopia. The pituitary fossa may be normal or partially destroyed; it is not expanded as with pituitary tumours. *Pituitary Tumours* (Chap. 53). *Intracranial Aneurysms* (Chap. 56). *Tumours of the Nasopharynx.*—Carcinomas, reticulosarcomas and lympho-epitheliomas of the nasopharynx tend to invade the base of the skull and involve the optic and other cranial nerves. The tumour may be very small and escape detection in the pharynx.

Examination includes palpation of the nasopharynx. Sometimes it is necessary to split the palate along its median raphe before the tumour can be demonstrated. The two halves are, of course, sutured together after the examination. Radiotherapy is indicated for these tumours. *Mucocele and Empyema of the Ethmoid and Sphenoid Sinuses.*—Diverticula of the ethmoid or sphenoid sinuses may occur through erosions in the

wall of the optic canal and cause optic atrophy. During the stage of acute inflammation, headache or periorbital pain occurs, and is followed by diminishing vision in the affected eye. Sometimes radiography reveals enlargement of the optic canal. If discovered during intracranial exploration, the diverticulum should not be incised, for intracranial infection is likely to follow. The affected sinus should be drained extracranially.

*Non-specific Arachnoiditis.*—In some cases of optic atrophy, dense leptomeningeal adhesions are localised around the optic nerves and chiasm. Improved vision has been reported following removal of the adhesions in some cases.

*Tumours of the Orbit*—Tumours of many varieties, both primary and secondary, occur in the orbit. The nature of some of them is still undecided. The group of primary tumours includes : Glioma, neurofibroma and meningioma of the optic nerve ; pseudotumour (having a granulomatous appearance under the microscope) ; lipoma ; osteoma ; adenoma of the lachrymal gland ; meningocele ; angioma and lymphangioma. All of these cause unilateral proptosis (exophthalmos). Those arising from, or within, the optic nerve push the globe directly forward (Fig. 350). Tumours arising



FIG. 350. Protrusion of the right eye directly forward in a girl age twelve, with a meningioma arising from the optic nerve.



FIG. 351. Forward and downward protrusion of the left eye caused by a primary malignant tumour arising outside the muscle cone. The tumour was examined by several pathologists ; all agreed it was malignant, but its exact nature was not determined. (The histology of orbital tumours is notoriously difficult.)

outside the muscle cone tend to push the globe forwards and downwards (Fig. 351). Some of these tumours cause optic atrophy which may be

primary or associated with papilloedema. Orbital tumours are nowadays usually removed through the roof of the orbit approached by a frontal exposure. The orbital plate is exposed extradurally and removed with the aid of a sphenoid punch. The exophthalmos tends to be replaced by enophthalmos after operation.

**Secondary Optic Atrophy.** Optic atrophy caused by papilloedema is termed secondary optic atrophy. The disc is white, its edges are blurred and the physiological cup is filled in by proliferated glia. Near the disc, the vessels have white lines along them. The glial tissue tends to disappear in time so that the appearance becomes indistinguishable from primary optic atrophy. Nowadays papilloedema caused by increased intracranial pressure is often relieved before optic atrophy develops, and when the swelling of the discs subsides they appear completely normal.

**Consecutive Optic Atrophy.** This is the term applied to optic atrophy following extensive lesions of the retina, e.g. retinitis pigmentosa.\*

**Papilloedema.** At first there is blurring of the disc margins. At this stage it may be confused with the indistinct nasal margin not infrequently seen in normal adults. Later the physiological cup is filled in; the nerve head becomes swollen above the surface of the retina, and the identity of the disc margin is lost (Fig. 352). The retinal vessels lose their light reflex where they pass to and from the swollen disc, for they are then perpendicular to the retina and are thus unable to reflect the light coming from the ophthalmoscope. Flame-shaped hæmorrhages and/or white exudates may be seen on the disc and in its vicinity. The retinal veins are usually distended. The amount of disc swelling may be measured with the aid of an ophthalmoscope after the pupils have been dilated with a mydriatic (homatropine). Beginning with a +12 lens, the examiner gradually reduces the strength of the lenses until he can see a retinal vessel without blurring. The process is then repeated with the same vessel as it passes across the optic disc. The difference between the lenses required gives the amount of swelling in dioptres (3 dioptres are equivalent to 1 mm. of swelling). Thus, if a vessel is best seen on the retina with a +3 lens and on the disc with a +6 lens, there are 3 dioptres of papilloedema.

**Causes of Papilloedema. Intracranial Tumours.**—Papilloedema is one of the cardinal signs of an intracranial tumour although the optic discs are normal in a substantial proportion of cases. Neither its presence nor its degree are related to the size of the tumour. Some of the smallest tumours cause marked papilloedema whereas some of the largest cause

\* The terms primary, secondary and consecutive optic atrophy are used only for clinical convenience. The term secondary is not used in its usual sense as indicating an effect following any known cause, but one cause only, namely papilloedema. Strictly speaking, optic atrophy is always secondary; there is no such thing as idiopathic (primary) optic atrophy.

none. When papillœdema is unilateral the tumour may be within, or compressing the corresponding cerebral hemisphere. Papillœdema on one side associated with primary optic atrophy on the other (Foster-Kennedy syndrome) is found with meningiomas arising from the medial end of the lesser wing of the sphenoid bone or the olfactory groove on the side of the optic atrophy; the pressure of the tumour on the homolateral optic nerve prevents the development of œdema. *Brain Abscess*.—A moderate degree of papillœdema is frequently found with cerebral or cerebellar abscesses. *Intracranial Hæmatomas*.—The development of an extradural hæmatoma (syn. middle meningeal hæmorrhage) is usually too rapid for papillœdema to occur, although it is seen in some cases of slow evolution. Chronic subdural hæmatomas may produce papillœdema. Spontaneous or traumatic intracerebral hæmatomas also sometimes cause papillœdema. *Hydrocephalus*.—In infantile hydrocephalus, papillœdema is rare, for, as the bones of the calvarium have not united, spontaneous decompression is afforded by enlargement of the head. *Meningitis*.—Any form of meningitis may cause mild papillœdema by raising the cerebrospinal fluid pressure. *Marked swelling of the discs*, however, should lead to the suspicion of an associated abscess, or, in the case of tuberculous meningitis, of a tuberculoma. *Subarachnoid Hæmorrhage* (Chap. 56).—Severe subarachnoid hæmorrhage, whatever its cause, raises intracranial pressure, and sometimes produces papillœdema. There may also be associated subhyaloid (pre-retinal) hæmorrhages. *Thrombosis of intracranial Venous Sinuses*.—Septic or aseptic thrombosis of the venous sinuses may follow middle-ear and mastoid infection, or inflammatory diseases of the nasal region. A moderate degree of papillœdema is commonly found. *Arteriovenous aneurysms* of the internal carotid artery and the cavernous sinus (Chap. 56). Bilateral proptosis and papillœdema result from the increased venous pressure. *Orbital Tumours*.—Papillœdema may occur whether the tumour is primarily orbital, or an extension of an intracranial tumour. *Thrombosis of Retinal Veins*.—Sudden loss of vision occurs in the affected eye. There is swelling of the optic disc with hæmorrhages and sometimes exudates on the disc and in the retina. The retinal veins are very engorged and tortuous. *Ethmoid and Sphenoid Sinusitis*.—Inflammation in the sinuses close to the optic nerve may cause optic atrophy with or without papillœdema. When papillœdema is present in sinusitis, the possibility of an intracranial abscess should be considered. *Malignant Arterial Hypertension*.—One of the cardinal signs of this disease is papillœdema, which is usually, but not necessarily, accompanied by nipping of the retinal veins by arteriosclerotic arteries. There are usually also exudates and a macular star. The blood pressure is raised and there is albuminuria. Papillœdema and retinopathy may also be found in toxæmia of pregnancy. *Chronic Nephritis*.—Examination of the fundi shows arteriosclerosis, hæmorrhages, large exudates (cotton-wool patches), and swollen discs and retinæ. *Polycythæmia Rubra*

*Vera.*—Papillœdema may be found in this disease. *Chronic Bronchiectasis and Emphysema.*—The papillœdema which is sometimes found in this disease is thought to arise from secondary polycythæmia. *Thoracic Venous Obstruction.*—This may be caused by any space-occupying lesion in the mediastinum, e.g. thyroid adenoma. *Disseminated Sclerosis.*—The optic "neuritis" of this disease may affect the nerve head and causes swelling. There is sudden loss of central vision with a corresponding scotoma (central field defect). Remissions are characteristic of this disease. *Poisons.*—Mild swelling and congestion of the discs may be found in methyl alcohol poisoning. *Fistulous wounds of the eye* or excessive drainage following trephine treatment of glaucoma can produce papillœdema as a result of ocular hypotension.

*Causes of Unilateral Papillœdema:* (a) Some intracranial tumours; (b) thrombosis of the central retinal vein; (c) infection of the ethmoid and sphenoid sinuses; (d) some orbital tumours.

**Pseudopapillœdema.** This is a congenital heaping-up of the optic nerve fibres. It occurs exclusively in hypermetropic (long-sighted) eyes. The nerve head is smooth and glistening, and the vessels are not concealed by œdema. Venous engorgement, hæmorrhages or exudates do not occur. The blind spot is not enlarged as it is in papillœdema. The appearance of the discs remains constant.

**Opaque Nerve Fibres.** They may be mistaken for papillœdema. Myelination of the optic nerve fibres usually ends behind the lamina cribrosa. In some cases, however, nerve fibres, while remaining non-myelinated in the region of the lamina cribrosa, are myelinated for a short distance beyond the edge of the disc (Figs. 353). The fine lines of the nerve fibres radiating from the disc are characteristic. The myelin disappears in the event of optic atrophy.

**Drusen.** This somewhat rare condition may simulate papillœdema. It is a developmental anomaly in which amorphous deposits of yellow or white waxy material are found in the optic discs. As in papillœdema the blind spot is enlarged. Drusen are likely to cause most difficulty when the hyaline deposits are buried below the surface of the optic disc. Although the blind spot is enlarged there is no venous congestion and there are no hæmorrhages. There is no evidence of the diseases which cause papillœdema.

**Visual Fields.** The visual pathways are long and devious, and, therefore, are frequently involved in injury and disease. The visual fields are first examined by *confrontation*. The patient covers one eye while looking directly forward. The examiner's moving index finger is brought inward from the periphery from each quadrant of the field in turn, and the patient, without deflecting his gaze, indicates when the moving finger is seen. The procedure is then carried out on the other side. If the moving index finger passes all the way across the visual field, it may be possible to demonstrate a central defect (scotoma). *Visual attention* is next tested.



FIG. 332. Typical papilloedema. The patient had an epidermoid cyst in the posterior fossa. Complete recovery followed operation and the optic disc returned to normal.

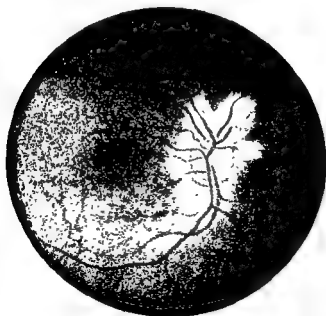


FIG. 333. Opaque nerve fibres. Confusion with papilloedema is most likely when opaque fibres completely encircle the disc.





The patient looks straight forward without deflecting his gaze ; the examiner's moving right index finger is brought inwards from the right until it is seen by the patient ; the moving left index finger is brought inwards until it is seen by the patient ; and then both index fingers are moved together. Normally the patient sees the movements of both fingers together, but sometimes with a lesion of one parietal lobe the movements of the finger on the opposite side may not be seen (i.e. there is *extinction* of the image). The *periphery* of the visual fields is plotted on charts with the aid of a *perimeter*. The size of the test object and the distance of the patient's eye from the fixation point are recorded in millimetres and expressed as a fraction. Thus  $\frac{3}{330}$  means that the test

object is 3 mm. across and the eye is 330 mm. from the fixation point—the distance for most perimeters. Central and paracentral field defects (scotomas) are more accurately investigated on a large flat black screen (Bjerrum screen). The patient usually sits 2 metres (2,000 mm.) away. If the test object measures 10 mm. in diameter, the conventional fraction

is  $\frac{10}{2,000}$ . Large test objects are required when visual acuity is impaired ;

small test objects or coloured ones are necessary to demonstrate early field defects. The visual acuity is tested after correction of any errors of refraction and recorded on the field chart. Opacities of the visual media may cause errors in the interpretation of field defects.

### THE THIRD, FOURTH AND SIXTH CRANIAL NERVES

The third nerve supplies the levator palpebræ superioris and all the extra-ocular muscles except the external rectus (supplied by the sixth) and the superior oblique (supplied by the fourth). The latter muscle causes the eye to look downwards and outwards. The third nerve also carries the parasympathetic fibres to the eye (subserving contraction of the pupillary and ciliary muscles). In complete paralysis of the third nerve there is ptosis ; the eye looks outwards and slightly downwards ; the pupil is dilated and does not react to light or accommodation. The effects of paralysis of the fourth or sixth nerves are easily deduced from the above information.

**Causes of Ophthalmoplegia.** *Subarachnoid carotid aneurysms* and *intracavernous aneurysms* commonly cause ophthalmoplegia (Chap. 56). *Head Injuries* (see Chap. 50). *Raised Intracranial Pressure.*—Unilateral, or sometimes bilateral sixth nerve palsy may occur as the result of kinking of the nerves at their dural foramina, caused by caudal displacement of the brain. A total third nerve palsy is rarely produced by raised intracranial pressure although a dilated pupil is frequently found on the side of a cerebral lesion (e.g. tumour ; extradural, subdural or intracerebral

hæmatomas ; cerebral infarct). Some authorities believe this phenomenon to be caused by herniation of the medial part of the homolateral temporal lobe over the edge of the tentorium on to the third nerve. However, if this is the explanation, it is surprising that a total third nerve palsy is not produced. *Orbital Tumours*.—Any space-occupying lesion inside the orbit may cause ophthalmoplegia. *Exophthalmic Ophthalmoplegia*.—Severe exophthalmos with paralysis of movements of the eyes sometimes follows thyroidectomy. The same syndrome also occurs without a history of thyroid disease. The cause of the condition is thought to be over-activity of the thyrotropic pituitary hormone. Naffziger's decompression of the superior and lateral walls of the orbits through a frontal extradural approach affords some measure of relief and should be carried out before corneal ulceration occurs. Operation is performed first on one side and then on the other with an interval of a fortnight between the two operations. If corneal ulceration has already occurred, the eyelids should be sewn together (tarsorrhaphy). *Meningitis*.—Any or all of the three nerves supplying the muscles of the eye may be paralysed by meningitis whatever the nature of the organism. The effects on the nerves are produced directly by the inflammatory process or by kinking due to raised intracranial pressure. The sixth cranial nerve (abducens) is sometimes involved at the tip of the petrous bone together with the fifth (trigeminal) nerve in mastoid infection. There is an internal squint, an absent corneal reflex, and impaired sensation on the face on the side of the disease (Gradenigo's syndrome). These signs usually disappear after successful treatment of the mastoid infection. *Malignant Tumours*.—Primary tumours of the nasopharynx (carcinoma, reticulosarcoma and lympho-epithelioma) invade the base of the skull and affect the cranial nerves, including those now being discussed. Pain may be produced by involvement of the trigeminal nerve. At first, tumours in the nasopharynx may not produce nasal symptoms, but reveal themselves by causing cranial nerve palsies.

Rare causes of ophthalmoplegia are : (a) Pituitary tumours (Chap. 53) ; (b) suprapituitary tumours (craniopharyngiomas) which occasionally become malignant and invasive ; (c) meningiomas of the anterior part of the middle cranial fossa (sphenoid wing meningiomas, Chap. 53) or of Meckel's cave ; (d) metastatic tumours in the bone in the region of the superior orbital fissure. Unilateral palsy of several cranial nerves is more often due to cranial and intracranial infiltration by a tumour of the nasopharynx than any other disease (ap. G. Jefferson).

*Congenital Ophthalmoplegia*. The mechanism is unknown. There is suppression of vision in the affected eye. The deformity may be corrected by operations on the external ocular muscles. *Encephalitis Lethargica*.—Brain stem signs such as loss of conjugate upward movement of the eyes, or loss of convergence are common manifestations of this disease. *Disseminated Sclerosis*.—Diplopia (double vision) is one of the classical

symptoms of this disease. One of the ocular nerves may be involved in a degenerated part of the brain stem. *Poliomyelitis*.—Sometimes the sixth cranial nerve is involved in this disease. *Polyneuritis*.—Paralysis of one or several of the cranial nerves, with or without neuritis of the limbs, may occur. Recovery can be expected in most cases. *Lumbar puncture*, with or without injection of a spinal anæsthetic, may cause a sixth cranial nerve palsy.

## THE FIFTH CRANIAL NERVE

### (TRIGEMINAL NERVE)

**Tic Douloureux** (Syn. Trigeminal Neuralgia). This syndrome is characterised by *attacks* of severe pain in the distribution of one or more divisions of the trigeminal nerve; the first division is often spared and is rarely affected alone. The essential features are the spasmodic nature of the pain and its sudden onset. Attacks are frequently fired off by stimulation of a "trigger" area, which is commonly situated inside the mouth, and therefore mastication is made difficult or impossible. The spasms of pain may occur every few minutes. In some cases there are intervals of freedom lasting months, whereas in others attacks continue without remission. Elderly people are most often affected, but occasionally trigeminal neuralgia occurs in young adults. It is occasionally caused by disseminated sclerosis. The important points in the diagnosis are the spasmodic character of the pain and the absence of abnormal neurological signs. It is of paramount importance to distinguish the syndrome from other causes of trigeminal pain, for, whereas injection of the Gasserian ganglion with alcohol or sensory root section are successful in the treatment of true trigeminal neuralgia, these procedures are usually ineffective in the treatment of all other types of facial pain. The cause of trigeminal neuralgia is unknown except in those cases occurring with disseminated sclerosis.

Other causes of trigeminal pain are: Dental caries and dental root infection, frontal sinus and antrum infection, post-herpetic "neuralgia," carotid aneurysm in the cavernous sinus, injuries of the facial bones, and, rarely, glaucoma, carcinoma of the antrum, meningioma arising in Meckel's cave, acoustic nerve tumour, thrombosis of the posterior inferior cerebellar artery, tabes dorsalis and leontiasis ossea.

**Treatment of Tic Douloureux.** *Alcoholic injections* through the foramen ovale should be tried before operation is considered. The technique is not easy and unpleasant complications may occur; they include ophthalmoplegia and facial paralysis. This is not surprising, for the method is a blind one, and the foramen ovale is close to that anatomical junction, the cavernous sinus. Alcohol spreads quite slowly after injection; therefore there is a short interval before complications appear. The

method to be described is designed to reduce risks to a minimum. The patient's full co-operation is needed when the injection is being carried out. Therefore premedication is omitted. The patient's face is cleaned thoroughly, and a vertical line marked on it with triple dye  $1\frac{1}{2}$  inches in

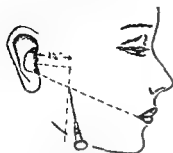


FIG. 354. The surface markings for trigeminal injection by the lateral route.

front of the centre of the external auditory meatus (Fig. 354). This line is a little in front of the coronal plane passing through the foramen ovale. The site of entry of the needle is on this line at its point of cross-section with another line drawn between the incisura intertragica and the angle of the mouth (Fig. 354). Edentulous patients are made to bite on a cork to lower the sigmoid notch. An intracutaneous weal is made at the above point with 1 per cent. procaine. A Harris's needle (Fig. 355) is passed through the anæsthetic area slightly backwards and

upwards. The depth of the foramen ovale varies from approximately  $\frac{1}{2}$  to 6 cm., and when the needle slips through it the patient experiences pain in the lower jaw. Sufficient procaine is injected to anæsthetise the mandibular area and thus prove the needle to be in the foramen ovale. If too much procaine is used, the whole of the side of the face is anæsthetised, and the effect of the subsequent alcohol injection cannot be observed.

Aspiration always precedes injection of procaine or alcohol, and if blood or cerebrospinal fluid is withdrawn, the position of the needle is changed. Absolute alcohol is drawn up into the

syringe and several injections of 0.1 ml. are given. Between each injection an interval of not less than one minute is allowed for observation of the pupils, external ocular movements and facial symmetry. Further injections are postponed until another occasion if any abnormal signs develop, e.g. dilatation of the pupil, external rectus palsy or facial palsy. These small injections are repeated until the desired area of analgesia is obtained, depending on the divisions involved. When it is desired to block the upper divisions of the nerve the needle is advanced slightly before injection. If the corneal reflex is abolished, the patient should wear an eye-shield, and should be issued with written instructions regarding the care of the eye. On the rare occasions when injection fails, operation is necessary, for drugs do not control the pain.

*Division of the Sensory Root of the Trigeminal Nerve (Spiller-Frazier Operation).* Most neurosurgeons perform this operation with the patient in the sitting position to minimise venous bleeding. A vertical incision

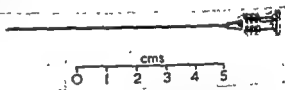


FIG. 355. A Harris's needle with its stilet in situ.

is made in the temporal region *immediately in front of the external auditory meatus*. The temporal muscle is divided in the same line as the skin incision, and an opening is made in the bone about the size of a five-shilling piece. The dura mater is retracted upward and the middle meningeal artery is followed to the foramen spinosum where it is coagulated and divided. Occasionally it is necessary to plug the foramen with the point cut from the end of a sharpened match-stick. The mandibular division of the nerve is found anterior and medial to the foramen

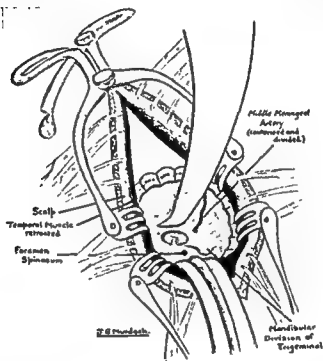


FIG. 356. Exposure and division of the sensory rootlets of the trigeminal nerve.

spinosum. The most important part of the operation is the finding of the plane of cleavage between the dural sheath of this nerve and the dura mater of the overlying brain. Dissection is carried out in this plane to obtain a good exposure of the separate dural coverings of: (a) The mandibular nerve; (b) the maxillary nerve; (c) the Gasserian ganglion with the roots of the trigeminal nerve (Fig. 356). The sheath of the ophthalmic division is not exposed. The dura enclosing the nerve roots (i.e. Meckel's cave) is incised and the glistening arachnoid is torn with a sharp dural hook. Each sensory rootlet is caught with a blunt hook and divided (Fig. 356). When there is no pain in the distribution of the ophthalmic division of the nerve, the medial rootlets are left intact to preserve the sensory supply of the cornea. The motor root is covered by

the sensory rootlets, but can sometimes be recognised by its larger calibre and oblique course downward to the mandibular division. *If all the rootlets are divided, the patient must wear an eyeshield and must be given written instructions regarding the care of the eye.* The sensory root of the fifth cranial nerve may also be divided through a unilateral posterior fossa approach (*Dandy's operation*). *Trigeminal Tractotomy (Sjöqvist's operation).*—After the sensory rootlets of the trigeminal nerve enter the pons, the fibres conveying pain pass downward, forming the descending tract of the trigeminal nerve. These fibres have a cell station in the nucleus of the descending tract. The fibres from the first division of the nerve go to the *inferior* part of the nucleus; those from the second division to the intermediate part; and those from the third division to the *uppermost* part of the nucleus. The neurons conveying sensation of touch pass upward to the main sensory nucleus of the fifth nerve in the pons. Sjöqvist showed that it is possible to interrupt the pain fibres by division of the descending tract in the medulla oblongata, but owing to the arrangement of the fibres referred to above, it is difficult to obtain analgesia in the mandibular distribution of the nerve. The great advantage of the operation is that sensation of touch is preserved. Unfortunately, however, the operation has a limited application, for in the majority of cases of trigeminal neuralgia the mandibular division of the nerve is affected. An incision is made extending from the external occipital protuberance to the fourth cervical spine. The occipital muscles are separated in the midline to expose the occipital bone in the region of the foramen magnum. The posterior margin of the foramen magnum is removed. The dura mater is opened longitudinally. (Note.—The occipital sinus is occasionally rather large.) The arachnoid is punctured and separated from the medulla oblongata. The cerebellar tonsil on the side of the proposed tractotomy is retracted upward (Fig. 357). The landmarks are the obex at the inferior extremity of the fourth ventricle, the rootlets of the spinal part of the accessory nerve, and sometimes the descending tract itself which may produce a visible prominence, the *tuberculum cinereum*, lying anterolateral to the fasciculus cuneatus and posteromedial to the inferior olive. The incision is made into the descending tract 4 mm. caudal to the obex and to a depth of 3 mm. If the operation is performed under local anaesthesia, the area of sensory denervation can be checked, and if necessary the tractotomy can be extended or repeated at a higher level. However, there is the serious disadvantage that division of the tract is painful and the patient is likely to move.

**Dental Pain.** By the time patients with facial pain reach the neurosurgeon, some or all of the teeth have usually been removed. However, the mouth, including the teeth, should always be carefully examined.

**Herpes Zoster (Syn. Shingles).** The trigeminus is one of the most commonly affected nerves. The disease is caused by a virus which most

often attacks elderly people, and produces pain and small cutaneous vesicles in the distribution of the affected nerve. In the case of the trigeminal nerve, the ophthalmic division is most frequently affected. The corneal reflex is absent, and there is analgesia or hypalgesia (diminished sensation to pain) over the affected skin. If the cornea is involved, the eyelids should be sewn together (tarsorrhaphy). The vesicles are protected from secondary infection by 1 per cent. chloromycetin ointment applied aseptically. If the vesicles become secondarily infected unsightly

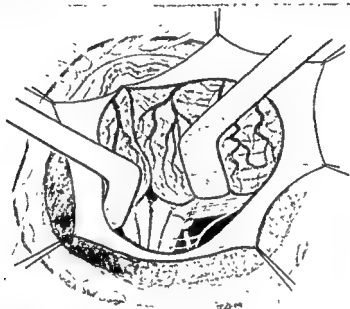


FIG. 357. Division of the descending tract of the trigeminal nerve. Note.—There is no visible dividing line between the two sensory tracts of the posterior column.

scarring occurs. There is a tendency for the pain to persist after the acute infection (post-herpetic "neuralgia"). This complication is most intractable to all forms of treatment including injection or section of the sensory root of the trigeminal nerve.

**Head Injury.** The *supraorbital nerve* is the part of the trigeminal nerve most commonly involved in head injuries. Damage to the nerve is caused by a blow on the forehead. There is usually no fracture. The *nasociliary nerve* may be injured in severe fractures of the frontal bone with comminution of the frontal and ethmoid sinuses; unilateral loss of sensation over the tip of the nose is produced. The *maxillary nerve* (including the infra-orbital nerve) is the second most commonly injured branch of the trigeminal. Damage to the nerve is usually associated with fractures of the maxilla. Other cranial nerves, especially the sixth, may be involved, although the resulting ophthalmoplegia is often temporary. Sensory loss



in the maxillary area after head injury is a sign of fracture of the maxilla. The infraorbital nerve may be injured in operations on the antrum, and severe intractable pain may result. Injury of the mandibular nerve is usually caused by fractures of the horizontal ramus of the mandible and sometimes by tooth extraction. The latter injury is due to downward pressure of the extracting forceps on the inferior alveolar canal which is thereby crushed. Recovery occurs in most cases. Injury of the *trigeminal ganglion and root* is uncommon. It may be caused by blunt injury of the skull, especially in fractures of the apex of the petrous bone, or by missiles. Permanent neurological defects most often follow the penetrating injuries. There is nearly always coincident injury of other cranial nerves. Patients complain of numbness rather than pain.

**Tumours of the Cerebello-pontine Angle.** Tumours in this situation affect the roots of the trigeminal nerve but pain is rarely produced (Chap. 53).

**Occlusion of the Posterior Inferior Cerebellar Artery,** or the *vertebral artery*, which usually gives origin to the former vessel, is commonly caused by atherosclerosis (atheroma). Occasionally, embolic occlusion occurs (e.g. in mitral stenosis or bacterial endocarditis). The resulting infarction of part of the lateral aspect of the medulla oblongata causes sudden onset of vertigo and vomiting. Loss of sensation to pain and temperature is found over the face on the affected side from involvement of the descending tract of the trigeminal nerve. Other effects are loss of pain and temperature sensibility on the *opposite* side of the body (limbs and trunk); homolateral cerebellar dysfunction (nystagmus, hypotonia and inco-ordination); homolateral Horner's syndrome; homolateral paralysis of the soft palate, pharynx and vocal cords (nucleus ambiguus). There may be persistent intractable pain in the face on the affected side and sometimes in the opposite limbs.

**Carcinoma of the Maxillary Antrum.** One of the characteristics of this disease is severe and intractable pain in the maxillary region. Other manifestations are nasal obstruction, discharge from the homolateral nostril (sometimes bloodstained), swelling of the antral region, proptosis, and epiphora from obstruction of the lachrymal duct.

**Tumours at the Foramen Magnum** (also Chap. 59). A meningioma or neurofibroma at this level may cause compression of the descending tract of the trigeminal nerve, for the tract extends down to the third cervical segment. The number of divisions of the trigeminal nerve affected depends on the extent of involvement of the descending tract. Nerve fibres from the first (ophthalmic) division are situated at the *lower* end of the tract, those from the second (maxillary) division in an intermediate position, and those from the third (mandibular) at the upper end. Paræsthesia in the face is caused rather than pain. Absence of the corneal reflex may be found before loss of pain and temperature sensation. Light touch is preserved, for the nerve fibres concerned go to the main sensory

nucleus in the pons without entering the descending tract. Tumours at the uppermost level of the spinal cord may also cause pressure on the descending tract of the trigeminal nerve.

**Meningiomas of Meckel's Cave.** These are rare tumours which produce a rather characteristic syndrome. At first they cause continuous pain, paræsthesia in the corresponding half of the face; hypalgesia in the same area; absence of the corneal reflex; and unilateral paralysis of the muscles of mastication. Later they burrow under the dura mater in both the middle and posterior fossæ. In the middle fossa, they cause ophthalmoplegia, and in the posterior fossa paralysis of the cranial nerves which arise there. Late in their history cerebellar signs appear, and there is raised intracranial pressure.

**Intrinsic Lesions of the Medulla Oblongata.** The nuclei of the descending tracts of the trigeminal nerves send fibres across the midline to form the trigeminal lemnisci (quinto-thalamic tracts). The fibres are arranged in such a way that a lesion expanding in the centre of the medulla oblongata (e.g. syringobulbia, glioma) causes, not a divisional loss of pain and temperature, but loss involving the posterior part of the face bilaterally and as the lesion progresses, loss of sensation advances forward so that the nose and lips are the last to be affected.

### THE FACIAL NERVE

The motor nucleus lies deep in the pons with the nucleus of the descending tract of the trigeminal nerve lateral to it (Fig. 358). Those neurons which supply the muscles of the upper half of the face, including the orbicularis oculi, receive pyramidal (cerebrospinal) axons from the facial part of the motor cortex of both cerebral hemispheres, whereas the neurons supplying the lower facial muscles, including the orbicularis oris, receive pyramidal axons from the opposite cerebral hemisphere only. Thus, upper motor neuron paralysis is chiefly confined to the lower half of the affected side of the face, whereas lower facial neuron paralysis involves the whole of one side of the face.

The lower motor axons from the facial nucleus have a peculiar course (Fig. 358). At first they run dorsally and medially until they reach the lateral aspect of the abducens nucleus where they turn medially behind the nucleus and, having almost completely encircled it, retrace their course towards the facial nucleus which they pass on its lateral side. The facial nerve then emerges on the anterior surface of the pons close to its inferior border and medial to the auditory nerve. Some of the efferent facial fibres originate in the superior salivary nucleus in

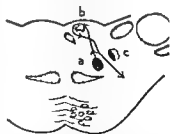


FIG. 358. The origin and course of the motor neurons of the facial nerve in the pons. (a) Nucleus of the facial nerve. (b) Nucleus of the abducens nerve. (c) Descending tract (and nucleus) of the trigeminal nerve.

the pons. They are the secretomotor fibres for the submandibular (submaxillary) and sublingual salivary glands. They leave the facial nerve via the chorda tympani (a mixed nerve—*vide infra*) which conveys them to the lingual nerve and the submandibular (submaxillary) ganglion. The sensory root of the facial nerve (nervus intermedius) takes origin in the ganglion of the facial nerve (geniculate ganglion) in the facial canal of the petrous bone, and runs centrally to terminate in the upper part of the nucleus of the tractus solitarius. It conveys sensation of taste (sweet, salt, bitter, acid) from the *sensory part* of the chorda tympani which carries taste fibres from the lingual nerve and therefore from the anterior two-thirds of the tongue.

The facial nerve, after leaving the pons, crosses the subarachnoid space, and accompanies the auditory nerve into the internal auditory meatus. Inside the meatus, it enters the facial canal which passes laterally above the vestibule of the labyrinth to the medial wall of the middle ear. It then makes a right-angled bend (site of geniculate ganglion), and runs backwards on the medial wall of the middle ear above the promontory of the fenestra vestibuli. On reaching the medial wall of the aditus of the tympanic antrum the facial nerve turns downwards and emerges on the inferior surface of the temporal bone at the stylomastoid foramen. The facial nerve immediately gives off a branch to the *posterior* belly of the digastric muscle and the stylohyoid muscle, and also the posterior auricular nerve which ascends behind the ear to supply the posterior and superior auricular muscles and the occipitalis. The facial nerve then passes through the space between the mastoid and styloid processes to enter the parotid gland. Within the gland the nerve passes lateral to the posterior facial vein and the external carotid artery. The following terminal branches are then given off: (a) The *temporal* branch emerging from the upper border of the parotid gland to supply the facial muscles above the zygoma and the frontalis; (b) the *upper zygomatic* branch running forward from the upper end of the anterior border of the parotid gland to the level of the zygoma and supplying the facial muscles in this locality; (c) the *lower zygomatic* branch running forward from the anterior border of the gland above the parotid duct and accompanied by the transverse facial vessels, supplying the muscles of the nose and upper lip; (d) the *buccal* branch which emerges from the anterior border of the gland below the duct to supply the buccinator and the orbicularis; (e) the *mandibular* branch appearing at the anterior border of the parotid gland and running forward close to the lower border of the mandible to supply the risorius and the lower half of the orbicularis oris; and (f) the *cervical* branch descending from the lower border of the gland below the angle of the mandible to supply the platysma.

#### LESIONS OF THE FACIAL NERVE

**In the Pons.** Very occasionally there is agenesis of the facial nuclei resulting in bilateral congenital facial palsy. Unilateral or bilateral facial

paresis may result when the pons is involved in the following diseases : Vascular occlusion or hæmorrhage, intrinsic tumour (glioma or metastasis), syringobulbia, disseminated sclerosis, progressive muscular atrophy, encephalitis and poliomyelitis. Surrounding structures are usually involved along with the facial nuclei.

**In Cerebello-pontine Angle.** The facial nerve may be affected with the acoustic nerve by sclerosing meningitis, including that caused by tuberculosis or syphilis. An *acoustic neurinoma* may cause little or no facial paresis, but after removal of the tumour paralysis is often complete (Chap. 53). Other tumours occurring in this situation are meningiomas, including those arising in Meckel's cave, and epidermoids (Chap. 53).

**In the Facial Canal.** The occurrence of facial palsy in chronic inflammation of the middle ear is suggestive of tuberculosis, especially in infants and children, or in adults with known phthisis. The nerve may be injured in the facial canal by fractures of the skull, gunshot wounds, mastoid operations, and occasionally during the operation of sensory root section of the trigeminal nerve for tic douloureux. When the temporal route is employed for the latter procedure, facial paralysis may arise from traction on the greater superficial petrosal nerve when the dura mater is being dissected off the upper surface of the petrous bone, for the greater superficial petrosal nerve is connected to the geniculate ganglion of the facial nerve. When the posterior fossa route is employed, damage to the facial and the auditory nerves may be caused by the retraction required for exposure of the fifth nerve. *Metastatic tumours* in the temporal bone account for a few cases of facial paralysis. *Epidermoids* may also occur in the petrous bone and cause facial paralysis and deafness (Chap. 58). *Nasopharyngeal tumours* (carcinomas, reticulosarcomas or lympho-epitheliomas), have a great tendency to invade the base of the skull and cause cranial nerve palsies before causing nasal obstruction. These tumours are treated by radiotherapy. *Carcinoma of the middle ear* causes local pain, a blood-stained aural discharge, deafness and facial paralysis. After a positive biopsy, wide excision is carried out, and is followed by radiotherapy. *Glomus jugulare tumours* arise in the jugular bulb from cells akin to those forming the carotid body. They grow slowly, and first invade the middle ear. Patients present with a bloody aural discharge and vascular polypi are observed in the ear. Cranial nerve palsies follow, and eventually the posterior fossa is invaded. At this stage, cerebellar signs are found and perhaps also evidence of raised intracranial pressure. A "bruit" may be heard on auscultation of the mastoid process. The diagnosis is confirmed by aural biopsy. Radiography shows erosion of the petrous bone in some cases. Radical surgery has a prohibitive mortality because of the dangerous situation of these tumours and their great vascularity. Radiotherapy alone is therefore recommended. *Glomus jugulare tumours* occur much more frequently in women than in men in the ratio of five to one.

**Bell's Palsy.** The onset of facial paralysis is sudden, and is often noticed on waking in the morning. Sometimes there is a history of exposure to a draught the day before. Taste is lost (*ageusia*) over the anterior two-thirds of the tongue when the chorda tympani is affected. Spontaneous recovery occurs in most cases (80 per cent.), but facial expression on the affected side is likely to be distorted, or mass movements (*synkinesia*) may occur, especially on smiling. Immediate treatment consists in frequent bathing of the eye and its protection with plain glasses fitted with a shield around the rim on the paralysed side. Terence Cawthorne advocates decompression of the facial nerve if after three months there is no clinical evidence of recovery and there is a reaction of degeneration. Josephine Collier, however, maintains that many patients in this group recover spontaneously. If electromyography detects functioning motor units after two to three weeks, there can be nothing to decompress. The few patients who do not recover can have their appearance greatly improved by facio-hypoglossal anastomosis, facio-accessory anastomosis, or the fascial sling operation.

**Ramsay Hunt's Syndrome** (herpes zoster of the geniculate ganglion). Pain occurs in the ear and mastoid region, and radiates to the tonsil and vertex. Vesicles are found in the auricle and sometimes on the anterior pillar of the fauces. Facial paralysis and loss of taste over the anterior two-thirds of the tongue occur on the affected side. Clonic facial spasm is sometimes observed. Recovery from the facial paralysis is often incomplete. There is no indication for surgery.

**In the Parotid Gland.** *Adenomas* of the parotid gland (formerly called mixed parotid tumours) usually grow slowly and only infrequently cause facial paralysis. *Carcinomas of the parotid*, on the other hand, are malignant tumours which grow rapidly and invade the branches of the facial nerve. Parotid adenomas tend to arise in the superficial part of the gland and can often be removed without producing facial paralysis. The capsule must always be removed with the tumour. Carcinomas are treated by wide excision and radiotherapy. The branches of the facial nerve may be injured by incisions made for *suppurative parotitis*. *Metastatic tumours* situated in the lymph gland behind the mandible may cause facial paralysis. *Sarcoidosis* is also an occasional cause. *Birth injury* of the facial nerve sometimes follows difficult forceps delivery; recovery always occurs. In warfare, the facial nerve, in its somewhat exposed position, may be injured by blast; recovery is the rule in surviving patients.

**Operations for Facial Palsy.** Facial palsy caused by fractures of the skull usually recovers spontaneously. Injuries of the nerve occurring during mastoid operations or in missile wounds usually need grafting, although sometimes end-to-end suture is feasible. Radical mastoidectomy is first performed to expose the facial canal, and the defect in the nerve is bridged by a graft taken from one of the cutaneous nerves of the thigh.

In the facial canal the graft requires no sutures, for it lies securely and accurately in apposition with the nerve stumps. If, however, a defect occurs in the facial nerve in the soft tissues (as in some missile injuries), the graft has to be sutured to the divided ends of the nerve. When the central end of the facial nerve is inaccessible (e.g. in facial palsy following removal of an acoustic tumour), anastomosis of the distal part of the nerve with the central end of the deliberately divided hypoglossal or accessory nerve is carried out: An incision is made in the neck along the anterior border of the sternomastoid muscle to expose these nerves. A single through-and-through suture is all that is required for the anastomosis. The descendens hypoglossi is divided and the proximal end sutured to the distal end of the donor nerve to maintain tone in the muscles deprived of their nerve supply. According to Lathrop, facio-hypoglossal anastomosis should be carried out on patients whose living depends on manual labour, and facio-accessory anastomosis on those whose occupation is sedentary. Penetrating wounds immediately anterior to the lobule of the ear may destroy the *pes anserinus* (the branching point, literally the goose's foot) of the facial nerve so that grafting or nerve anastomosis is impossible. The fascial-sling operation is then indicated (strips of fascia lata are fixed above to the temporalis fascia and passed downwards and inwards to the angle of the mouth and then across the midline of the upper and lower lips to be fixed into the orbicularis oris on the opposite side).

**Physiotherapy for Facial Palsy.** The aim is to diminish wasting and over-stretching of muscles while the nerve is regenerating. Treatment with *galvanism* is effective if given regularly and begun soon after the palsy has occurred. *Patients must be trained to restrict the movement of the sound side to avoid stretching the paralysed side.* Therefore exercises are avoided until re-innervation has occurred. They are then performed in front of a mirror. A plastic hook is attached to the upper teeth to keep the angle of the mouth in the neutral position. Light massage, performed by the patient, helps to maintain the circulation in the paralysed muscles.

### THE AUDITORY NERVE

This nerve consists of two parts, the cochlear and the vestibular nerves. The *cochlear nerve* is concerned with hearing. It arises in the *spiral ganglion* of the cochlea and runs medially in the petrous bone passing through the internal auditory meatus to reach and enter the lower border of the pons lateral to the facial nerve, and separated from it by the *vestibular nerve*. Most of the fibres end in the ventral and dorsal cochlear nuclei (Fig. 359). From the *ventral cochlear nucleus* new fibres run transversely across the pons to the opposite side, and ascend through the posterolateral part of the pons and midbrain as the *lateral lemniscus*, which terminates in the *inferior corpus quadrigeminum* and medial geniculate body (the lower auditory centres). From the *dorsal cochlear nucleus* fibres run medially beneath the floor of the fourth ventricle (*striæ auditoriæ*) to the midline

**Bell's Palsy.** The onset of facial paralysis is sudden, and is often noticed on waking in the morning. Sometimes there is a history of exposure to a draught the day before. Taste is lost (*ageusia*) over the anterior two-thirds of the tongue when the chorda tympani is affected. Spontaneous recovery occurs in most cases (80 per cent.), but facial expression on the affected side is likely to be distorted, or mass movements (*synkinesia*) may occur, especially on smiling. Immediate treatment consists in frequent bathing of the eye and its protection with plain glasses fitted with a shield around the rim on the paralysed side. Terence Cawthorne advocates decompression of the facial nerve if after three months there is no clinical evidence of recovery and there is a reaction of degeneration. Josephine Collier, however, maintains that many patients in this group recover spontaneously. If electromyography detects functioning motor units after two to three weeks, there can be nothing to decompress. The few patients who do not recover can have their appearance greatly improved by facio-hypoglossal anastomosis, facio-accessory anastomosis, or the fascial sling operation.

**Ramsay Hunt's Syndrome** (herpes zoster of the geniculate ganglion). Pain occurs in the ear and mastoid region, and radiates to the tonsil and vertex. Vesicles are found in the auricle and sometimes on the anterior pillar of the fauces. Facial paralysis and loss of taste over the anterior two-thirds of the tongue occur on the affected side. Clonic facial spasm is sometimes observed. Recovery from the facial paralysis is often incomplete. There is no indication for surgery.

**In the Parotid Gland.** *Adenomas* of the parotid gland (formerly called mixed parotid tumours) usually grow slowly and only infrequently cause facial paralysis. *Carcinomas of the parotid*, on the other hand, are malignant tumours which grow rapidly and invade the branches of the facial nerve. Parotid adenomas tend to arise in the superficial part of the gland and can often be removed without producing facial paralysis. The capsule must always be removed with the tumour. Carcinomas are treated by wide excision and radiotherapy. The branches of the facial nerve may be injured by incisions made for *suppurative parotitis*. *Metastatic tumours* situated in the lymph gland behind the mandible may cause facial paralysis. *Sarcoidosis* is also an occasional cause. Birth injury of the facial nerve sometimes follows difficult forceps delivery; recovery always occurs. In warfare, the facial nerve, in its somewhat exposed position, may be injured by blast; recovery is the rule in surviving patients.

**Operations for Facial Palsy.** Facial palsy caused by fractures of the skull usually recovers spontaneously. Injuries of the nerve occurring during mastoid operations or in missile wounds usually need grafting, although sometimes end-to-end suture is feasible. Radical mastoidectomy is first performed to expose the facial canal, and the defect in the nerve is bridged by a graft taken from one of the cutaneous nerves of the thigh.

epitheliomas of the canal interfere with hearing. *Primary otitis externa* (i.e. inflammation of the external meatus which is not secondary to otitis media) causes deafness only by blocking the external auditory canal with inflammatory debris—an important point in differential diagnosis from otitis media. Acute and chronic otitis media interfere with the function of the ossicles (i.e. the conducting mechanism; hence the term conductive deafness). Complete resolution of acute otitis media frequently occurs with complete restoration of hearing, but chronic infection causes some degree of permanent deafness. Prompt treatment of acute infection is therefore vital if hearing is to be preserved. One of the dangers of mastoiditis is that if it is not adequately treated permanent ankylosis of the ossicles tends to occur. In infants and young children or in patients with pulmonary tuberculosis, chronic otorrhœa may be caused by tuberculosis. Therefore the pus should be examined for tubercle bacilli. Occasionally chronic infection of the middle ear is caused by syphilis. Deafness is commonly caused by catarrhal blocking of the Eustachian tube. It may also occur in head injuries or may be produced by blast. In *otosclerosis* deafness is caused by ankylosis of the foot-piece of the stapes. This disease usually occurs in young adults. One form of *congenital deafness* affects the middle ear. *Neoplasms* (e.g. carcinoma of the middle ear and glomus jugulare tumours) are rare causes of middle-ear deafness. In the *internal ear*, *labyrinthitis* (secondary to otitis media, meningitis or mumps) causes perceptive deafness. Aplasia of the labyrinth is one of the causes of congenital deafness and therefore of *deaf-mutism*. *Senile deafness* commonly results from atrophy of the *ligamentum spirale*, beginning in the lower whorl of the cochlea and thus causing loss of hearing for high frequencies. *Boilermaker's deafness* is caused by the continuous noise of machinery. The auditory nerve may be affected in the petrous bone by *fractures*, tumours invading the bone from the nasopharynx, or metastasis from primary bronchial or thyroid carcinoma. Rare causes of auditory nerve paralysis in the petrous bone are: Epidermoids (pearly tumours, Chap. 58), osteitis deformans (Paget's disease), leontiasis ossea and platybasia (basilar impression). Lesions in the petrous bone also cause facial palsy. *Acoustic neurinomas* and other cerebello-pontine angle tumours produce their effect on the auditory nerve both within the internal auditory meatus and in the cerebello-pontine angle. The auditory nerve is very sensitive and may be permanently damaged by retraction in posterior fossa operations. The auditory nerve may be involved in meningitis (acute, tuberculous or syphilitic). Improvement in hearing has been reported to follow careful removal of meningitic adhesions. Streptomycin given over a long period may cause deafness by its action on the auditory nerve or labyrinth. Tumours in the pons occasionally cause deafness, and a very rare cause of bilateral deafness is pressure on the lower auditory centres (inferior colliculi and medial geniculate bodies) by a *pineal tumour* or an intrinsic tumour of



where they turn sharply ventrally, decussating with the corresponding fibres from the opposite side; they then turn dorsally to join the *lateral lemniscus* which, as previously stated, goes to the lower auditory centres. The latter connect with the superior temporal convolution. Unilateral destruction of the superior temporal convolution does *not* cause deafness, for hearing is bilaterally represented in the cerebral cortex. The *vestibular nerve* is concerned with equilibrium. It arises in the *vestibular ganglion* within the internal auditory canal.

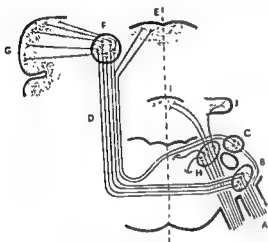


FIG. 359. The central connections of the auditory nerve. A. The cochlear nerve. B. The ventral cochlear nucleus. C. The dorsal cochlear nucleus. D. The lateral lemniscus. E. The inferior corpus quadrigeminum. F. The medial geniculate body. G. The superior temporal convolution. H. The vestibular nuclei. I. The nodulus. J. The flocculus.

The vestibular ganglion receives nerve fibres from the membranous semicircular canals, the utricle and the saccule. The vestibular nerve leaves the internal auditory meatus, and then passes to the pons medial to the cochlear nerve and lateral to the facial nerve. It joins the various vestibular nuclei which send axons to the nodulus and flocculi (these parts of the cerebellum are now known to be concerned with the maintenance of equilibrium).

#### Tests of Auditory Nerve Function.

These tests are employed to distinguish between deafness caused by lesions of the external auditory canal or middle ear (conductive deafness), and lesions of the labyrinth or auditory nerve including its central con-

nections (perceptive deafness). *Weber's test* is carried out with a vibrating tuning-fork (C.256) which is applied to the patient's vertex or forehead in the midline. Normally, the sound appears to originate in the midline. In conductive deafness bone transmission is enhanced on the affected side, and therefore the sound is referred to the side of the lesion. In perceptive deafness the sound is referred to the normal (opposite) side. In *Rinne's test* the vibrating tuning-fork is applied to the mastoid process while the external auditory meatus is closed by pressure on the tragus. When the patient can no longer hear the sound the fork is held at the open meatus; in conductive deafness the sound cannot be heard at the meatus when bone transmission ceases, but it can be heard in *incomplete* perceptive deafness. If the perceptive deafness is complete there will be no bone transmission whatever on the affected side. Deafness is also investigated by *audiometry*; loss of low-pitched tones is found in conductive deafness and loss of high-pitched ones in incomplete perceptive deafness.

**Causes of Deafness.** *Obstruction of the external auditory canal* is sometimes caused by wax and occasionally by foreign bodies; osteomas and

vomiting, nystagmus, loss of balance and past-pointing (if tested). The cause of Ménière's syndrome is not definitely known, but destruction of the affected labyrinth or division of the vestibular nerve stops the attacks, and the caloric tests may demonstrate defective vestibular function; the labyrinth therefore appears to be the site of the disorder. *Hydrops* of the membranous labyrinth has been described in some postmortem cases. *Treatment*.—A salt-free diet and restricted fluid intake are recommended on the basis of the hydrops just mentioned. Phenobarbitone is said to reduce the number and the severity of attacks. There are three surgical procedures which may be carried out when the side of the affected labyrinth can be demonstrated: (a) Alcoholic injection of the labyrinth; (b) opening of the bony external semicircular canal (i.e. the most accessible canal) and removal of a portion of the membranous labyrinth; this operation (labyrinthotomy) effectively arrests all labyrinthine function on the affected side; and (c) division of the vestibular part of the auditory nerve through a unilateral posterior fossa approach. With the aid of a blunt hook, the auditory nerve is easily separated from the more anteriorly placed facial nerve. If there is useful hearing, only the anterior half of the auditory nerve (vestibular part) is divided or crushed, but if the patient's cochlear function is absent on the affected side the whole of the auditory nerve is divided. Tinnitus often persists after operation.

*Post-traumatic Vertigo*. Head injury is the second commonest cause of severe vertigo (not to be confused with the mild variety of dizziness which is so often a sequel of concussion). Deafness and tinnitus may also occur. Diminished vestibular function is observed with persistent vertigo. Section of the vestibular nerve may be carried out if the lesion can be lateralised, but tinnitus usually persists.

*Labyrinthitis*. When the labyrinth is invaded in acute or chronic otitis media, acute labyrinthine symptoms are produced. In chronic otitis media there may be slight dizziness for several days before the onset of severe vertigo. At this early stage pressure on the ear increases the dizziness (the *fistula sign*). Radical mastoidectomy is required when labyrinthitis is caused by middle-ear and mastoid infection. In *meningitis*, infection may attack the labyrinth from the inside and this may sometimes happen bilaterally. *Mumps* is sometimes complicated by acute labyrinthitis. *Ramsay Hunt's syndrome* (acute facial palsy with a vesicular eruption in the external ear) may be complicated by acute labyrinthitis. Sudden labyrinthine symptoms may occur in *epidemics*, and they may follow excessive intake of alcohol, salicylates, and quinine, and long-continued treatment with *streptomycin*. Anæmia and vascular hypotension also cause dizziness.

*Central Causes of Vertigo*. Lesions of the vestibular nerve and its central connections, including the vestibular nucleus, flocculo-nodular lobe of the cerebellum or the temporal lobes, are much less often the cause of vertigo than lesions of the labyrinth, and, with few exceptions, the

midbrain. Deafness arising from cerebral lesions is extremely rare, for hearing is bilaterally represented in the superior temporal convolutions. One form of congenital deafness, however, is evidently due to lack of auditory perception in the brain; *idioglossia* (production of meaningless vocal sounds) results.

*Tinnitus* (ringing in the ears) is a symptom of lesions affecting the auditory system and is therefore frequently associated with deafness. It is akin to *paræsthesia*. Numerous patients complain of tinnitus without apparent cause. The known causes are: Wax in the external auditory canal; eustachian catarrh; acute otitis media; excess of certain drugs (e.g. quinine and salicylates); acute labyrinthitis; arteriosclerosis (atherosclerosis); hyperpiesia; anæmia; aortic incompetence; otosclerosis; intracranial arteriovenous aneurysms; intracranial angiomas and vascular malformations; meningitis; and acoustic tumours. Tinnitus is sometimes a manifestation of temporal lobe epilepsy or it may follow a head injury. Whenever possible the causal lesion should be treated appropriately. The symptomatic treatment of tinnitus is unsatisfactory.

**The Caloric Tests.** In neurosurgery, tests of vestibular function are most often used in the investigation of patients in whom an acoustic tumour is suspected. The tests are carried out with the patient lying down and the head raised by two pillows. The head is thus inclined approximately 30 degrees upward from the bed and the horizontal semi-circular canals are vertical. Cold water is run into the ear on the suspected side by means of a catheter and funnel. A current is produced in the endolymph as would occur with rotation of the head, except that the effect has the advantage of being unilateral. In the presence of a tumour compressing the vestibular nerve, the normal reaction of *nystagmus*, *vertigo*, *vomiting* and *past-pointing* does not appear, or is delayed. On the normal side the reaction should appear within about one minute. This test should not be carried out if there is a perforation of the drum.

**Vertigo.** The term vertigo refers to a *sensation* of movement which does not exist. It varies from a feeling of unsteadiness to a sensation of rotation in one direction and of the surroundings in the opposite direction. Loss of balance, vomiting and nystagmus may occur. If labyrinthine function is intact, vertigo can be deliberately produced by rotation, or by instilling warm or cold water into the external auditory canal (caloric test). Patients suffering from vertigo complain of giddiness or dizziness.

**Peripheral Causes of Vertigo.** The great majority of attacks of severe vertigo in which there is a feeling of rotation have their origin in the labyrinth.

*Ménière's syndrome* is the commonest example. It occurs in middle age, affecting men more than women in the proportion of three to two. Patients have tinnitus which is accentuated during the attacks, and there is progressive deafness of one or both ears. During attacks, all the features of acute labyrinthine dysfunction are observed: Vertigo,

the occipital protuberance (Fig. 384). The bone of the posterior fossa is removed on the affected side and the dura mater opened. The cerebellum is retracted upwards immediately lateral to the base of the cerebellar tonsil. The spinal accessory nerve comes into view first, followed by the vagus nerve and then the glossopharyngeal nerve which is thus the most anteriorly placed of this group of nerves; it is separated slightly from them by a dural septum as they pass out of the skull through the jugular foramen. The glossopharyngeal nerve is gently separated from the others and divided.

### THE VAGUS AND BULBAR ACCESSORY NERVES

The vagus is connected with the nucleus ambiguus, dorsal nucleus and the nucleus of the tractus solitarius. The bulbar part of the accessory nerve is entirely a motor nerve connected only with the nucleus ambiguus; its fibres soon join the vagus through which they are distributed, providing the vagus with most of its motor fibres for the pharynx and larynx and possibly also for the heart. The vagus receives sensory fibres from behind the ear and deep inside the external auditory meatus (auricular branch), from the posterior surface of the epiglottis, the piriform fossa, and the larynx above the vocal cords (via the internal laryngeal branch of the superior laryngeal nerve), from the larynx below the vocal cords (recurrent laryngeal nerve), from the trachea, bronchi, œsophagus and the heart. Afferent vagus fibres have not been definitely traced below the stomach. Motor fibres of the vagus are distributed to the larynx and pharynx. The *superior laryngeal nerve* supplies the cricothyroid (the tensor of the vocal cords), and the inferior constrictor muscle. The *recurrent laryngeal nerve* supplies all the other intrinsic laryngeal muscles. The vagus also supplies motor fibres to the thoracic and most of the abdominal viscera.

*Clinical Tests.* The movement of the palate is observed when the patient says "ah." In unilateral paralysis the palate moves away from the paralysed side. If the base of the tongue is depressed with a spatula the posterior wall of the pharynx may also be seen to move away from the paralysed side (*signe de rideau*). The palatal and pharyngeal reflexes are elicited on each side by a touch with cotton wool on a stick. The sensory component of the palatal reflex (i.e. the sensory supply of the inferior surface of the soft palate) is derived from the trigeminal nerve, and that of the pharyngeal reflex from the glossopharyngeal nerve. The motor component of both reflexes is mostly derived from the vagus. Unilateral pharyngeal paralysis causes no symptoms, but bilateral paralysis causes dysphagia. *Unilateral total laryngeal paralysis* causes hoarseness, but the opposite vocal cord is eventually able to cross the midline and meet the paralysed one which lies in the neutral or cadaveric position. A lesion anywhere between the nucleus ambiguus and the termination of the recurrent laryngeal nerve can cause this type of

vertigo is mild (giddiness). *Deafness, except with acoustic tumours, usually does not accompany vertigo of central origin.* Instead, signs of involvement of the central nervous system are found.

The *acoustic tumour* (Chap. 53) grows slowly and rarely causes severe rotatory vertigo although mild dizziness is a common symptom. *Occlusion of the posterior inferior cerebellar artery* by atherosclerosis (atheroma) or embolus—a sudden event—causes among other things sudden severe vertigo which, however, soon passes off. Infarction includes the vestibular nucleus and the flocculo-nodular lobe of the cerebellum. *Disseminated sclerosis and syringobulbia* sometimes affect the vestibular nucleus or its nerve fibres and thus cause vertigo. In disseminated sclerosis the onset may be sudden and severe, but the attack of vertigo soon passes off. *Temporal lobe epilepsy* sometimes begins with an aura of vertigo and might be confused with severe attacks of Ménière's syndrome. *Intracranial tumours* often cause dizziness.

### THE GLOSSOPHARYNGEAL NERVE

The central nuclei of this nerve are situated in the medulla oblongata, and form part of the nuclear system which serves also the vagus and bulbar part of the accessory nerve (i.e. the nucleus ambiguus, dorsal nucleus and nucleus of the tractus solitarius). The glossopharyngeal nerve runs in intimate contact with the vagus and bulbar accessory nerve in its intracranial course, although at its exit from the skull through the anterior part of the jugular foramen, the *glossopharyngeal nerve occupies a separate dural sheath*. The motor part of the nerve supplies the stylopharyngeus and gives off the secretomotor fibres to the parotid gland (tympenic branch of the glossopharyngeal nerve → tympanic plexus → lesser superficial petrosal nerve → otic ganglion → auriculo-temporal nerve → parotid gland). The sensory part supplies: (a) The posterior third of the tongue and the anterior surface of the epiglottis, both with ordinary sensory fibres and taste fibres; (b) the tonsils; (c) the free edge and superior surface of the soft palate; (d) the pharynx; (e) the eustachian tube and the middle ear. The glossopharyngeal nerve is anatomically and functionally so closely related to the vagus and bulbar part of the accessory nerve that clinical tests involve all these nerves (*vide infra*), and more often than not they are affected together in disease. Glossopharyngeal neuralgia is an exception.

**Glossopharyngeal Neuralgia.** Neuralgia of the ninth cranial nerve is characterised by recurrent attacks of severe pain in the tonsillar region, pharynx and middle ear. The pain lasts for a few seconds or minutes, and is often brought on by swallowing, yawning or clearing the throat. Most of the patients affected are over fifty years of age. Intracranial division of the glossopharyngeal nerve gives excellent results. The operation is carried out through a unilateral posterior fossa approach. A longitudinal incision is made midway between the mastoid process and

the occipital protuberance (Fig. 384). The bone of the posterior fossa is removed on the affected side and the dura mater opened. The cerebellum is retracted upwards immediately lateral to the base of the cerebellar tonsil. The spinal accessory nerve comes into view first, followed by the vagus nerve and then the glossopharyngeal nerve which is thus the most anteriorly placed of this group of nerves; it is separated slightly from them by a dural septum as they pass out of the skull through the jugular foramen. The glossopharyngeal nerve is gently separated from the others and divided.

### THE VAGUS AND BULBAR ACCESSORY NERVES

The vagus is connected with the nucleus ambiguus, dorsal nucleus and the nucleus of the tractus solitarius. The bulbar part of the accessory nerve is entirely a motor nerve connected only with the nucleus ambiguus; its fibres soon join the vagus through which they are distributed, providing the vagus with most of its motor fibres for the pharynx and larynx and possibly also for the heart. The vagus receives sensory fibres from behind the ear and deep inside the external auditory meatus (auricular branch), from the posterior surface of the epiglottis, the piriform fossa, and the larynx above the vocal cords (via the internal laryngeal branch of the superior laryngeal nerve), from the larynx below the vocal cords (recurrent laryngeal nerve), from the trachea, bronchi, œsophagus and the heart. Afferent vagus fibres have not been definitely traced below the stomach. Motor fibres of the vagus are distributed to the larynx and pharynx. The *superior laryngeal nerve* supplies the cricothyroid (the tensor of the vocal cords), and the inferior constrictor muscle. The *recurrent laryngeal nerve* supplies all the other intrinsic laryngeal muscles. The vagus also supplies motor fibres to the thoracic and most of the abdominal viscera.

*Clinical Tests.* The movement of the palate is observed when the patient says "ah." In unilateral paralysis the palate moves away from the paralysed side. If the base of the tongue is depressed with a spatula the posterior wall of the pharynx may also be seen to move away from the paralysed side (*signe de rideau*). The palatal and pharyngeal reflexes are elicited on each side by a touch with cotton wool on a stick. The sensory component of the palatal reflex (i.e. the sensory supply of the inferior surface of the soft palate) is derived from the trigeminal nerve, and that of the pharyngeal reflex from the glossopharyngeal nerve. The motor component of both reflexes is mostly derived from the vagus. Unilateral pharyngeal paralysis causes no symptoms, but bilateral paralysis causes dysphagia. *Unilateral total laryngeal paralysis* causes hoarseness, but the opposite vocal cord is eventually able to cross the midline and meet the paralysed one which lies in the neutral or cadaveric position. A lesion anywhere between the nucleus ambiguus and the termination of the recurrent laryngeal nerve can cause this type of

paralysis. *Unilateral abductor paralysis* is the usual result of a unilateral lesion of the recurrent laryngeal nerve. The affected vocal cord lies at or near the midline. Phonation and coughing are not affected, although there may be inspiratory stridor (during normal inspiration abduction of the cord occurs). *Bilateral total laryngeal paralysis* may be caused by bilateral lesions anywhere between the nucleus ambiguus and the termination of the recurrent laryngeal nerves. Both cords assume the cadaveric position. Phonation and coughing are impossible. There is no dyspnoea but inspiratory stridor occurs. *Bilateral abductor paralysis* may result from bilateral nuclear lesions or lesions of both recurrent laryngeal nerves. The voice is little affected and coughing is normal, but there is marked dyspnoea and stridor. Tracheotomy may be required. N.B.—There is no satisfactory explanation for the abductor paralysis usually occurring with lesions of the recurrent laryngeal nerves which supply both abductors and adductors of the vocal cord. *Bilateral adductor paralysis* is usually hysterical. Phonation is impossible, for without adduction the patient can only whisper. Adduction occurs on coughing in these cases.

**Lesions of the Vagus and its Branches. Nuclear Lesions.** Bulbar palsy (*bulb = medulla oblongata*) may be caused by vascular occlusion, syringobulbia, intrinsic tumour, progressive muscular atrophy, encephalitis lethargica, anterior poliomyelitis, rabies, Landry's paralysis or polyneuritis (e.g. in diphtheria). There is palatal, laryngeal and pharyngeal paralysis when the nuclei are bilaterally involved. Bilateral abductor paralysis of the larynx alone can be caused by a nuclear lesion. Acute lesions also cause marked slowing of the pulse and respiration, with a rising blood pressure and eventual loss of consciousness.

**Posterior Fossa Lesions.** The vagus, glossopharyngeal, accessory and sometimes hypoglossal nerves may be involved together by tumours (e.g. acoustic tumours or meningiomas). They may also be invaded by malignant tumours of the nasopharynx, glomus jugulare tumours or secondary tumours of bone. The term *pseudobulbar* paralysis refers to bulbar paralysis caused by bilateral lesions of the supranuclear pathways of the bulbar nuclei. The usual cause is widespread cerebral atherosclerosis (atheroma).

**Nerve Lesions.** Unilateral injury of the vagus may occasionally be caused by percutaneous angiography. The left recurrent laryngeal nerve, because of its special relations with the aorta, is more vulnerable than the right. It may be compressed by an aneurysm of the aorta or occasionally by an enlarged auricle in mitral stenosis. Either or both recurrent laryngeal nerves may be damaged during a difficult thyroidec-tomy. This is a recognised hazard, and may happen when the operation is performed by a surgeon of great skill and experience. Malignant glands are particularly liable to cause paralysis of these nerves.

**The Spinal Accessory Nerve.** The *bulbar* part of the accessory nerve has already been discussed. The *spinal* part is derived from cells in the

lateral part of the anterior horn of the grey matter of the spinal cord from the first to the fifth cervical segments. The rootlets emerge from the cord between the anterior and posterior cervical nerve roots and posterior to the denticulate ligament. The rootlets join to form the nerve which runs upward and passes into the skull through the foramen magnum and then soon passes out through the jugular foramen in the same dural sheath as the vagus. The spinal accessory nerve supplies the sternomastoid muscle and the upper half of the trapezius (the lower half is supplied by the cervical plexus, C.3 and C.4). Lesions of the spinal accessory nerve cause weakness of rotation of the head to the opposite side, and winging of the scapula. At rest the scapula is rotated downwards and outwards so that the winged inferior angle is closer to the spine than the superior angle. This position is exaggerated when the shoulder is abducted against resistance, but on forward flexion the winging of the inferior angle almost disappears. There is drooping of the shoulder and atrophy of the upper part of the trapezius. Patients are unable to raise the arm above the horizontal, and raising the shoulder is severely impaired.

*Lesions of the Spinal Accessory Nerve.* Nuclear lesions may occur in poliomyelitis, progressive muscular atrophy, syringomyelia and tumours of the cervical cord. Nerve lesions may be found with platybasia (basilar impression), meningiomas of the posterior fossa and foramen magnum, malignant tumours of the nasopharynx and glomus jugulare tumours. Other cranial nerve lesions occur at the same time. In the neck, the spinal accessory nerve may be affected alone by malignant glands. It is sometimes damaged during dissection of glands or by gunshot wounds or other types of penetrating injuries. It is one of the nerves which may be used for anastomosis with the peripheral stump of the divided facial nerve in persistent facial palsy.

*The Hypoglossal Nerve.* The hypoglossal nucleus lies in the medulla oblongata just under the floor of the fourth ventricle and close to the midline. The efferent axons pass forward across the medulla to reach the surface between the pyramid and the olive. After crossing the subarachnoid space, the rootlets join together and pass through the anterior condylar canal. Outside the skull, the hypoglossal nerve comes into intimate relationship with the ninth, tenth and eleventh cranial nerves between the internal jugular vein and the internal carotid artery, and then curves sharply forward just below the origin of the occipital artery from the external carotid artery, and above the hyoid bone to enter the tongue. The hypoglossal nerve supplies the muscles of the tongue. A unilateral lesion causes hemiatrophy, and the protruded tongue deviates towards the paralysed side (protrusion being impossible on the paralysed side). Speech and swallowing are not affected. In bilateral lesions the tongue cannot be protruded at all. Speech, and to some extent swallowing, are affected. The hypoglossal nerve tends to be involved in disease along with the other nerves of the medulla oblongata in the syndromes of



paralysis. *Unilateral abductor paralysis* is the usual result of a unilateral lesion of the recurrent laryngeal nerve. The affected vocal cord lies at or near the midline. Phonation and coughing are not affected, although there may be inspiratory stridor (during normal inspiration abduction of the cord occurs). *Bilateral total laryngeal paralysis* may be caused by bilateral lesions anywhere between the nucleus ambiguus and the termination of the recurrent laryngeal nerves. Both cords assume the cadaveric position. Phonation and coughing are impossible. There is no dyspnoea but inspiratory stridor occurs. *Bilateral abductor paralysis* may result from bilateral nuclear lesions or lesions of both recurrent laryngeal nerves. The voice is little affected and coughing is normal, but there is marked dyspnoea and stridor. Tracheotomy may be required. N.B.—There is no satisfactory explanation for the abductor paralysis usually occurring with lesions of the recurrent laryngeal nerves which supply both abductors and adductors of the vocal cord. *Bilateral adductor paralysis* is usually hysterical. Phonation is impossible, for without adduction the patient can only whisper. Adduction occurs on coughing in these cases.

**Lesions of the Vagus and its Branches.** *Nuclear Lesions.* Bulbar palsy (*bulb* = *medulla oblongata*) may be caused by vascular occlusion, syringobulbia, intrinsic tumour, progressive muscular atrophy, encephalitis lethargica, anterior poliomyelitis, rabies, Landry's paralysis or polyneuritis (e.g. in diphtheria). There is palatal, laryngeal and pharyngeal paralysis when the nuclei are bilaterally involved. Bilateral abductor paralysis of the larynx alone can be caused by a nuclear lesion. Acute lesions also cause marked slowing of the pulse and respiration, with a rising blood pressure and eventual loss of consciousness.

*Posterior Fossa Lesions.* The vagus, glossopharyngeal, accessory and sometimes hypoglossal nerves may be involved together by tumours (e.g. acoustic tumours or meningiomas). They may also be invaded by malignant tumours of the nasopharynx, glomus jugulare tumours or secondary tumours of bone. The term *pseudobulbar paralysis* refers to bulbar paralysis caused by bilateral lesions of the supranuclear pathways of the bulbar nuclei. The usual cause is widespread cerebral atherosclerosis (atheroma).

*Nerve Lesions.* Unilateral injury of the vagus may occasionally be caused by percutaneous angiography. The left recurrent laryngeal nerve, because of its special relations with the aorta, is more vulnerable than the right. It may be compressed by an aneurysm of the aorta or occasionally by an enlarged auricle in mitral stenosis. Either or both recurrent laryngeal nerves may be damaged during a difficult thyroidec-tomy. This is a recognised hazard, and may happen when the operation is performed by a surgeon of great skill and experience. Malignant glands are particularly liable to cause paralysis of these nerves.

**The Spinal Accessory Nerve.** The bulbar part of the accessory nerve has already been discussed. The spinal part is derived from cells in the

## CHAPTER 53

### INTRACRANIAL TUMOURS

#### DIAGNOSIS

A DETAILED history is taken from a near relation, for that given by the patient may be vitiated by the effects of the tumour. An accurate description of fits may have great localising value. Early events tend to have more localising significance than those occurring late in the history, for as tumours grow they may affect structures at a distance from their site of origin, or they may cause displacement of the brain and thus produce false localising signs.

The physical examination should be complete, for the nervous system may be affected by disease in any part of the body. Neurological examination begins with observations on the mental state, which is sometimes affected directly by the tumour (e.g. the apathy found with frontal tumours), or indirectly by an increase in intracranial pressure. Attention is paid to memory and speech, including reading and writing ; and it is recorded whether patients are right- or left-handed. The head is palpated for swellings or tenderness, for either may sometimes be found in association with underlying intracranial tumours (e.g. some meningiomas). The neck is tested for stiffness, for it occurs in advanced degrees of raised intracranial pressure, and is thought to be a reflex response to herniation of the cerebellar tonsils through the foramen magnum. Auscultation of the skull may reveal the murmur of an arteriovenous aneurysm, or vascular malformation. The *cranial nerves* are all examined. They are discussed in detail in Chap. 52.

Examination of the *upper limbs* begins with the patient holding them both outstretched with the eyes closed. One of the early signs of a frontal lesion, even before weakness can be detected, is the falling slowly downward of the *contralateral* extremity. In cerebellar lesions, the *homolateral* limb tends to drop slowly downward and also to deviate laterally. Power, tone, co-ordination, quickly repeated movements, sensation (pain, touch, stereognosis, two-point sense and position sense), and the reflexes are examined. *Hoffmann's reflex* is a dependable sign of involvement of the pyramidal tract (quickly flexing and releasing the terminal phalanx of the patient's middle finger, causes flexion of the fingers and thumb in pyramidal lesions). The lower limbs are examined for power, tone, co-ordination and sensation (pain, touch and position sense). A positive Babinski response and/or absence of superficial reflexes (abdominal, cremaster and gluteal) occur contralaterally in pyramidal lesions. An equivocal Babinski response on one side with a definitely downgoing toe

bulbar and/or pseudobulbar palsy, or by invasion by tumours of the nasopharynx. It may be injured during dissections of the internal and external carotid arteries, and it is sometimes deliberately divided and joined to the peripheral end of the facial nerve in some cases of facial palsy. Hemiatrophy of the tongue occurs in the syndrome of hemiatrophy of the face, and occasionally as a result of pressure by aneurysms of the vertebral artery. The commonest cause of bilateral hypoglossal palsy is *progressive muscular atrophy*.

Increase in width, tortuosity, or number of the vascular markings of the skull may be found with a meningioma. Some meningiomas cause increased growth in adjacent bone (Fig. 362) whereas others erode bone. The lungs are X-rayed, for carcinoma of the bronchus is the commonest source of intracranial metastasis. Sometimes the primary tumour of the bronchus is too small to be identified in the chest films.

Angiography of the cerebral vessels was introduced by Moniz of Lisbon in 1927. *Diodone* (pharmacopoeial name for proprietary products Pyelosil, Perabrodil, Diodrast and Umbradil), an iodine-containing

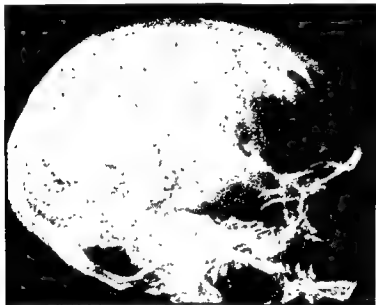


FIG. 361. Calcification of a small part of a large meningioma growing from the falx cerebri to within 1.5 cm. of the parietal cortex.

substance, also used for intravenous pyelography, is the medium now universally employed for arteriography. Unlike *iodoxyl* (i.e. Pyelectan and Uroselectan B) it is not irritating to the tissues. Originally, the common carotid artery was injected after exposure through an incision in the neck, but nowadays the percutaneous method is employed, although the open method is occasionally necessary when the percutaneous technique fails. The following apparatus is required: A supply of freshly sharpened, short-bevelled, 17-gauge needles, 9 cm. long; a piece of pressure tubing 25 cm. long with a needle adapter at one end and a syringe adapter at the other (a small section of glass tubing is incorporated in the rubber tubing); 20-ml. and 10-ml. syringes; and finally a lead-rubber screen for the protection of the operator and his assistant.

General anaesthesia is preferred to local anaesthesia, for conscious patients experience an unpleasant burning sensation in the head after

on the other is accepted as a positive sign. *Oppenheim's sign* is sometimes useful when excessive sensitivity of the feet causes difficulty in interpretation of the Babinski reflex (*Oppenheim's sign* : firm pressure along the tibia causes upward deflection of the big toe in pyramidal lesions). It cannot be too strongly emphasised that minor disturbances of neurological function should be recorded. The neurosurgeon cannot afford to ignore an unobtrusive sign for the sake of establishing a syndrome. After the clinical evidence has been considered, the likely location and pathology of

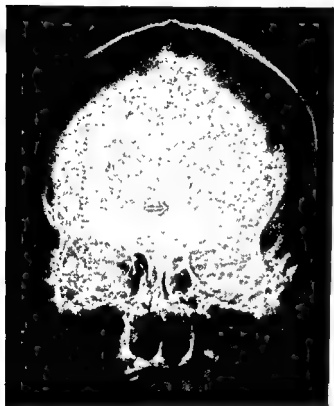


FIG. 360. Displacement of the calcified pineal gland to the patient's left by a tumour in the right cerebral hemisphere.

the tumour are decided. *The anatomical site must always be discussed before the pathology.* Appropriate investigations then follow the clinical opinion.

**Radiography.** Any or all of the following signs of long-standing raised intracranial pressure may be found : (a) Rarefaction or destruction of the posterior clinoid processes ; (b) exaggerated convolucional markings (beaten-silver sign) ; (c) flattening and enlargement of the pituitary fossa ; (d) thinning of the greater wing of the sphenoid ; and (e) separation of the sutures (diastasis) in infants. If the pineal gland is calcified, it may be seen displaced away from the side of the tumour (Fig. 360). Calcification can be demonstrated in some of the following tumours : Meningiomas (Fig. 361), astrocytomas, ependymomas, oligodendrogliomas and angiomas. Suprapituitary cysts are almost always calcified.

flow of saline is resumed until the radiographer is ready for the antero-posterior projections. The anteroposterior exposures are longer, therefore only four films are used. Early films show the arteries (arteriogram) and the late films show the veins (venogram). If filling on both sides is desired from a unilateral injection, the contralateral common carotid artery is compressed at the time of injection.

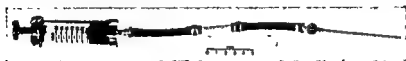


FIG. 363. The syringe, tubing and needle assembled for percutaneous arteriography.

The *vertebral artery* can also be injected percutaneously. The operator presses firmly with his left thumb immediately lateral to the upper border of the thyroid cartilage to displace the carotid sheath laterally. The needle is then inserted through the skin until it strikes a transverse process which can usually be felt with the tip of the index finger. The needle is directed upward through the transverse foramen. The rest of



FIG. 364. Showing the method of piercing the artery, after disconnecting the syringe. The artery is represented by a piece of rubber tubing.

the procedure is similar to that used for carotid angiography, but the diodone must be injected very quickly if good contrast is to be obtained. The following arteries are visualised: The vertebral (sometimes reflux filling of the opposite vertebral also occurs), the basilar, the homolateral posterior inferior cerebellar (just above the foramen magnum), and the superior cerebellar and the posterior cerebral arteries on both sides. The isolated mass of small posterior thalamic arterioles, arising from the posterior cerebral arteries, may also be seen. Sometimes the posterior communicating artery and parts of the middle and anterior cerebral arteries are filled. The possible complications of angiography are: (a) Hæmatoma of the neck (prevented by firm pressure on the neck after

injection, and are therefore likely to move. The operator stands on the patient's right side for the injection of either common carotid artery. The skin at the site of injection is first pierced with a pointed tenotome or sharp needle to prevent blunting of the angiography needle. The latter, with the tubing and syringe of saline (20 ml.) attached (Fig. 363), is thrust through the skin, and then the syringe is disconnected to allow the operator freedom of movement (Fig. 364). The needle and tubing remain



FIG. 362. An increased growth of bone of the inner table in the left parietal bone produced by an underlying meningioma.

filled with saline so that there is no fear of air embolus when the syringe is reconnected. The common carotid artery is found by palpation, steadied by the fingers, and *transfixed by a sudden jab of the needle*. The needle is then slowly *withdrawn* until entry of the point into the artery is shown by a *rapid flow of drops of saline* from the free end of the tube and blood flowing into the glass connection. A 20-ml. syringe of normal saline is once again attached to the rubber tube and the assistant delivers a steady flow of saline until the radiographer is ready and the protective screen has been placed in position. A 10-ml. syringe filled with 42.5 per cent. diodone replaces the 20-ml. syringe; the contrast medium is then injected quickly and five lateral exposures made in rapid succession. The

In angiograms, a meningioma is suggested by : (a) Sharp deflections of cerebral arteries ; (b) a circumscribed and smooth outline of the vascular pattern of a tumour ; (c) a blood supply partly derived from external carotid branches ; or (d) signs of a tumour at one of the classical sites for meningiomas (e.g. the olfactory groove). A glioblastoma multiforme (the common type of malignant glioma) is suggested by : (a) The presence of arteriovenous communications in the tumour ; (b) the ill-defined boundary of any tumour pattern ; and (c) lack of vascular displacement in the presence of a large tumour pattern. A cystic tumour is suggested by marked vascular displacement around a *completely* avascular zone. However, too much reliance must not be placed on the above characteristics. *The type of tumour must always be verified by biopsy or exploration.*

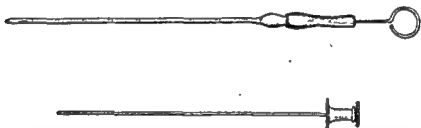


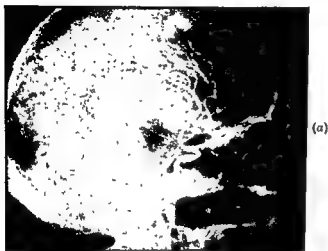
FIG. 366. Two types of brain needle or cannula. The upper one (Cushing pattern) is fitted with a short piece of rubber tubing to prevent rocking of the needle when a syringe is attached. The lower one, designed by Jefferson, is much thinner and is used without a rubber connection.

**Ventriculography.** Under local anaesthesia an incision about 1 inch long is made in the posterior parietal region on each side, 3 to 4 cm. from the midline and 5 to 10 cm. upward from the external occipital protuberance. Burr-holes and dural incisions are made, and brain cannulae (Fig. 366) are passed through the brain into each lateral ventricle. When ventriculography is indicated, the ventricles are often abnormal in size and position. Entry of the cannula into the ventricle on the side of a supratentorial tumour may be difficult or impossible. Therefore when the operator has difficulty on one side, an attempt should soon be made to enter the ventricle on the other side. The ventricular pressure is measured, and cerebrospinal fluid is withdrawn, about 2 ml. at a time, and replaced with air. The air is first aspirated into a syringe through a wad of cotton wool which acts as a filter. The syringe is then attached to the brain cannula and the air injected slowly into the ventricles. If air is injected too quickly, the ventricles may expel most of it and there may be difficulty in filling them again. The air is made to pass from one part of the ventricular



withdrawal of the needle); (b) cerebral embolus from thrombosis of the carotid artery; (c) cervical sympathetic paralysis (Horner's syndrome); (d) unilateral laryngeal paralysis; and (e) iodine skin reaction (very rare). Vertebral angiography is more difficult and considerably more risky than carotid angiography.

An advantage of angiography over ventriculography is that it does not



(a)

FIG. 365. (a) Arteriogram showing the characteristic upward curve of the middle cerebral artery produced by a temporal tumour. The burr-hole of a previously negative biopsy is also seen. (b) Sagittal section of the brain showing the temporal lobe glioblastoma multiforme which was localised by the angiogram illustrated in (a).



(b)

disturb intracranial pressure. It is therefore performed in preference to ventriculography when there are lateralising signs, for *unilateral* angiography may then give all the information required. Angiography may reveal the presence of a tumour by displacement of arteries or veins from their normal position, and sometimes by a "blush" produced by the tumour vessels. Angiography is of special value in the diagnosis of temporal fossa tumours which produce a characteristic upward curve of the middle cerebral artery (Figs. 365a and b). Ventriculography may fail to give convincing evidence of a tumour in this situation because the temporal horn may not fill with air.

accurate information about the third ventricle, the aqueduct and the fourth ventricle, than negative contrast ventriculography (Fig. 368). Ventriculography is indicated: (a) When there are no localising or lateralising signs; (b) for all posterior fossa tumours, except perhaps acoustic neurinomas which usually give rise to an unmistakable syndrome; and (c) when angiography fails to give all the desired information. The procedure should not be undertaken unless all neurosurgical facilities are available for the immediate removal of the tumour, as the patient's condition may deteriorate rapidly after ventriculography unless the tumour is removed.

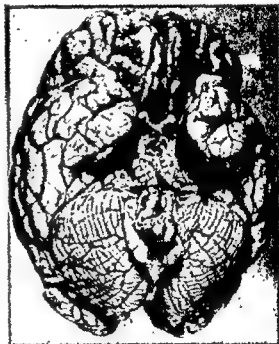


FIG. 369. A cerebellar pressure cone caused by a tumour in the posterior fossa. Note the deep groove separating the tonsils from the rest of the cerebellum. The cone blocks the foramen of Magendie causing hydrocephalus which, by forcing the cone further into the foramen magnum, produces compression of the medulla oblongata.

**Lumbar Puncture.** This investigation should not be carried out if there is clinical or radiological evidence of raised intracranial pressure. In such circumstances it does not contribute to the diagnosis, but may cause a rapid decline in the patient's condition by producing a temporal or cerebellar pressure cone (Fig. 369). Queckenstedt's test (compression of the internal jugular veins) must never be carried out when a space-occupying intracranial lesion is present or suspected, for the sudden rise in intracranial pressure accentuates the coning effect. When there is no clinical or radiological evidence of raised intracranial pressure, examination of the lumbar fluid may give valuable information when a tumour forms part of the differential diagnosis. If the patient is relaxed, pressures exceeding 160 mm. of cerebrospinal fluid may be significant, and if tests for syphilis are negative an increase in protein in the cerebrospinal fluid



FIG. 367. Displacement of the ventricles to the right by a left frontotemporal meningioma.

system to another by changing the position of the head. In this way radiographs of the various parts of the ventricles can be obtained. The position of the tumour is revealed by displacement and distortion of the ventricles (Fig. 367). Positive contrast ventriculography carried out with *Myodil* (2 to 3 ml. injected through a *frontal* burr-hole) gives more

FIG. 368. Lateral ventriculogram with air (negative contrast) showing the anterior horns of dilated lateral ventricles, and *Myodil* (positive contrast) showing the posterior end of the third ventricle and the aqueduct which is dilated and angulated forward. In the posterior fossa the triangular fourth ventricle is seen, and some *Myodil* has found its way into the occipital horn of one lateral ventricle and into the cisterna magna and spinal canal. These appearances are characteristic of a tumour in the posterior fossa. In this case it was an acoustic tumour.



completely free from risk and discomfort. There is no doubt that electroencephalography is extremely valuable, and should be carried out as a routine in all cases of suspected cerebral tumour.

**Burr-hole Biopsy.** An incision about 1 inch long is made through the scalp over the site of the tumour. Observations begin at the time of the skin incision, for shaving of the head may reveal a previously unnoticed swelling of the skull. A burr-hole is made in the skull. Excessive bleeding from the bone, especially if arterial, is suggestive of an underlying meningioma. A tense non-pulsating dura mater indicates raised intracranial pressure. Sometimes the burr-hole will show part of the puckered dura mater forming the attachment of a subjacent meningioma. A *subdural hæmatoma* (Chap. 50) may be revealed by the plum-coloured appearance of the dura, although a large cortical vein produces a similar effect. The dura mater is steadied with a sharp-pointed hook, and incised with a round-ended tenotome. An avascular part of the brain is pierced with a pointed tenotome, and a cannula passed inward. With practice the operator learns to feel with the cannula. Meningiomas are usually firmer than normal brain whereas gliomas may be either firmer or softer than the brain. Sudden diminution of resistance may indicate entry into a diffuent tumour, a cyst, an area of softening, or a ventricle. A flow of yellow or dark fluid occurs from cysts whereas clear fluid flows from the ventricles. The cyst may be visualised radiologically (cystography) by injection of 2 to 3 ml. diodone, or by replacement of the fluid with air. Diodone must *not* be injected into the ventricles. When a solid tumour is encountered, a biopsy specimen is obtained by aspiration, or if the tumour is too firm to be aspirated a piece is removed with a very small rongeur (a miniature of the pituitary rongeur).

The technique of neurosurgical diagnosis is tedious. It always begins with a detailed history and clinical examination which determine appropriate investigations. However hopeless the patient's condition, and however overwhelming the evidence in favour of a malignant tumour, the pathology must be proven by burr-hole biopsy or exploration.

### THE CLINICAL PRESENTATION OF TUMOURS

An expanding intracranial lesion may present with manifestations of : (a) Raised intracranial pressure alone ; (b) raised intracranial pressure and a regional neurological disturbance ; (c) a regional neurological disturbance without raised pressure ; or (d) raised intracranial pressure and false localising signs.

**Raised Intracranial Pressure.** *Headache* is the commonest symptom of intracranial tumours. It is most often bifrontal, but sometimes occurs unilaterally on the side of a *supratentorial tumour*. Occipital headache may occur when pressure is very high ; it is thought to be caused by herniation of the cerebellar tonsils through the foramen magnum. Headache associated with raised intracranial pressure is often worse after the

is strong evidence of an intracranial tumour. Malignant gliomas have a tendency to cause a moderate increase in white cells.

**Pneumoencephalography.** Radiological visualisation of the ventricles, sulci and cisterns is obtained by replacing cerebrospinal fluid with air by means of lumbar or cisternal puncture. In the presence of raised intracranial pressure this procedure is even more dangerous than diagnostic lumbar puncture. It is therefore carried out only when there is no evidence of increased intracranial pressure; the presence of a tumour may then occasionally be revealed. Failure of the air to enter the ventricles is suggestive of the presence of an intracranial tumour.

**Electroencephalography.** When the eyes are closed and the brain is relatively at rest, electrical waves, originating in the parieto-occipital lobes, can be recorded; in normal subjects they have a rate of about ten per second (alpha rhythm). Much faster waves (about twenty-five per second), the beta waves, and even faster ones, the gamma waves (both of low potential), are also sometimes found in apparently normal people, but have no application in tumour diagnosis. Brain tumours themselves are electrically inactive, but they often cause delta waves (three per second) in the cerebral cortex in their immediate vicinity. These slow waves, however, occur when the cerebral cortex is disturbed in other ways (e.g. in cerebral oedema, injury, alkalosis, etc.) and are therefore

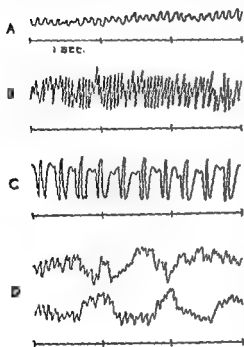


FIG. 370. E.E.G. recordings. (A) Alpha rhythm (10 per second)—normal. (B) Grand mal epilepsy (20 per second)—high potential waves. (C) Juvenile petit mal (3 per second)—waves and epikes. (D) Large delta waves (2 per second), in two leads with phase-reversal, in a patient with a cerebral tumour.

not specific for cerebral tumours. The focus of electrical abnormality produced by a space-occupying lesion is best localised by the demonstration of phase-reversal (Fig. 370). That is to say, the deflections of the delta waves point in opposite directions when simultaneous tracings are taken from two sides of the focus. The larger the amplitude of the waves, the larger is the mass of abnormally functioning brain, and the slower the rate of the waves, the more acute is the lesion. From this it follows that a slowly growing meningioma gives rise to a minimum abnormality, the average glioma to a moderate change, and an abscess to the most marked signs. Electroencephalography is not accurate enough to replace angiography or ventriculography, although it has the advantage of being

the patient is fully conscious. "*Cerebellar*" Signs.—Contralateral hypotonia and inco-ordination sometimes occur with frontal tumours, and occasionally there is nystagmus. This is of considerable practical importance, for tumours of the cerebellum itself tend to produce minimal localising signs. Signs of increased intracranial pressure are common with frontal tumours, but their absence does not exclude such tumours.

**Differential Diagnosis.** *Posterior Fossa Tumours.*—Slowly growing tumours in the posterior fossa tend to produce minimal or no cerebellar signs, and sometimes tumours of the frontal lobe may cause unilateral hypotonia, inco-ordination and even nystagmus. *Temporal Lobe Tumours.*—Anteriorly placed tumours of the temporal lobe are unlikely to cause defects of the visual fields, and may therefore be confused with tumours of the frontal lobe. *Intraventricular Tumours.*—Tumours in the ventricular system cause hydrocephalus without localising signs, and may therefore be mistaken for frontal tumours. *Hypertensive Encephalopathy.*—This is suggested by the presence of vascular hypertension, "nipping" of retinal veins, cardiac enlargement and albuminuria. However, a patient with these signs is not excluded from having a cerebral tumour. *Presenile Cerebral Atrophy* (of the Pick type).—In this condition dementia is likely to be more advanced than with a frontal tumour. *Cerebral Syphilis.*—Other signs of the disease, including a positive Wassermann's test, establish the diagnosis. Nevertheless, patients with a positive Wassermann and evidence of a space-occupying lesion, should be completely investigated as tumour suspects, and it is worthy of note that cerebral gummas do not respond to antisyphilitic treatment.

**Investigations.** *Radiography.*—Plain radiographs may show any of the features of raised intracranial pressure and occasionally calcification in a tumour. *Angiography.*—The anterior cerebral artery is often displaced towards the opposite side, and the middle cerebral artery and its branches may be deflected downward. Parasagittal meningiomas tend to produce a semicircular downward curve in the anterior cerebral artery as seen in lateral views. Olfactory groove meningiomas cause backward displacement of the anterior cerebral arteries. *Ventriculography.*—The antero-posterior view shows displacement of the ventricles towards the opposite side and deformity or absence of filling of the anterior horn on the affected side (Fig. 367). The third ventricle usually remains in the same antero-posterior plane as the septum lucidum whereas temporal tumours tend to cause angulation of these two structures.

#### PATHOLOGY OF TUMOURS OF THE FRONTAL REGION

**Gliomas**, as the name suggests, arise from the glia. Tumour cells infiltrate between the neurons and their processes, tending to produce no neurological disturbance until degeneration, hæmorrhage, or cyst formation occurs. A cyst is suspected when the onset of symptoms is sudden or when remissions occur. The commonest glioma in any region

patient has been lying down. Very severe lancinating headache tends to occur with posterior fossa tumours because they so easily obstruct the flow of cerebrospinal fluid. *Vomiting* is frequently associated with headache caused by raised intracranial pressure, and when the medulla oblongata is directly compressed by tumours, vomiting tends to be repetitive. *Neck stiffness* sometimes occurs when the pressure is high and it may be brought on by an ill-advised lumbar puncture. Other causes of neck stiffness should be kept in mind (meningitis, subarachnoid hæmorrhage, tumours in the cervical spinal canal, occipital fracture and disease or injury of the cervical vertebrae).

*Papilloedema* (swelling of the optic discs) is the most important sign of a space-occupying lesion, and is present in a large proportion of patients with intracranial tumours. It is discussed fully in Chap. 52. *Exophthalmos* is sometimes associated with raised intracranial pressure and may be more pronounced on one side than the other. *External Rectus Palsy*.—This is common and may be bilateral. It is caused by kinking of the abducens nerve(s) when the brain stem is displaced downward by a space-occupying lesion. The oculomotor nerve is sometimes affected instead of the abducens. Diplopia may occur when no squint is discernible on simple inspection. These external ocular palsies are signs of an expanding lesion only, and usually give no clue to its location; they are therefore "false localising signs." *Lividity* of the skin of the face and ears may be observed in the late stages of intracranial compression.

### TUMOURS OF THE FRONTAL REGION

**The Frontal Lobe Syndrome.** Lesions in this region give rise to personality changes which may be obvious to the clinician, or may be revealed only after searching enquiry. More important facts may be elicited from near relations than from patients. *Apathy* is the keynote of the syndrome. Thus there may be loss of interest in work or hobbies. There is a special type of incontinence associated with a frontal lobe lesion: patients although fully alert pass urine and faeces anywhere without shame. Mental *euphoria* (abnormal cheerfulness) is a common feature; patients are not impressed by the seriousness of the situation, and tend to regard the prospect of a major operation with unusual equanimity. A general blunting of mental processes (dementia) occurs; this is sometimes observed with lesions in other situations when marked hydrocephalus is present. Tumours in the dominant frontal lobe may cause motor aphasia.

Tumours extending backwards in the frontal lobe involve the motor cortex or pyramidal fibres. Epilepsy is a common manifestation of frontal lesions. The attacks are more often generalised than Jacksonian. *Grasp Reflex*.—Some frontal tumours produce a grasp reflex on the opposite side. The fingers automatically close around an object placed in the palm, and the grip is tenacious. The reflex is of localising value only when

growth of meningiomas appears sometimes to be initiated by trauma, for an overlying scar or fracture line may be found. Some meningiomas cause *hyperostosis* and some cause destruction of adjacent bone. The hyperostosis may be invaded by tumour cells and should therefore be removed at operation.

There are several histological varieties of meningiomas. The commonest is the *endotheliomatous* meningioma which is composed of whorls of polygonal cells separated by bundles of reticulum fibres. Numerous small calcified bodies (calcospherites) may be present. The term *psammoma* (psammos = sand) applies to meningiomas containing calcified bodies; it has no other histological significance. Calcification is sometimes sufficient to be revealed radiologically. In the *fibroblastic* type there are interlacing bundles of spindle cells, containing fibroglial fibres. *Angioblastic* meningiomas consist of masses of capillary spaces, and *myxomatous* meningiomas are composed of a mixture of spindle and stellate cells separated by mucin and reticulum fibrils.

*Parasagittal meningiomas* are those which arise from the superior sagittal (longitudinal) sinus or falx. Some of them are bilateral. Ligation and excision of the affected part of the venous sinus is permissible when the tumour arises from the anterior half of the sinus, but the posterior ligature must be well anterior to the veins draining the motor cortex. A thin layer of tumour tissue is left attached to the sinus when tumours arise further back. Angiography shows downward and sometimes lateral displacement of the anterior cerebral artery, and a vascular pattern is produced by some tumours. Meningiomas which arise from the falx without involving the superior longitudinal sinus can be completely removed with their dural attachment.

*Olfactory Groove Meningiomas.* In addition to the frontal syndrome, and perhaps raised intracranial pressure, these tumours invariably cause anosmia (loss of sense of smell) on one or both sides. Backward extension may affect one or both optic nerves and sometimes the chiasm. When only one of the optic nerves is compressed there may be optic atrophy with loss of visual acuity on the affected side, and papilloedema on the other side (Foster-Kennedy syndrome). In the early stages of compression of an optic nerve, examination of the visual fields shows a central scotoma. Plain radiography may show thickening of the floor of the anterior fossa, and angiography demonstrates upward and backward displacement of the anterior cerebral arteries. Olfactory groove meningiomas tend to envelop these arteries which are therefore liable to injury at operation. Clipping of one anterior cerebral artery may have no neurological effect, or it may cause fatal spasm or thrombosis extending into the circle of Willis. Clipping of both anterior cerebral arteries causes fatal coma or marked dementia and paralysis of both legs.

*Suprasellar Meningiomas* (Syn. Meningiomas of the Tuberculum Sellæ). These tumours arise from the tuberculum sellæ and grow upwards



of the *cerebrum* is the *glioblastoma multiforme* (syn. spongioblastoma multiforme). The glioblastoma is a very malignant tumour. On macroscopical examination, some glioblastomas appear to be circumscribed (Fig. 365b) whereas others have ill-defined limits and may involve two or more lobes of the brain. The opposite hemisphere may be invaded through the corpus callosum, or tumours arising in the corpus callosum may spread into both hemispheres. The cut surface of glioblastomas reveals characteristic dark areas of hæmorrhage (Fig. 365b) and sometimes cystic degeneration. The microscopic appearance often varies from one part of a tumour to another. The most characteristic feature is the great variation in the size and shape of the cells. Multinucleated neoplastic giant cells and mitosis are frequently observed. Some areas may have the appearance of an astrocytoma, supporting the current view that glioblastomas arise from astrocytomas. Thus many neuropathologists have abandoned the term glioblastoma and describe gliomas according to the Kernohan classification (grades 1, 2, 3 and 4). Grade 1 corresponds with the astrocytoma, a relatively benign tumour, and grade 4 with the glioblastoma multiforme. Intermediate grades are indefinite and tend to vary with the sections examined. The grade 1 gliomas occur somewhat infrequently in the *cerebrum*. They are usually of firmer consistency than normal brain. They sometimes appear circumscribed although tumour cells penetrate beyond the apparent plane of cleavage. These gliomas may give rise to cysts. The fluid in the cysts is yellow, and contains a large amount of protein. Cysts may arise within tumours or on their outer surface. In the latter case, most of the cyst wall is smooth and free from tumour cells, and removal of the tumour is all that is necessary to prevent reaccumulation of cystic fluid.

*Oligodendrogliomas*, as the name suggests, are thought to be derived from oligodendrocytes (so named because of their paucity of dendritic processes). They are rare tumours (less than 2 per cent. of all intracranial tumours) with a maximum incidence in the fourth and fifth decades. They occur in the cerebral hemispheres (*especially the frontal lobes*) more often than in any other part of the brain. They are composed of closely packed cells with spherical darkly staining nuclei, each surrounded by a ring ("halo") of feebly staining cytoplasm. Calcification is common and can usually be demonstrated radiologically. The importance of this tumour is that *there is a good prospect of long survival after removal*.

**Meningiomas** (Syn. Dural Endotheliomas). About 15 per cent. of all primary intracranial tumours are meningiomas (Cushing), and they are found more often in the frontal compartment than elsewhere in the cranium. They are almost always attached to the dura mater, but they are *enveloped* by the brain from which some of their blood supply may be derived. The majority of meningiomas are benign, but recur if their dural attachment is not completely excised. Although attached to the dura mater, meningiomas are thought to arise from arachnoid cells. The

## TUMOURS OF THE TEMPORAL REGION

**The Temporal Lobe Syndrome.** Rapidly expanding tumours in the temporal fossa frequently cause acute symptoms because of their strategic position in relation to the brain stem. Thus consciousness is often impaired or lost and localising evidence is therefore not obtained.

**Field Defects.** The optic radiation sweeps widely forward in the upper posterior part of the temporal lobe before proceeding backward to the occipital cortex. Thus temporal tumours invade the optic radiation from below, and because light rays from above strike the *lower* part of the retina, homonymous *upper* quadrantic field defects are produced. This is in contrast with parietal tumours which invade the optic radiation from above and cause homonymous *lower* quadrantic field defects. A tumour arising in either situation may eventually cut across the whole of the radiation and produce a complete homonymous hemianopia. Involvement of the visual fields suggests the presence of a destructive tumour within the substance of the brain rather than a tumour indenting the lobe from the outside.

**Personality Changes.** The temporal cortex is concerned with the reception, directly or indirectly, of association-fibre systems of other areas of the cerebral cortex, and therefore receives the resultants of cortical activity as a whole (Le Gros Clark). Memory, the pattern of adult behaviour, and intelligence have their "centre" near the posterior half of the boundary between the parietal and temporal lobes of the dominant hemisphere. It is surprising, therefore, that personality changes are not found more often with temporal lobe tumours. When they do occur they take the form of *anxiety*, *irritability* and occasionally *aggressiveness*. This is in marked contrast with the *apathy* produced by frontal lesions. Temporal tumours are one of the causes of temporal lobe epilepsy which is discussed in detail in Chap. 57.

**Nominal dysphasia** occurs with lesions of the superior temporal convolution of the dominant hemisphere. Tumours which spread upwards and backwards from the dominant temporal lobe eventually cause global (i.e. total) aphasia by destroying the speech centres in the parietal lobe.

**Temporal Lobe Herniation.** The growth and associated cedema of temporal lobe tumours frequently cause herniation of the hippocampal gyrus between the edge of the tentorium and the brain stem (Fig. 371). It is this mechanism which is responsible for the rapid deterioration in the condition of a large proportion of patients with temporal gliomas. The herniation causes lowering of the level of consciousness, dilatation of the homolateral pupil (due to pressure of the herniating hippocampal gyrus on the oculomotor nerve), peripheral vascular failure as seen by lividity of the skin, and pyramidal signs which may be contralateral, bilateral or homolateral. Pyramidal signs on the side of the lesion are caused by the

and backwards *elevating the optic chiasm*. Large tumours of this kind envelop the internal carotid arteries and their branches. The syndrome is characterised by bilateral diminishing visual acuity, optic atrophy, and *bitemporal hemianopia*. There is, however, no ballooning of the pituitary fossa. (Note.—The other causes of bitemporal hemianopia are : Pituitary tumours, suprapituitary cysts, rarely aneurysms of the internal carotid artery and trauma.) Plain radiography may show hyperostosis of the tuberculum sellæ and erosion of the posterior clinoid processes. Ventriculography shows *deformity of the anterior end of the third ventricle*. Bilateral carotid arteriography should be done to show the relationship of the arteries to these tumours. A bifrontal approach is indicated for suprasellar meningiomas. Any part of the capsule adherent to the internal carotid arteries and its branches, or the optic nerves and chiasm, is left undisturbed.

**Secondary Tumours.** Intracranial metastasis may occur from primary malignant tumours anywhere in the body, although carcinoma of the bronchus is the tumour most often responsible for intracranial metastasis. About one-third of secondary tumours of the brain are solitary, they are therefore removed if accessible and if the prognosis of the primary tumour is reasonably good.

**Intracerebral Hæmatomas** (see Chap. 56).

**Frontal Extensions of Pituitary Tumours.** *Chromophobe adenomas* of the pituitary gland occasionally grow into the frontal region and produce the frontal syndrome. This may happen even without compression of the optic chiasm.

**Epidermoid Tumours** (Syn. Pearly Tumours of Cruveilhier, or Cholesteatomas). These rare tumours sometimes occur in the frontal region (other sites are the cauda equina, the skull and the posterior fossa). There is a capsule of epidermal tissue, without dermal appendages, enclosing a mass of epithelial debris and cholesterol crystals. In ventriculograms the air in the deformed ventricle has a characteristic mottled appearance. All debris and crystals must be removed at operation otherwise a fatal aseptic meningitis may occur.

**Colloid cysts** are somewhat rare. They arise from paraphysial remnants, and are found in the third ventricle obstructing the foramen of Monro. They are formed of a capsule of *epithelial* tissue and contain viscid colloid material. Despite their intimate anatomical relationship with the hypothalamus they only occasionally disturb its function. They cause intermittent hydrocephalus. Thus there are episodes of headache and vomiting, sometimes associated with papilloedema. A history of recurrent weakness of the legs (presumably due to pressure on the cerebral peduncles) is sometimes obtained. Colloid cysts have a meagre attachment to the roof of the third ventricle. They are removed through an incision made into the anterior horn of the dilated lateral ventricle, on the non-dominant side.

There is a characteristic upward curve of the middle cerebral artery and its branches (Fig. 365a). In the absence of localising or lateralising signs, *ventriculography* is usually carried out before angiography. Anteroposterior views show displacement of the ventricles towards the opposite side with angulation of the third ventricle on the septum lucidum (Fig. 372). Marked displacement of the third ventricle towards the opposite side indicates temporal lobe herniation and therefore brain stem compression. Lateral views taken after manœuvres to fill the temporal horns may demonstrate absence of filling, deformity or displacement of the temporal horn.

### **PATHOLOGY OF TUMOURS OF THE TEMPORAL REGION**

**Gliomas.** The commonest glioma in this situation is the glioblastoma multiforme (the characteristics of the various gliomas are described under frontal tumours). Many neurosurgeons do not operate on malignant tumours when the diagnosis is proven by biopsy, for recurrence occurs so quickly that the reprieve is not considered worth while. However, it may be worth rescuing patients from coma by removing such a tumour. Patients may then be able to put their affairs in order. Severe headache is possibly also an indication for the same procedure. The classical subtemporal decompression (removal of the lateral bony wall of the temporal fossa under the temporalis muscle, and opening the dura mater) is of little or no value and is therefore very rarely carried out nowadays.

**Meningiomas.** *Deep (medial) sphenoid ridge meningiomas* produce a characteristic syndrome. Patients complain of unilateral loss of vision and sometimes unilateral exophthalmos. There is homolateral optic atrophy, diminished visual acuity, and, in the early stages, a central scotoma. There may be papilloedema on the other side (Foster-Kennedy syndrome: optic atrophy with central scotoma on one side and papilloedema on the other). When the optic tract is involved there is an homonymous hemianopia. Ophthalmoplegia (paralysis of the eye muscles) is present in the majority of cases, and results from occlusion of the orbital fissure. The ophthalmic division of the trigeminal nerve is affected in some cases; patients then complain of "pins and needles" over the forehead where there is diminished or absent sensation; the corneal reflex is diminished or absent. Temporal lobe epilepsy (Chap. 57) may occur, and is sometimes the presenting symptom. Plain radiography may show hyperostosis or erosion of the inner end of the lesser wing of the sphenoid and the homolateral anterior clinoid process. These deeply placed tumours present a difficult technical problem, for they tend to envelop the internal carotid artery and to adhere to the optic nerve. They are approached along the lesser wing of the sphenoid through a fronto-temporal flap.

*Middle sphenoid ridge meningiomas* usually grow to large dimensions before revealing themselves. Manifestations of raised intracranial

opposite cerebral peduncle being forced against the edge of the tentorium (Kernohan-Woltman phenomenon).

*Neighbourhood Effects.* Expansion of temporal tumours upwards often causes slight facial weakness on the opposite side due to involvement of

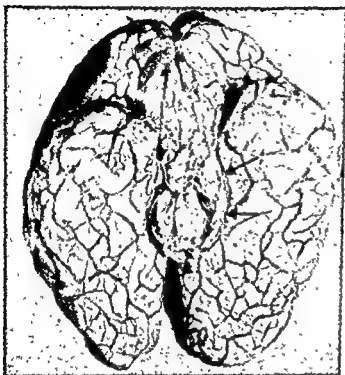


FIG. 371. Temporal lobe herniation. The groove made on the under surface of the left temporal lobe (reader's right) is indicated by arrows. The brain stem was compressed by the hippocampal gyrus being wedged between it and the edge of the tentorium. Note also the enlargement of the left temporal lobe caused by a glioblastoma multiforme.

the face area of the motor cortex. Expansion upwards and inwards causes contralateral hemiparesis by interference with the homolateral cerebral peduncle. Expansion upwards and backwards on the dominant side causes aphasia.

**Differential Diagnosis.** When temporal tumours cause no visual field defect they are likely to be confused with tumours in other relatively silent regions, e.g. frontal and posterior fossa tumours. Abscess of the temporal lobe usually causes no difficulty because there is an associated middle-ear infection. The sudden onset of symptoms caused by some temporal lobe tumours may lead to confusion with vascular disease.

**Investigations.** *Angiography* is of great value in the diagnosis of space-occupying lesions of the temporal fossa.

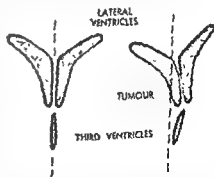


FIG. 372. Drawing of a ventriculogram (anteroposterior view) showing on the right the characteristic angulation of third ventricle and septum lucidum. On the left is shown the normal appearance.

Those on the dominant side may cause memory defects or nominal dysphasia. Radiography may reveal hyperostosis, or erosion of bone. Upward angulation of the middle cerebral artery is seen in angiograms with meningiomas arising along the sphenoid ridge. There may also be increased vascularity of the skull.

*Meningiomas en plaque* are tumours which lie like a carpet over the dura mater, and are characterised by marked increase in thickness of the adjacent bone and lack of neurological effects. The tumour most often grows in relation to the sphenoid bone and runs a chronic course. The presenting symptom is unilateral exophthalmos which is produced by thickening of the bony walls of the orbit. These tumours are inoperable but exploration may be considered worth while, for the same clinical picture can arise from the globular type of meningiomas of the sphenoid described above.

*Sylvian Fissure Meningiomas.* These tumours are attached to the dura mater overlying the posterior ramus of the Sylvian fissure, and penetrate between the frontal and temporal lobes. Localising evidence may be found, and includes personality changes, contralateral facial paresis, Jacksonian attacks beginning in the face, and nominal dysphasia with tumours on the dominant side.

Metastatic tumours, epidermoids and extrasellar extensions of chromophobe pituitary tumours may occur in the middle fossa. Their characteristics have been discussed under Tumours of the Frontal Region.

*Intracerebral Hæmatomas* (Chap. 56).

### TUMOURS OF THE PARIETO-OCCIPITAL REGION

**The Parieto-occipital Syndrome.** Bizarre subjective sensory phenomena, referred to the opposite side of the body, may be produced by lesions of the sensory area of the cerebral cortex. Patients sometimes complain of formication (sensation of ants creeping on the skin) on one side of the body. These strange sensory phenomena are periodic and are probably epileptic. Indeed they may form the aura of more fully developed fits. Invasion of the optic radiation (as also with tumours of the temporal lobe) may produce visual hallucinations. Lesions which affect the visual cortex cause unorganised visual sensations (flashes of light). It is easy to understand why patients with parietal lobe tumours may be referred to a psychiatrist by mistake. When tumours arise in the dominant parietal lobe, speech is affected. Destruction of the angular gyrus produces the syndrome of alexia (inability to read) and homonymous hemianopia. A destructive lesion further forward, affecting the supramarginal gyrus, causes global (i.e. total) aphasia. A tumour on the non-dominant side may cause a defect of body image so that the patient becomes unaware of the opposite side of the body (*corporeal agnosia*).

On examination there may be disturbances of sensation (diminished or absent sensation of light touch, inability to appreciate small differences

pressure may be associated with personality changes produced by involvement of the frontal and temporal lobes. Sometimes the presenting symptom is unilateral exophthalmos (Fig. 373). Plain radiography may



FIG. 373. Unilateral exophthalmos in a patient with a small middle ridge meningioma.



FIG. 374. Radiograph showing thickening of the lateral wall of the orbit produced by the tumour referred to in Fig. 373.

show hyperostosis or erosion of the lesser wing of the sphenoid. Occasionally the hyperostosis is widespread, involving much of the orbit and middle fossa (Fig. 374).

*Lateral sphenoid ridge meningiomas* produce few or no localising signs.

sudden approach of an object towards the eyes causes blinking. This *blink reflex* may be absent when objects approach the eyes from the side opposite to a parietal lesion. A tumour extending anteriorly involves pyramidal fibres causing contralateral paresis. Because of the inclination of the motor cortex the arm and leg are more likely to be affected than the face.

**Differential Diagnosis. Mental Disease.**—When evidence of raised intracranial pressure is lacking, the strange subjective sensory disturbances occurring with some parietal tumours may lead to the mistaken diagnosis of mental disease. **Cerebral Haemorrhage.**—A tumour producing an acute onset of symptoms may be mistaken for a vascular lesion. The presence of papilloedema would favour the diagnosis of tumour.

**Investigations. Angiography.**—Parietal tumours tend to cause downward displacement of the Sylvian vessels (terminal branches of the middle cerebral artery). There may also be a vascular pattern. The angiogram, however, may be entirely negative, as some of these tumours lie towards the termination of the arteries. **Ventriculography.**—Anteriorly placed tumours show deformity of the body of the lateral ventricle, whereas posteriorly placed tumours show deformity of the occipital horn. The ventricles may be displaced to the opposite side. Cysts can be visualised by injection of air or diodone (Fig. 375).

#### PATHOLOGY OF PARIETO-OCCIPITAL TUMOURS

**Gliomas.** The commonest is the glioblastoma multiforme. As elsewhere it may be cystic or solid. When the diagnosis is established with certainty by biopsy, there is no indication for removing these tumours even on the non-dominant side.

**Meningiomas. Parasagittal meningiomas** are common in this region, especially anteriorly where they involve the motor as well as the sensory cortex. Because the motor area for the lower extremity is on the *medial* surface of the hemisphere, the leg tends to be involved before the arm and face. Bilateral parasagittal meningiomas cause weakness of both lower extremities. Plain radiography may show: (a) Increased vascular markings; (b) hyperostosis; (c) erosion of bone; (d) calcification of the tumour. Often there is no radiological evidence to indicate a meningioma.

At operation, tumours arising from the falx are likely to be concealed by the brain, whereas those arising from the superior longitudinal sinus are likely to be encountered immediately after reflection of the dura mater. Some meningiomas in this region arise from the dura over the surface of the parietal lobe without involving the superior longitudinal sinus. Ablation of all but the very smallest meningiomas in this situation should begin with piecemeal removal of the interior of the tumours. The capsule then collapses away from the brain and thus damage to the motor and sensory cortex is avoided. Great care must be taken to prevent injury to veins draining into the superior longitudinal sinus. If a tumour



of weight, loss of perception of the size and shape of objects, i.e. *astereognosis* or *tactile agnosia*). The reception zone for stereognosis is widespread in the parietal lobes. *Visual agnosia* (inability to recognise objects when vision is intact) is sometimes found with posterior parietal lesions on the dominant side. *Apraxia* (inability to perform purposeful acts when the pyramidal pathways are intact) is sometimes observed with parietal lesions.

A tumour extending downwards or backwards interrupts the optic

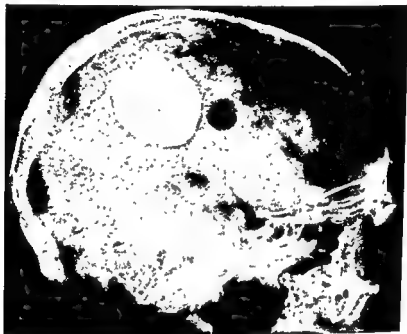


FIG. 373. Cystogram, obtained by injecting diodone into a right parietal cyst. Biopsy adjacent to the cyst revealed a glioblastoma multiforme.

radiation, affecting the uppermost fibres first. Therefore, because light rays from the lower half of the visual field go to the upper half of the retina, homonymous *lower* quadrantic field defects are produced. This contrasts with temporal tumours which affect the optic radiation from *below* and cause homonymous upper quadrantic defects. When both the upper and lower parts of the optic radiation are affected by tumours, there is hemianopia with sparing of macular vision, although complete division of the optic radiation, as in removal of the occipital lobe, causes complete homonymous hemianopia, including transection of the macular part of the field. *Visual attention* (Chap. 52) is sometimes lost with lesions of the parieto-occipital lobe, and attention to other forms of sensory stimuli may also be defective. Thus light touch may be felt equally well on both sides of the body when they are tested separately, whereas simultaneous stimulation of both sides together results in *extinction* of appreciation of the stimulus on the side opposite to the lesion. In a normal person, the

pyramidal signs may occasionally be produced by supratentorial space-occupying lesions (Kernohan-Woltman phenomenon), but it may not be appreciated that laterally placed expanding lesions in the posterior fossa may also cause homolateral pyramidal signs. The effect is produced in both instances by the same mechanism: The cerebral peduncle of the opposite side is forced against the edge of the tentorium, and thus pressure occurs on pyramidal fibres which later cross below in the medulla oblongata to the side of the lesion.

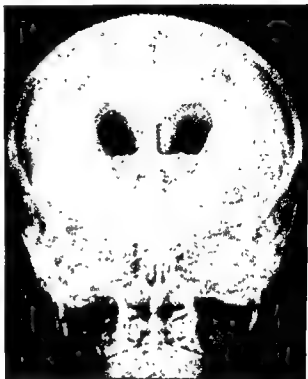


FIG. 376. Ventriculography showing dilatation of the lateral ventricles and the third ventricle, produced by a tumour in the posterior fossa.

*Slowly growing intrinsic cerebellar tumours not infrequently give little or even no evidence of involvement of the cerebellum, brain stem or cranial nerves, but present with manifestations of increased intracranial pressure alone. On the other hand, extrinsic tumours (e.g. acoustic tumours and meningiomas) commonly present with cranial nerve signs and later cause hydrocephalus. Rapidly expanding lesions (e.g. cystic tumours and cerebellar hæmorrhage) cause acute hydrocephalus and medullary compression.*

**Differential Diagnosis.** Slowly growing cerebellar tumours which have a tendency to cause little or no localising signs may be confused with "silent" tumours arising elsewhere (e.g. frontal tumours, temporal tumours, and colloid cysts of the third ventricle). Frontal tumours occasionally produce contralateral "cerebellar" signs.

**Investigations.** *Ventriculography* usually reveals dilatation of the lateral ventricles, the third ventricle and the aqueduct (Fig. 376). Dis-

is attached to the sinus, a thin layer of tumour tissue is left behind, for the sinus must not be damaged in this situation.

*Perilorcular meningiomas* are rare. They may arise from any of the four quadrants around the venous confluence. Some cause homonymous visual field defects whereas others produce cerebellar signs. These tumours have a tendency to recur. The approach is by means of Krause's occipital flap based on the occipital muscles.

**Pineal Tumours.** These tumours obstruct the aqueduct of Sylvius and thus cause hydrocephalus. Pressure on the midbrain causes loss of upward conjugate movement of the eyes; the pupils may be dilated and inactive to light with preservation of reaction to accommodation. Sometimes there is partial bilateral deafness caused by compression of the medial geniculate bodies. Cerebellar signs may arise from involvement of the superior cerebellar peduncles. Pubertas præcox sometimes occurs in the male if the tumour is active before puberty, although the normal pineal gland has not been shown to have any endocrine function. Radiography may show calcification in the tumour, and ventriculography reveals hydrocephalus and deformity of the posterior end of the third ventricle. These tumours are approached through a parietal flap on the non-dominant side, and division of the splenium of the corpus callosum. Alternatively radiotherapy may be employed.

#### TUMOURS OF THE POSTERIOR FOSSA

Expanding lesions in the posterior fossa produce their effects in one or more of the following ways: (a) *By obstructing the fourth ventricle they cause hydrocephalus, and thus give rise to headache, vomiting and papilloedema;* (b) *by compression or destruction of the vermis they may cause trunk ataxia;* (c) *by compression or destruction of one of the lateral lobes of the cerebellum they may cause nystagmus, the amplitude of which is greater when the eyes look towards the side of the lesion than when they look towards the opposite side; also on the same side as the lesion there may be hypotonia, inco-ordination (demonstrated by the finger-nose test), intention tremor of the upper limb, dysidiadochokinesia (inability to perform quickly repeated movements), lateral and downward deviation of the arm when both arms are held outstretched, and past-pointing (the patient points with the index finger of the side being tested to the palm of the observer's hand, and, closing the eyes, moves the limb in a horizontal or vertical plane attempting to bring the finger back to the point of departure; in lesions of the cerebellar lobes the limb on the affected side comes to rest lateral to this position);* (d) *by compression of the medulla oblongata, space-occupying lesions of the posterior fossa may cause vertigo, dysarthria, dysphagia, repeated vomiting, bradycardia, slowing of respiration and eventually Cheyne-Stokes respiration;* (e) *compression of the cranial nerves of the posterior fossa;* and (f) *pressure on the pyramidal tracts, on either or both sides. It is well known that homolateral*

pyramidal signs may occasionally be produced by supratentorial space-occupying lesions (Kernohan-Woltman phenomenon), but it may not be appreciated that laterally placed expanding lesions in the posterior fossa may also cause homolateral pyramidal signs. The effect is produced in both instances by the same mechanism: The cerebral peduncle of the opposite side is forced against the edge of the tentorium, and thus pressure occurs on pyramidal fibres which later cross below in the medulla oblongata to the side of the lesion.



FIG. 376. Ventriculography showing dilatation of the lateral ventricles and the third ventricle, produced by a tumour in the posterior fossa.

*Slowly growing intrinsic cerebellar tumours not infrequently give little or even no evidence of involvement of the cerebellum, brain stem or cranial nerves, but present with manifestations of increased intracranial pressure alone. On the other hand, extrinsic tumours (e.g. acoustic tumours and meningiomas) commonly present with cranial nerve signs and later cause hydrocephalus. Rapidly expanding lesions (e.g. cystic tumours and cerebellar hæmorrhage) cause acute hydrocephalus and medullary compression.*

**Differential Diagnosis.** *Slowly growing cerebellar tumours which have a tendency to cause little or no localising signs may be confused with "silent" tumours arising elsewhere (e.g. frontal tumours, temporal tumours, and colloid cysts of the third ventricle). Frontal tumours occasionally produce contralateral "cerebellar" signs.*

**Investigations.** *Ventriculography usually reveals dilatation of the lateral ventricles, the third ventricle and the aqueduct (Fig. 376). Dis-*

placement, deformity and dilatation of the fourth ventricle may also be demonstrated. Tumours in the posterior fossa sometimes cause upward displacement of the third ventricle, or upward curving of the posterior horn of the lateral ventricle on the side of a laterally placed tumour. Marked indentation of the occipital horn occurs with dumb-bell meningiomas arising from the tentorium and growing supra- and infratentorially. Positive contrast ventriculography with Myodil (1 to 2 ml.) injected through the anterior horn of one of the lateral ventricles gives



FIG. 377. Positive contrast ventriculography (with Myodil 3 ml.) showing a filling defect of the right lateral recess of the fourth ventricle produced by an acoustic tumour (Towne's view). Above the fourth ventricle a small quantity of Myodil is seen in the third ventricle.

more reliable information regarding the aqueduct and fourth ventricle than air ventriculography (Figs. 368 and 377). However strong the clinical evidence of a posterior fossa tumour may be, ventriculography should always be done (see differential diagnosis), except perhaps when there is indisputable evidence of an acoustic tumour, which produces a characteristic syndrome. *Vertebral angiography* is more difficult and more dangerous than carotid angiography. Therefore it is not employed if the necessary information can be obtained otherwise. Aneurysms and vascular tumours are outlined in angiograms. *Electroencephalography*.—Electrical potentials cannot be recorded from the cerebellum.

#### PATHOLOGY OF POSTERIOR FOSSA TUMOURS

**Astrocytomas** are the commonest intrinsic cerebellar tumours. The maximum incidence is during the first two decades. *In the cerebellum,*

most astrocytomas are cystic and benign (grade I). Frequently there is a large cyst with a small mural tumour. The cystic fluid is yellow and contains a large amount of protein. Removal of the mural nodule is frequently all that is required.

**Medulloblastomas** occur somewhat less frequently than astrocytomas in patients of the same age group. They are very malignant tumours for which there is no curative treatment. Although they are very radio-sensitive, recurrence always occurs (in those cases confirmed histologically). These tumours are composed of small densely packed cells containing little cytoplasm. There are mitotic figures, occasional multinucleated neoplastic giant cells, and sometimes rosette formations of cells. There is a tendency for meningeal metastasis to occur. Medulloblastomas do not occur above the tentorium.

**Hæmangioblastomas** (Syn. Angioreticulomas). These benign tumours occur in the cerebellum; they are rare elsewhere in the nervous system. They are more often cystic than solid. The cysts tend to be large and contain mural tumours like those in cystic astrocytomas of the cerebellum. Unlike the latter tumours, however, hæmangioblastomas are found most often in adults, and they show themselves earlier in women than in men. Thus in women the maximum incidence occurs between twenty and forty years, whereas in men it is between forty and sixty years. Sometimes these tumours are familial; they then tend to be multiple. Therefore vertebral angiography should be done for suspected hæmangioblastomas when there is a family history of these tumours, otherwise multiple tumours may be overlooked at operation. The association with hæmangioblastomas elsewhere and cysts in the liver, pancreas or kidney, is known as Lindau's syndrome. Angioma of the retina (von Hippel's disease) may be one of the lesions forming part of Lindau's syndrome.

**Ependymomas** occur in the posterior fossa as well as elsewhere in the central nervous system. They are often, although not always, attached to the floor of the fourth ventricle. The base of those attached to the floor of the fourth ventricle is not removed. The cells of the tumour are formed into rosettes and contain blepharoplasts (darkly staining bodies close to the nuclei).

**Acoustic Nerve Tumours** (Syn. Acoustic Neurinomas, Schwannomas, Perineural Fibroblastomas or Neurilemmomas). These tumours are thought to arise from the endoneurium of the acoustic nerve within the internal auditory meatus. They grow in the subdural space, and as they expand come into relationship with the trigeminal nerve, cerebellum, pons, and eventually also with the ninth, tenth, eleventh and twelfth cranial nerves. They are solid benign tumours usually surrounded by a cyst formed by the arachnoid. Bilateral acoustic tumours are sometimes found in the "central" type of von Recklinghausen's neurofibromatosis. Acoustic tumours are composed of interlacing bundles of spindle cells

containing elongated nuclei. The cells tend to be arranged in rows ("palisading") and some form whorls around blood vessels. Reticulum fibrils are conspicuous between cells. Nerve fibres are rarely seen.

These tumours affect both sexes equally, and symptoms begin in middle age and have usually been present for two to three years before the neurosurgeon is consulted. The first symptoms are derived from compression of the eighth cranial nerve. Gradually progressive *deafness* in one ear, frequently accompanied by *tinnitus* (noises in the ear) and *dizziness* (severe vertigo is rare), is the first manifestation. Involvement of the vestibular division of the eighth cranial nerve is demonstrated by the caloric test (see Chap. 52).

*Paræsthesiæ* (numbness, tingling or burning sensation) in the distribution of the fifth cranial nerve are common, but trigeminal pain is rare. Absence of the corneal reflex may be the only sign of involvement of this nerve. There may be aching pain in the occipital muscles and tenderness behind the homolateral mastoid process. Later there are signs of compression of the cerebellum and brain stem. In some cases compression of the latter produces pyramidal signs which may be found on either or both sides. Homolateral pyramidal signs are caused by indentation of the opposite cerebral peduncle by the tentorium whereas contralateral pyramidal signs are caused by direct pressure of the tumour on the pons. Later there are signs of increased intracranial pressure (headache, vomiting and papilloedema, abducens nerve paralysis). Extension of the tumour to the tenth and eleventh cranial nerves causes unilateral palatal, pharyngeal and laryngeal paralysis and also partial paralysis of the sternomastoid and trapezius muscles.

*Radiography* often shows enlargement of the internal auditory meatus and sometimes considerable erosion of the petrous bone. In the presence of these signs no other investigations are necessary. When the syndrome is atypical, positive contrast ventriculography (Myodil 1 to 2 ml.) to show the fourth ventricle and the aqueduct is indicated (Fig. 377).

Removal of acoustic tumours is carried out through a unilateral approach with the patient in the sitting position. Operation still has a considerable mortality (up to 25 per cent.) because these tumours are usually referred to the neurosurgeon when there is marked indentation of the brain stem and advanced hydrocephalus (Fig. 378). Decompression alone is valueless, for these tumours are firmly attached to the petrous bone and therefore the pressure on the brain stem is not relieved. The facial nerve is not seen during operation, and facial palsy usually results. It is treated by facial-hypoglossal or facial-accessory anastomosis (Chap. 52).

**Meningiomas** in the posterior fossa may be attached to any part of the dura mater, but usually arise somewhere along the transverse or sigmoid sinuses. Sometimes they grow in the cerebello-pontine angle and mimic acoustic tumours. Meningiomas of Meckel's cave are described in

Chap. 52. *Dumb-bell meningiomas* are rare tumours which grow upward to indent the parieto-occipital lobe and downward into the posterior fossa. In the removal of meningiomas of the posterior fossa, the transverse sinus on one side may be removed, as there is usually an anastomotic channel between the two transverse sinuses. The presence of this connection may, however, be verified preoperatively by venography (the superior longitudinal sinus is injected through a burr-hole with 20 ml. of 42.5 per cent. diodone solution) and serial X-rays are taken.

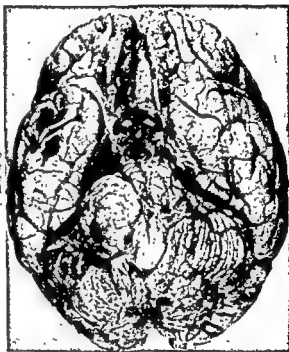


FIG. 378. The remains of a large right-sided (reader's left) acoustic tumour. There is considerable indentation of the pons and medulla oblongata by the tumour.

*Tuberculomas* are rare in countries with a high standard of living. They behave as tumours. Contrary to former teaching, they are rarely calcified. Nowadays removal is safe if streptomycin is given systemically and intrathecally for a few days before operation, and for about six weeks afterwards.

**Epidermoid Tumours** (Syn. Cholesteatomas, Pearly Tumours of Cruveilhier). These are congenital tumours arising from the meninges and consisting of a capsule of *epidermal* tissue enclosing a mass of epithelial debris and cholesterol crystals. They are not to be confused with the inflammatory type of "cholesteatoma" caused by chronic infection of the middle ear and mastoid. The whole of the contents of the "cyst" and as much of the capsule as possible must be removed, for if some cholesterol remains it causes an aseptic meningitis which may be fatal.



Ménière's disease may be confused with tumours in the cerebello-pontine angle. This disease is described in Chap. 52.

## PITUITARY TUMOURS

by

A. A. G. LEWIS and LESLIE C. OLIVER

**The Adenohypophysis.** The cells of the anterior pituitary lobe (adenohypophysis) are classified into three types according to the staining reactions of their cytoplasm: The eosinophil cells stain with acid dyes and the basophil cells with basic dyes. The chromophobe cells stain poorly or not at all; they probably do not produce hormones, but may be the parents of the other cells. The eosinophil cells produce the growth hormone, and the basophil cells produce the following: (1) The thyrotropic hormone (TSH); (2) the adrenocorticotrophic hormone (ACTH); (3) a hormone stimulating the ovarian follicles in the female, or the seminiferous tubules in the male (FSH); (4) the luteinising or interstitial-cell stimulating hormone (LH) or (ICSH); (5) the lactogenic hormone (prolactin). It is thought that the hypothalamus controls the production of these hormones by humoral influences which pass down the portal system of blood vessels surrounding the pituitary stalk.

**The neurohypophysis** (the median eminence of the tuber cinereum, the infundibular stem and the infundibular process or posterior pituitary lobe) produces the antidiuretic hormone (vasopressin or ADH), in response to impulses passing from the supraoptic nucleus of the hypothalamus down the supraoptico-hypophysial tracts. Section of the pituitary stalk, therefore, leads to irrecoverable loss of function of the posterior lobe, whereas that of the anterior lobe may recover when the portal system of blood vessels reforms.

When a pituitary tropic hormone stimulates an endocrine gland to produce another hormone this in turn usually suppresses the production of the tropic hormone itself. In this way a good deal of autoregulation occurs in the endocrine system. An adrenal tumour may produce sufficient hormone to suppress the production of ACTH by the pituitary, so that atrophy of the opposite adrenal may occur—a possibility which must be borne in mind when removing an adrenal tumour. Continuous cortisone therapy so depresses the production of ACTH that a dangerous phase of adrenal deficiency follows its sudden cessation.

The action of the pituitary hormones may be summarised as follows: (1) *The growth hormone* stimulates the growth of endochondral and membranous bone, although other factors are necessary for this as well (e.g. normal thyroid function and normal nutrition; the sex hormones also stimulate growth, but eventually end it by causing fusion of the epiphyses of the long bones). Whether or not the growth hormone is the

anterior pituitary factor which inhibits the conversion of glucose into glucose-6-phosphate under the influence of hexokinase (an inhibition opposed by insulin), it is probable that it does act to maintain the blood sugar level by retarding the rate at which the cells can take up glucose under the action of insulin. In the absence of this hormone, therefore, prolonged hypoglycæmia may occur on fasting. *TSH* stimulates the entry of thyroid hormone into the blood stream from the thyroid follicles; the thyroid gland in turn responds by concentrating iodine from the plasma and forming more hormone. *ACTH* stimulates the formation of 11-oxysteroids by the adrenal cortex. These hormones (of which the chief representative in man is *cortisol* or *compound F*.) maintain blood sugar and liver glycogen by increasing the formation of glucose from amino-acids (gluconeogenesis). They also cause lymphopenia and eosinopenia. Their action is essential to life. The adrenal cortex also produces *aldosterone*, which stimulates the kidney to retain sodium and excrete potassium, thus tending to preserve extracellular fluid volume and maintain cardiac output and blood pressure (cortisol exerts a similar but less constant action). The adrenal gland also produces *androgens*, possibly under the influence of LH. In the female, these are responsible for the growth of body hair at puberty and for the excretion of 17-ketosteroids in the urine (in the male these are formed also by the testis). Rhythmic production of *gonadotropins* (FSH and LH) are responsible for the normal female menstrual cycle. *Prolactin* stimulates the formation of milk in a breast already prepared by other factors, including the gonadotropins. Milk expulsion is stimulated by the oxytocic factor of the posterior pituitary lobe. *ADH* stimulates the renal tubules to absorb water from the glomerular filtrate with the formation of urine more concentrated than plasma. (S.G. rising to an upper limit of 1,040. In the absence of ADH, urinary S.G. may fall almost to 1,000.) Its formation is stimulated by a rise in the concentration of extracellular ions (particularly sodium), by a fall in pressure in the left auricle and also by emotional factors originating in the higher centres. The urinary solutes can, therefore, be excreted in a volume of water which can be varied greatly according to the need of the body to excrete or to retain water.

Deficiency of production of pituitary hormones may be shown by :  
 (1) *Failure of Growth*.—Pituitary dwarfism may be the main manifestation of hypopituitarism in childhood; body proportions are normal (i.e. the span of the arms is equal to the height). Sexual maturation does not occur, so that a very slow rate of growth may continue for many years. Though usually the result of a tumour such as a craniopharyngioma or, rarely, a chromophobe adenoma, the condition sometimes results from isolated deficiency of the eosinophil cells.  
 (2) *A poor response to stress and infection*, with a tendency to hypoglycæmia on fasting and to low blood pressure.  
 (3) *Failure of thyroid function*, with myxœdema. The B.M.R. is low, below minus 20, and the blood cholesterol raised above 300 mg/100

ml. The patient is cold and apathetic, with a dry waxy skin, constipation and bradycardia. (4) *Sexual Failure*.—In the female there is amenorrhœa, genital atrophy and sterility, in the male loss of libido and impotence. In both sexes the growth of the body hair, and in the male the size of the penis, give some indication of androgen production. (5) *Absence of Lactation*. (6) *The presence of diabetes insipidus* (passing of excessive quantities of urine of low specific gravity, and constant thirst). In the absence of ADH, provided that some function of the *adenohypophysis* persists, the clinical condition of diabetes insipidus develops.

In addition to the above evidence, the following tests may indicate pituitary failure: (1) *The Insulin Tolerance Test*.—In the normal subject intravenous injection of 0.1 unit of insulin per kilogramme of body weight, produces a fall in blood sugar greater than 50 per cent. of the resting value; the blood sugar returns to its former level in less than two hours. In pituitary deficiency the fall is greater, and therefore not more than one-third of the above dose of insulin should be injected in a suspected case; there is also "hypoglycæmia unresponsiveness," i.e. the blood sugar level does not return to normal. (2) *Estimation of the Twenty-four-hourly excretion of 17-Ketosteroids in the Urine*.—The normal range is 10 to 20 mg. in males, 5 to 15 mg. in females. (3) *Estimation of corticosteroids in the blood or urine* is more difficult, but gives a more precise indication of adrenal activity. Adrenal deficiency can be distinguished from that of the pituitary by the failure in the former condition of the urinary excretion of 17-ketosteroids or of 11-oxysteroids to rise after the intravenous infusion of 25 units of ACTH given over eight hours, or the injection of 40 units of a long-acting preparation of ACTH on each of two successive days.

**Chromophobe Adenomas.** These are the pituitary tumours most frequently needing operative treatment. They arise from the apparently functionless chromophobe cells of the anterior pituitary lobe. Cystic degeneration occurs more often than in acidophil adenomas. As these tumours grow they replace functioning acidophil and basophil cells and expand the pituitary fossa. Extension above the pituitary fossa brings them into contact with the optic chiasm and nerves, and the hypothalamus.

Patients are usually middle-aged, and present clinically with disturbance of vision, usually *amblyopia* (dimness of vision). Examination reveals loss of visual acuity and often primary optic atrophy in one or both eyes. The classical and commonest visual field defect is a *bitemporal hemianopia* which first affects the upper quadrants. This apparent discrepancy arises from the fact that although the tumours press on the chiasm from below, the light rays falling on the lower part of the retina come from above. An *homonymous hemianopia* indicates: (a) Pressure on the optic tract; (b) a prefixed optic chiasm\*; or (c) an extension of the tumour into the temporal lobe. If one eye is blind and the other has

\* With a prefixed chiasm there is little or no space between it and the tuberculum sellæ.

a temporal field defect, then originally there was either a bitemporal or an homonymous hemianopia. The history may help to decide which of these two defects existed. A central scotoma in one or both eyes without defects of the peripheral part of the fields usually indicates preflexation of the chiasm. As previously stated, pressure on the optic nerve causes a central scotoma; in preflexation of the chiasm pituitary tumours force the optic nerves against the upper margins of the optic foramina.

Upward and backward growth of pituitary tumours indents the hypothalamus, but because the *central* nervous system is able to adapt itself to *slowly* increasing pressure, little or no hypothalamic dysfunction may be observed. *Polydipsia* (excessive thirst) together with polyuria (excessive passage of urine), i.e. diabetes insipidus, sometimes occurs and may be attributable to pressure on the supraoptico-hypophysial tracts or the neurohypophysis. However, the importance of hypothalamic extensions is that acute disturbances of hypothalamic function are apt to follow operative interference, and the mortality in such cases is high.

Although invasion by malignant pituitary adenomas into the cavernous sinus does occur, symptoms of involvement of the structures of the cavernous sinus may also occur with benign tumours. The oculomotor nerve is affected more often than the abducens and trochlear nerves. Very rarely the internal carotid artery is obstructed and ischæmia of the corresponding hemisphere occurs. Extension of pituitary tumours into the frontal or temporal regions may be silent, or produce localising signs and papilloedema.

Chromophobe adenomas replace secreting cells and thus cause hypopituitarism. There is tiredness, mental inertia, sensitivity to cold, loss of libido, impotence in the male, amenorrhœa in the female, and loss of hair on the body, including the axillæ and pubis. The hair of the head is fine and the skin feels thin and smooth, due to loss of connective tissue. Patients suffering from hypopituitarism are *not* emaciated. Their hypersensitivity to drugs (e.g. opiates and barbiturates) and anæsthetics should be remembered when surgery is contemplated.

**Acidophil adenomas or hyperplasia** of the acidophil cells of the pituitary gland cause the endocrine picture of *acromegaly* (akros = extremity, and megale = large). Some adenomas cause pressure on the optic nerves and chiasm, and may also spread well beyond the limits of the pituitary fossa, and produce *the same neurological disturbances described under chromophobe adenomas*. The cells of acidophil adenomas produce an excess of the growth or somatotrophic hormone (STH) and the allied diabetogenic hormone. Thus *gigantism* occurs if the epiphyses are still active and *acromegaly* if they are united, and diabetes mellitus is common. Sexual desire and potency may be reduced or abolished, presumably by suppression of the basophil cells. When not caused by an expanding tumour, the abnormal endocrine secretion may abate or cease altogether. Operation is indicated when an adenoma causes pressure on the optic

nerves and chiasm. It is followed by radiotherapy. Some improvement in the endocrine state may follow operation but is not sufficient to warrant operation for endocrine manifestations alone.

**Adenocarcinomas** of the anterior pituitary lobe are rare. Their cells contain no granules and produce no endocrine secretion. Either benign or malignant adenomas may affect the structures in the cavernous sinus.

**Basophil adenomas** are small and *very* rarely expand the pituitary fossa to compress the optic pathways. *Basophilism* (Cushing's disease) is described in Chap. 72.

**Pituitary Apoplexy.** Rarely, pituitary adenomas enlarge so rapidly that they outgrow their blood supply, and acute necrosis, with or without hæmorrhage, occurs. The onset of symptoms is sudden with headache, amblyopia, drowsiness, external ocular palsies and sometimes subarachnoid bleeding. Patients may soon die from acute compression of the hypothalamus. Those who survive are in a state of evident or concealed hypopituitarism. Sometimes there are no previous symptoms of a pituitary adenoma, although signs of long-standing hypopituitarism or acromegaly are likely to be found, and radiography shows ballooning of the pituitary fossa. When subarachnoid hæmorrhage occurs confusion with an aneurysm is likely. Operation is undertaken only after steps have been taken to correct the acute hypopituitarism occurring in these cases.

**Craniopharyngiomas** (Syn. *Epidermoids*, *Tumours of the Hypophysial Duct*, *Tumours of Rathke's Pouch*, *Adamantinomas*). These tumours arise in remnants of Rathke's pouch (craniopharyngeal duct)—the ectodermal projection from the developing buccal cavity which joins with the downward projection from the developing nervous system to form the pituitary gland. They are thought to be solid at first, but by the time they cause symptoms they are partly or entirely cystic. The cysts are filled with yellow or dark brown liquid containing cholesterol crystals; some of the cysts are multilocular. Calcification of the cyst wall almost always occurs and can be demonstrated radiologically. The clinical picture depends on the site of origin along the line of possible epithelial rests. A tumour arising in the pituitary fossa below the sellar diaphragm (rare) causes hypopituitarism and pressure on the optic chiasm from below, i.e. the same syndrome as that of a chromophobe adenoma (*vide supra*). A tumour arising above the sellar diaphragm may cause mild *hypothalamic* effects and pressure on the chiasm from *above* (the *bitemporal hemianopia* begins in the lower quadrants). Some tumours are too high to affect the optic pathways. Those arising above the sellar diaphragm indent the floor of the third ventricle earlier than those arising in the pituitary fossa, and are likely to obstruct the foramen of Monro and thus cause hydrocephalus.

Declining vision in early life is the commonest presenting manifestation of craniopharyngiomas, although sometimes symptoms are delayed until adult life. When a craniopharyngioma arises in the pituitary fossa, it

depresses the function of the pituitary gland *during childhood* and thus arrests growth. Not all pituitary dwarfs, however, have tumours.

Pressure of the tumour on the floor of the third ventricle may cause one or more of the following hypothalamic manifestations: Episodes of diabetes insipidus, lack of emotional control, euphoria alternating with depression, somnolence or excessive wakefulness, low blood pressure (more often than a high pressure), hyperchlorhydria, sometimes with peptic ulceration, achlorhydria, and some degree of adiposity. *Fröhlich's syndrome*, which is *now* thought to be *extremely rare*, consists of failing vision with bitemporal hemianopia, hypogonadism and adiposity (*dystrophia adiposogenitalis*), but there are innumerable fat boys with small genitalia who are not suffering from Fröhlich's disease. Chronic pressure on the hypothalamus is usually well tolerated. Therefore the hypothalamic manifestations described above tend to be unobtrusive or absent before operation, whereas after operation they may be acute and sometimes fatal.

**Radiography.** Almost all tumours which arise in the pituitary fossa below the sellar diaphragm cause ballooning of this structure. Its outline appears roughly circular in lateral X-ray views. The dorsum sellæ and posterior clinoid processes become thin and rarefied, and may finally disappear. *Acidophil* adenomas produce signs in the skull associated with acromegaly (enlarged nasal air sinuses and mastoid processes, enlargement of the superciliary ridges, lower jaw and malar bones, and separation of the teeth). Occasionally, pituitary adenomas are entirely or almost entirely extrasellar. There is then no enlargement of the pituitary fossa. Craniopharyngiomas almost always show calcification of all or part of the cyst wall. They may also be outlined by ventriculography. When there is no evidence of raised intracranial pressure, *air-encephalography* should be carried out in patients with pituitary tumours to demonstrate any extrasellar extension. An attempt is made to fill the basal cisterns (*cisternography*) around the pituitary tumour. On those occasions when the intracranial pressure is raised, ventriculography is carried out instead of encephalography. If signs of endocrine dysfunction are minimal or absent, bilateral carotid angiography is done to exclude an internal carotid aneurysm.

**Differential Diagnosis.** The syndrome of failing vision with bitemporal hemianopia or other field defects may be produced by: (a) Pituitary adenomas; (b) craniopharyngiomas; (c) suprasellar meningiomas, which tend to destroy the pituitary fossa without expanding it, although not infrequently the pituitary fossa is unaffected; (d) a rare variety of aneurysm of the internal carotid artery (Fig. 390); (e) gliomas of the optic nerve and chiasm; they produce enlargement of the optic canals without affecting the pituitary fossa; and (f) non-specific arachnoiditis.

**Operations.** Pituitary adenomas are approached through a small frontal flap on the non-dominant side. The bone flap is planned to avoid the frontal air sinuses, and the supraorbital saw-cut is made as close to

the supraorbital ridge as possible. The dura mater may be opened along the lesser wing of the sphenoid ("extradural" method), or a dural flap may be reflected from the convexity of the frontal pole towards the superior longitudinal sinus ("intradural" method). A much better exposure of the pituitary region is obtained through a small free bifrontal bone flap; in this approach, the anterior end of the superior longitudinal sinus is divided between ligatures and both frontal lobes are retracted.

The tumour is usually found bulging upwards anterior to the optic chiasm and between the optic nerves. Aspiration of the tumour is carried out to exclude an aneurysm and to determine whether the tumour is cystic or solid. Small vessels on the surface of the tumour are coagulated with a weak current. A cruciate incision is then made in the capsule and the interior of the tumour evacuated piecemeal by means of a pituitary rongeur or suction. The capsule is gently dissected from the chiasm and optic nerves, but no attempt is made to remove any part of the capsule attached to the hypothalamus (floor of the third ventricle), as fatal hypothalamic disturbances may follow. Patties (small squares of cottonoid) are packed into the cavity for a few minutes to arrest bleeding. Rapid deterioration of vision after operation indicates the presence of an intrasellar clot which should be immediately evacuated.

*Craniopharyngiomas* within the pituitary fossa are removed in the same way as pituitary adenomas, but the treatment of suprasellar craniopharyngiomas demands careful circumspection. When the object is relief of pressure on the optic pathways, this should be accomplished without disturbing the part of the capsule in contact with the hypothalamus. Large cysts should be *slowly* emptied before the part compressing the chiasm and optic nerves is attacked. Obstruction of the foramen of Monro without visual disturbances should be treated by ventriculocisternostomy (Torkildsen's operation—described in Chap. 55). Drainage of *both* lateral ventricles into the cisterna magna is necessary, with two separate catheters.

**Post-operative Complications.** *Hyperthermia* is a common complication of operations in the pituitary region and results from interference with the hypothalamus. It must be treated promptly by cold sponging. This is begun when the temperature rises above 102° F.

Failure of the patient to regain consciousness after operation may be due to the following causes: (a) Damage to the hypothalamus, suggested by associated hyperthermia; there is no effective treatment. (b) Hypersensitivity of these patients to sedative drugs and anaesthetics. Antidotes should be used (e.g. picrotoxin). (c) Cerebral oedema caused by retraction of the frontal lobe. (d) Reactionary hæmorrhage suggested by contralateral hemiplegia and a tense flap. (e) Hypopituitarism (*vide infra*).

**Hypopituitarism.** *Chronic hypopituitarism* may be revealed preoperatively by clinical examination and the tests previously described. Acute or chronic hypopituitarism may follow at *any* time after removal of

intrasellar or parasellar tumours. Therefore there is a good case for hormone replacement therapy to be begun several days before operation and to be continued post-operatively as long as needed. Adrenocorticotrophic hormone (ACTH) 25 to 40 mg., or cortisone 50 to 75 mg., reduced after a few days to a maintenance dose of 25 mg., are given daily by intramuscular injection, together with 32 mg. ( $\frac{1}{2}$  gr.) of thyroid B.P. and methyl testosterone 25 mg. by mouth.

*Acute hypopituitarism* (hypopituitary coma) may follow operations on tumours within the pituitary region, but it has to be distinguished from : (a) Hypothalamic coma which may arise from disturbing the tumour capsule in contact with or adherent to the hypothalamus, or as the result of post-operative hæmorrhage in the pituitary fossa ; (b) reactionary extradural or subdural hæmorrhage.

As hypoglycæmia is present in a large proportion of cases, glucose should be given without waiting for the results of investigations. The glucose is given by mouth if the patient can be roused ; otherwise it is given intravenously (50 ml. of 50 per cent. glucose followed by continuous 5 per cent. glucose). *Hydrocortisone alcohol* (100 mg. in 500 ml. of normal saline) or *microcrystalline cortisone acetate* (Cortone) are given intravenously as soon as possible (hydrocortisone has a quicker effect than cortisone). Cortisone acetate (50 mg.) is also given every six hours by nasal tube until consciousness is restored. An Addisonian adrenal crisis in which there is severe sodium depletion, azotæmia and circulatory collapse, is uncommon in hypopituitarism, but nevertheless because cortisone may at first increase the sodium output it is advisable to give DCA (deoxycorticosterone acetate) 5 mg. daily for a few days and to increase the intake of sodium.

*Polydipsia* and *polyuria* sometimes follow operations in the pituitary region and are caused by interference with the supraoptico-hypophyseal tracts or the neurohypophysis. Established diabetes insipidus is, however, rare after intrasellar operations, for as previously stated it cannot occur in the absence of functioning anterior pituitary tissue. Diabetes insipidus is treated by intramuscular injections of Pitressin tannate 1 to 2 ml. in oil given on alternate days (Pitressin is a proprietary extract of the vasopressor and antidiuretic factors of the posterior pituitary lobe, i.e. minus the oxytocic fraction).

**Radiotherapy** undoubtedly produces improvement of vision in some cases of pituitary adenoma and may improve the endocrine state found with acidophil adenomas. Some tumours, however, continue to increase in size after radiotherapy. Without operation there is no way of knowing whether the tumour is cystic or solid, or of confirming the diagnosis. Cystic tumours are, of course, insensitive to X-rays. The best results are obtained by operation followed by radiotherapy. There is no indication for radiotherapy in the treatment of craniopharyngiomas, which are pathologically benign and almost always cystic.



## CHAPTER 54

### OPERATIVE TECHNIQUE AND EXPOSURES OF INTRACRANIAL SURGERY

THE successful outcome of intracranial operations depends to a great extent on perfect hæmostasis and replacement of any blood loss as soon as it occurs. An intravenous saline infusion is begun immediately before operation and blood substituted when necessary. In most other branches of surgery, ligation of vessels is the usual way of securing hæmostasis, but in neurosurgery this method is rarely applicable. Diathermy is more frequently employed than any other means of arresting hæmorrhage; bleeding from bone is controlled with Horsley's wax which is pressed into the bone after all soft tissue has been removed with the aid of a rugine; the larger intracranial arteries and veins are occluded with silver or tantalum clips (Fig. 379); and bleeding from venous sinuses is controlled by the application of Gelfoam, gelatin sponge or pounded muscle (obtained from the temporalis). Occasionally ligation of the transverse sinus or

the anterior end of the superior longitudinal sinus is carried out with silk or thread (not catgut which tends to slip). Bleeding points are revealed by saline irrigation followed by suction. When the dura mater has been opened, patties (small squares of cottonoid) are interposed between the tissue and the sucker, except when it is necessary deliberately to suck it away (e.g. in some open head injuries when removal of disintegrated brain is part of the débride-ment).

**Opening the Skull.** The scalp is carefully shaved with a sterile razor, and prepared with an antiseptic solution. (Note.—Some detergents are not sterile.) The skin incision is marked out with a suitable dye (e.g. "triple dye"). When infiltration

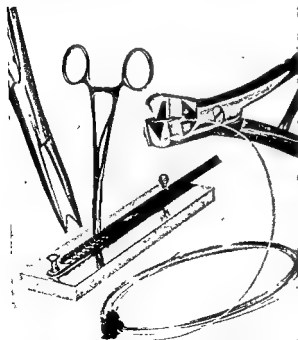


FIG. 379. The McKenzie Cushing clip outfit. On the left a clip is shown mounted in a clip-holding forceps. In the centre is the magazine partially open, and a clip is being picked up. On the right the instrument for making clips from silver wire is shown in action.

anæsthesia is used, the solution is injected subcutaneously and in the subaponeurotic plane along the line of the incision and across the base of the flap. The pericranium, bone, dura (except in the vicinity of the arteries), arachnoid, pia and brain are insensitive. Pressure is applied to the scalp on both sides of the line of the incision by the assistants' fingers until the surgeon has placed fine artery forceps on the cut edge of the

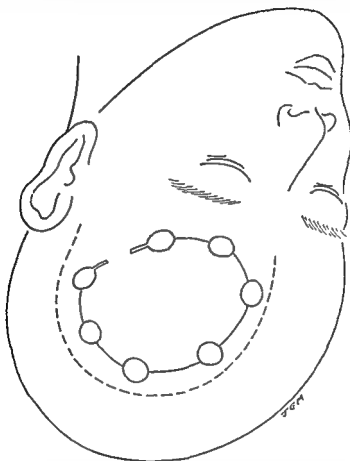


FIG. 380. The frontal osteoplastic flap. The head is orientated as at operation. The skin incision is shown by the broken line.

divided aponeurosis. When these are turned back over the skin edge, their pull controls most of the oozing from the scalp. Vessels which continue to bleed are coagulated with diathermy. The scalpel used for the skin incision is discarded and, as far as is possible, the no-touch technique is maintained. *The neurosurgeon must insist on impeccable aseptic technique, for the consequences of sepsis can be disastrous despite the ever increasing variety of antibiotics.*

In supratentorial approaches, bone flaps are usually based on the temporal muscle (Fig. 380). In this way some blood supply to the bone is

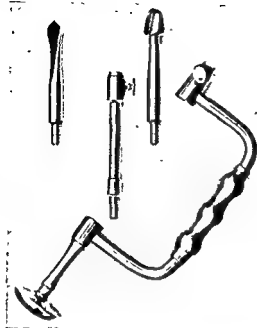


FIG. 381. Hudson's brace on the right, with above, from left to right, a perforator, extension piece for the brace, and a burr.

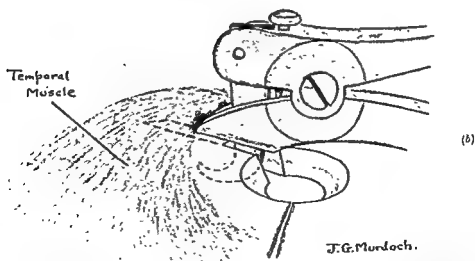
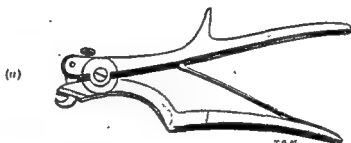


FIG. 382. (a) The De Vilbiss rongeur. The distance between the jaws is adjusted with the thumbscrew to correspond with the thickness of the bone. (b) The instrument is shown in action.

preserved. Small holes are made in the bone with a perforator and burr (Fig. 381). The trephine is no longer employed for this purpose as it lacerates the dura and consumes too much time and energy. The bone is cut between burr-holes with a Gigli saw, except at the base of the flap where it is deliberately fractured after the distance between the two basal burr-holes has been reduced by means of a de Vilbiss rongeur (Fig. 382).

The dura mater is steadied with a sharp hook which is passed into it (not through it), and incised with a round-ended tenotome (a pointed one is liable to puncture underlying cortical vessels). A director or patty (cottonoid square) is passed through the opening to protect the brain while the dura mater is opened. Frontal and parieto-occipital dural flaps are based on the superior longitudinal sinus. The dura is turned back carefully, for veins often pass between it and the brain. They are coagulated and divided unless they drain vital areas of the brain. Before



FIG. 383. A temporal skin incision is shown on the left, and frontoparietal and parieto-occipital incisions on the right.

closure, stitches are inserted between the edge of the dura mater and small holes drilled in the bone, to prevent extradural bleeding arising from separation of the dura mater from the bone. As much of the dural flap as possible is replaced and sutured, especially over vulnerable cortical areas. The bone flap is replaced and fixed by sutures passed through small drill holes in the flap and surrounding bone.

*The Frontal Approach.* The anterior limb of the incision is made in the midline to obtain a good cosmetic result (Fig. 380). When only a small bone flap is required, as in the exposure of a pituitary tumour, a visible scar can be avoided by making a transverse incision behind the hair line and turning the scalp forward over the patient's face. The bone flap is planned to avoid the frontal sinuses. When the flap is deliberately fractured across its base, the middle meningeal vessels are often torn, and if at the point of fracture they lie in a bony canal or deep groove, coagulation of the vessels is facilitated if the bone is nibbled away until they are freed.

Examples of incisions for *temporal*, *fronto-parietal* and *parieto-occipital* exposures are illustrated in Fig. 383.

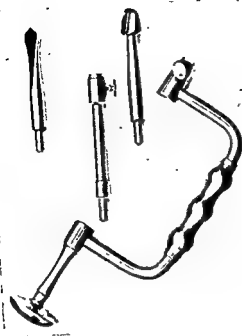


FIG. 381. Hudson's brace on the right, with above, from left to right, = perforator, extension piece for the brace, and a burr.

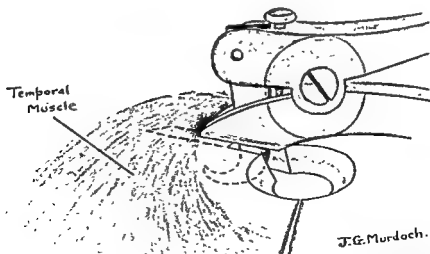
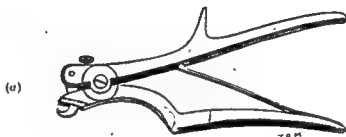


FIG. 382. (a) The De Vilbiss rongeur. The distance between the jaws is adjusted with the thumbscrew to correspond with the thickness of the bone. (b) The instrument is shown in action.

compression of the medulla oblongata. In addition to loss of consciousness, there is therefore slowing of the pulse and respiration rates, increasing blood pressure, and finally Cheyne-Stokes respiration. Supratentorial hæmorrhage, if untreated, finally produces the medullary syndrome as the result of downward coning of the cerebellum.

Immediate operation is indicated to evacuate post-operative clots and to secure hæmostasis. Operation is especially urgent when hæmorrhage occurs in the posterior fossa.

**Brain Swelling.** Swelling of the brain may be present before operation, as with some tumours. It may also occur or increase during operation ;



FIG. 385. Example of wound sepsis. The bone flap had to be removed before healing could begin. Later some form of cranioplasty would be necessary.

anoxia is sometimes an important contributory factor. Brain swelling may not appear until several days after operation. Evidence of early post-operative œdema is clinically indistinguishable from that of hæmorrhage. When the syndrome occurs after supratentorial operations the bone flap is removed ; after infratentorial operations it may be possible to extend the craniectomy and thus decompress the brain stem. All patients suffering from progressive brain swelling are treated by dehydration which may succeed alone if the compression is not too severe. Dehydration may be accomplished by magnesium sulphate enemas (3 oz. magnesium sulphate in 6 oz. water) repeated six-hourly, or by intravenous *sucrose* (50 to 100 ml. of a 50 per cent. solution in water). The latter method may also be applied during operations when marked œdema is observed. Dehydration produced by glucose or hypertonic saline is followed by a reactionary increase in œdema.

*Posterior fossa incisions* are illustrated in Fig. 384. Several burr-holes are made and intervening bone removed with rongeurs. Incisions are made in the dura mater over the cerebellar hemisphere(s), and continued to the foramen magnum. The occipital sinus (running in the midline from the region of the foramen magnum to the confluence of the transverse sinuses) is often absent or small; *but sometimes, especially in children, it is very large, and, if damaged, may cause torrential hæmorrhage.* When there is a marked pressure cone, the posterior arch of the atlas is

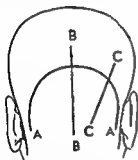


FIG. 384. A. The standard curved incision. B. The midline Frazier incision. C. The lateral oblique incision.

removed, and the dura opened as far as the upper border of the axis. After the removal of tumours, no attempt is made to suture the dura, for decompression is required to allow for cerebellar swelling, which might otherwise affect the vital medullary centres, and after most procedures carried out in the posterior fossa, the dura cannot usually be completely approximated because it shrinks after being incised.

At the end of all intracranial operations, complete hæmostasis is essential, but it is a common error for the inexperienced operator, in his enthusiasm to attain this ideal, to cause bleeding by sucking directly on the tissues to

verify hæmostasis. The scalp is sutured in two layers with fine waxed silk; small round-bodied curved split-eye needles are employed for the galea and sharp straight needles for the skin. Suturing the scalp in two layers discourages the formation of a cerebrospinal fistula and permits early removal of the skin stitches.

#### COMPLICATIONS OF INTRACRANIAL OPERATIONS

**Reactionary Hæmorrhage.** The fundamental cause of reactionary hæmorrhage is imperfect hæmostasis arising from: (a) Lack of care; (b) arterial hypotension which conceals open vessels at the time of operation, the hypotension may arise from shock or deliberate administration of *hypotensive drugs*; or (c) venous bleeding occurring when the patient is put to bed after being operated on in the sitting position. Reactionary intracranial hæmorrhage usually reveals itself within a few hours of operation. Consciousness may not be regained, or after recovery from anaesthesia the level of consciousness declines. The scalp over the operation site is likely to be tense. In supratentorial hæmorrhage there may be dilatation of the homolateral pupil and contralateral hemiplegia. Sometimes homolateral signs occur from brain-stem compression. The pulse rate increases at first, but later may be subnormal. Note.—This syndrome closely resembles that caused by post-traumatic extradural hæmorrhage (Chap. 50).

In the posterior fossa, hæmorrhage causes acute hydrocephalus and

## CHAPTER 55

### INFANTILE HYDROCEPHALUS

THE cerebrospinal fluid is produced by the choroid plexuses of the two lateral ventricles, the third ventricle and the fourth ventricle. The fluid flows from the lateral ventricles through the foramen of Monro into the third ventricle and through the aqueduct of Sylvius into the fourth ventricle. It leaves the ventricular system through the foramen of Magendie and the two foramina of Luschka to flow through the sub-arachnoid space, including the cisterns, and then to be absorbed mostly by the *capillaries* over the surface of the cerebral hemispheres. Obstruction anywhere along this pathway causes increased pressure and consequent dilatation of ventricles and cisterns "proximal" to the obstruction. This phenomenon is termed *hydrocephalus*. It has been stated that the increased production of cerebrospinal fluid occurring with papillomas of the choroid plexus can cause hydrocephalus, but *some* of these tumours also *obstruct* the flow of cerebrospinal fluid. Occlusion of the venous sinuses is not now thought to be a cause of hydrocephalus.

The clinical characteristics of hydrocephalus are enlargement of the head (see table), prominent forehead, stretching of the scalp, dilatation of the veins of the scalp, tense enlarged fontanelles, which do not become depressed when the patient is upright, separation of the sutures, displacement of the eyeballs downward, sometimes adduction of the eyes (abducens paralysis), and a "cracked pot" sound on percussion of the skull. There is seldom papilloedema, although optic atrophy may be observed in long-standing cases. If hydrocephalus is allowed to persist, dementia and spastic paralysis occur and some patients develop epilepsy. Eventually in patients who

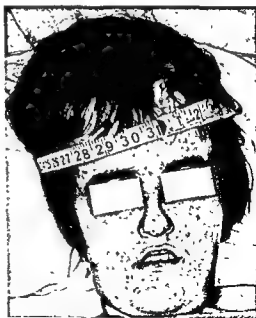


FIG. 387. A hydrocephalic girl of fifteen years with grand mal epilepsy. The circumference of her head was 31 inches (normal at this age 21 inches). The other signs were dementia, right abducens palsy, and paraplegia; radiography showed that all skull sutures had united.



**Wound Sepsis.** There are many links in the chain of asepsis, and a fault in any one of them may cause the chain to break, with perhaps disastrous consequences. The bone flap may become infected and have to be removed (Fig. 385). Infection may spread deeply and cause meningitis

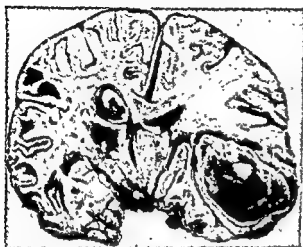


FIG. 386. Infection of the wound after removal of a benign tumour caused this unsuspected temporal abscess.

or a brain abscess (Fig. 386). The surgeon should periodically check all methods of sterilisation and, as far as neurosurgery permits, he and his assistants should adhere to the no-touch technique. (Note.—About 50 per cent. of gloves are punctured during operations; they should always be changed when the bone work is completed.) *Meningitis and brain abscess* are treated as described in Chap. 51.

#### Cerebrospinal Fluid Fistula.

This is sometimes caused by inadequate suturing. Wounds may also break down as the result of sepsis or unrelieved intracranial pressure. Conservative treatment is usually successful whereas operative interference makes matters worse. Fluid intake should be restricted. Strict asepsis, including the wearing of a mask by the dresser, should be observed at dressings. Broad spectrum antibiotics are given, and withdrawal of cerebrospinal fluid is carried out daily by lumbar puncture.

**Epilepsy** sometimes follows operations on the cerebrum. The attacks may be generalised or Jacksonian. Treatment follows the principles enunciated in Chap. 57.

localised swelling noted. As complete a neurological examination as the patient's age permits, is carried out. Plain radiography may give important information. Thus, in hydrocephalus there is separation of the sutures and sometimes rarefaction of the posterior clinoid processes. Some tumours are calcified, e.g. most craniopharyngiomas (see also Chap. 53). *Needling* is carried out through the separated sutures, when available, or through frontal, parietal and sometimes temporal burr-holes. This procedure may reveal a subdural hæmatoma or hygroma (syn. hydroma)—see also Chap. 50. If surface needling is negative, the needle is passed further inwards until the ventricle is entered and its depth from the surface of the brain noted. Sufficient air is then injected into both lateral ventricles (ventriculography) to fill only the anterior horns. Children do not tolerate a large volume of air. This investigation sometimes gives evidence of a supratentorial cyst or tumour. Occasionally, the needle enters a cyst instead of the ventricle, and typical yellow fluid of high protein content is withdrawn. A cystogram may then be obtained by injection of air or diodone (Pyelosil). The latter should never be injected into the ventricles. When air ventriculography does not give sufficient information, Myodil (1 to 2 ml.) is injected into one of the lateral ventricles in the direction of the foramen of Monro. This procedure will demonstrate lesions in the region of the foramen of Monro, the third ventricle, the aqueduct of Sylvius, the fourth ventricle and the cisterna magna. The use of Myodil supplants the somewhat unreliable dye tests employed to show whether the hydrocephalus communicates with the subarachnoid space or not. The use of Myodil (positive contrast ventriculography) gives more information than any other investigation.

### CAUSES OF HYDROCEPHALUS

(Classified according to sites of obstruction)

#### In the Lateral Ventricles

Papilloma of the choroid plexus (rare).

#### At the Foramen of Monro

*Craniopharyngioma* (syn. cyst of Rathke's pouch—*vide* Chap. 53).

*Colloid cyst* (*vide* Chap. 53)—although congenital, it usually shows itself in adult life.

*Gliomas* arising in the cerebrum obstructing the foramen of Monro.

#### In the Third Ventricle

*Glioma* of the basal ganglia and thalamus.

*Papilloma* of the choroid plexus (rare).

*Pineal tumours* (rare).

survive long enough, the head ceases to enlarge and the sutures unite (Fig. 387). Presumably production of cerebrospinal fluid is largely suppressed in such cases.

TABLE

*Normal Circumference of Head from Birth to Fourteen Years*

(From B. Myers: Statistics concerning the height, weight and other measurements of fourteen hundred London children. *Brit. J. Child. Dis.*, 1926, 23, 87.)

Age				Male		Female	
				cm.	inches	cm.	inches
0 - 1	month	.	.	36.0	14.4	35.0	14.0
1 - 2	months	.	.	37.9	15.2	37.6	15.0
2 - 3	"	.	.	39.0	15.6	38.4	15.4
3 - 4	"	.	.	40.0	16.0	39.2	15.7
4 - 5	"	.	.	41.7	16.7	40.8	16.3
5 - 6	"	.	.	41.9	16.8	41.1	16.4
6 - 9	"	.	.	43.5	17.4	44.0	17.6
9 - 12	"	.	.	45.5	18.2	44.7	17.9
12 - 15	"	.	.	46.5	18.6	44.9	18.0
15 - 18	"	.	.	48.1	19.2	46.2	18.5
18 - 21	"	.	.	47.7	19.1	46.3	18.5
21 - 24	"	.	.	47.2	18.9	48.3	19.3
2 - 2½	years	.	.	49.1	19.6	47.5	19.0
2½ - 3	"	.	.	48.6	19.4	47.7	19.1
3 - 4	"	.	.	49.8	19.9	48.7	19.5
4 - 5	"	.	.	49.5	19.8	49.3	19.7
5 - 6	"	.	.	50.6	20.2	49.5	19.8
6 - 7	"	.	.	50.6	20.2	50.2	20.1
7 - 8	"	.	.	51.9	20.8	50.5	20.2
8 - 9	"	.	.	51.9	20.8	50.6	20.2
9 - 10	"	.	.	51.4	20.6	51.2	20.5
10 - 11	"	.	.	52.3	20.9	51.6	20.6
11 - 12	"	.	.	52.9	21.2	51.6	20.6
12 - 13	"	.	.	52.9	21.2	51.9	20.8
13 - 14	"	.	.	53.6	21.4	52.0	20.8

**Diagnosis.** Children presenting with enlargement of the head *should be just as carefully examined and investigated as adults presenting with evidence of increased intracranial pressure.* Enlargement of the head may be caused by lesions producing obstruction of the cerebrospinal fluid pathway (hydrocephalus), by progressively expanding lesions which do not obstruct the flow of cerebrospinal fluid, or by lesions which cause raised intracranial pressure, both by obstructing the cerebrospinal fluid and by their increasing size. Rarely, enlargement of the head is not accompanied by any intracranial abnormality (*macrocrania*).

The circumference of the head is measured, and any asymmetry or

*Cystic glioma* obstructing the foramen of Monro or the third ventricle.

*Solid frontal gliomas* obstructing the foramen of Monro.

*Membranous occlusion* of the aqueduct of Sylvius.

*Cerebellar tumours* and *cysts* of the posterior fossa.

*Cerebellar abscess*.

*Papilloma* of the choroid plexus of the fourth ventricle.

*Congenital occlusion of the foramen of Magendie* (meningitic occlusion is usually accompanied by adhesions of the basal leptomeninges, and is therefore not amenable to direct attack).

*Arnold-Chiari Deformity.* Posterior fossa decompression and upper cervical laminectomy are performed and the cerebellar tonsils separated to re-establish the foramen of Magendie. If, however, obstruction of the aqueduct of Sylvius has been demonstrated by Myodil, a ventriculo-cisternostomy (*vide infra*) should be performed instead. If there is an associated spina bifida with paraplegia and paralysed sphincters, humanitarian principles contraindicate operation.

Direct attacks are planned and carried out according to the methods described under Intracranial Tumours (Chap. 53).

### Lesions Treated by Short-circuit

*Craniopharyngioma* (mortality of direct attack is prohibitive).

*Solid gliomas* obstructing the third ventricle or the midbrain.

*Papilloma* of the choroid plexus of the third ventricle.

*Congenital stenosis*, "forking" and *atresia* of the aqueduct.

*Meningitic occlusion* of the foramina of Magendie and Luschka (usually associated with basal leptomeningeal adhesions).

*Meningeal adhesions* (meningitic or due to hæmorrhage).

**Short-circuiting Operations.** *Ventriculo-cisternostomy* (Torkildsen's Operation). The indications for this operation are irremovable obstructive lesions affecting the foramen of Monro, third ventricle or the aqueduct of Sylvius. Permanent drainage of cerebrospinal fluid is established between one lateral ventricle and the cisterna magna through a small-size rubber tube. A burr-hole is made in the occipital region, usually on the right side, and the dura opened. Next, a small posterior fossa craniectomy, including the posterior margin of the foramen magnum, is carried out through a midline longitudinal incision.

The rubber tube is passed under the scalp between the burr-hole incision and the posterior fossa exposure. One end is passed several centimetres into the lateral ventricle, and the other is passed through a small incision in the dura and arachnoid into the cisterna magna. Here, the tube is sutured to the margins of the dural incision which is closed around the tube. N.B.—The occipital venous sinus may be large in infants and cause severe hæmorrhage during this operation. When the foramen of Monro is blocked (e.g. by a craniopharyngioma) both lateral ventricles must be drained into the cistern by separate tubes.

### At the Level of the Aqueduct of Sylvius

*Congenital stenosis* of the aqueduct.

*Congenital "forking"* of the aqueduct (instead of one aqueduct there are multiple small channels).

*Congenital atresia* of the aqueduct ; occasionally only a thin membrane may be present.

*Gliomas of the midbrain.*

*Periaqueductal gliosis.*

*Vascular malformations* which sometimes encircle and penetrate the midbrain.

### At the Level of the Fourth Ventricle

*Cerebellar tumours* (astrocytomas, medulloblastomas and ependymomas.)

*Cysts* (dermoid, epidermoid, arachnoid and ependymal).

*Chronic cerebellar abscess* (rare).

*Papilloma* of the choroid plexus (rare).

### At the Foramina of Magendie and Luschka

*Occlusion of the foramina* : (a) Congenital ; (b) meningitic (pyogenic or tuberculous)—in either acute or chronic stages.

*Arnold-Chiari syndrome* (tongue-like caudal prolongation of the cerebellum firmly adherent to the elongated and caudally displaced medulla oblongata). There is almost always an associated spina bifida. It is undecided whether this is a primary congenital deformity or secondary to prolonged intracranial pressure (i.e. an extreme example of a pressure cone—Chap. 53).

### In the Basal Cisterns and over the Cerebral Hemispheres

*Leptomeningeal adhesions* : (a) Meningitic (pyogenic or tuberculous)—in either acute or chronic stages ; (b) due to subarachnoid hæmorrhage resulting from birth injury.

The common causes of infantile hydrocephalus are stenosis, "forking" and atresia of the aqueduct ; cerebellar tumour ; and leptomeningeal adhesions caused by meningitis (pyogenic or tuberculous), or subarachnoid hæmorrhage (due to birth injury).

### TREATMENT

Treatment depends on the site and pathology of the obstructing lesion which, whenever possible, should be directly attacked. If the lesion is not amenable to direct attack, a short-circuiting operation is performed.

#### Lesions Attacked Directly

*Papilloma* of the choroid plexus of the lateral ventricle.

*Colloid cyst* (vide Chap. 53).

## CHAPTER 56

# THE SURGERY OF INTRACRANIAL VASCULAR DISEASES

### CONGENITAL ANEURYSMS

*Congenital saccular aneurysms* (syn. "berry" aneurysms) may be found anywhere on the intracranial arterial tree, although the common sites are : (a) *The internal carotid artery* in the cavernous sinus ; (b) *the internal carotid artery* in its subarachnoid course, i.e. between the point where it pierces the dura mater and where it divides into the anterior and middle cerebral arteries ; (c) *the anterior cerebral artery* at the point of origin of the anterior communicating artery—often referred to as *anterior communicating aneurysms* ; (d) *the middle cerebral artery* at the origin of the terminal branches ; and (e) *the peripheral course* of the anterior and middle cerebral arteries. Aneurysms in the posterior fossa are rare.

Congenital aneurysms are thought to result from absence of the media at the sites of origin. This view is supported by the occurrence of many aneurysms at arterial branching points where defects of the media often occur. Also, aneurysms themselves, when examined histologically, show defective or absent media. However, according to Dandy, this theory is untenable because so many aneurysms occur away from arterial junctions. He suggested that as arteries are originally derived from capillary networks, congenital aneurysms may be derived from incompletely absorbed capillaries.

**Clinical Manifestations.** Despite their apparent congenital origin, these aneurysms do not usually reveal their presence in the first two decades of life. Manifestations have their maximum incidence in the fifth and sixth decades. The commonest presentation, except in the case of intracavernous carotid aneurysms, is subarachnoid hæmorrhage [other causes of subarachnoid hæmorrhage are : (a) Cerebral hæmorrhage ; (b) vascular malformation (hamartoma\*) ; (c) primary tumour—a rare cause ; (d) secondary tumour—a rare cause, but subarachnoid hæmorrhage is a characteristic of secondary melanoma]. Some aneurysms produce focal neurological disturbances (*vide infra*). Occasionally an aneurysm is large enough to be mistaken for a tumour.

**Syndrome of Subarachnoid Hæmorrhage.** Patients are seized with sudden, severe, generalised headache and consciousness may be lost. On examination, there is neck-stiffness, a positive Kernig's sign and sometimes papilloedema. Occasionally pre-retinal (subhyaloid) hæmorrhages occur. Lumbar puncture reveals blood in the cerebrospinal fluid which, after centrifuging, is usually found to be xanthochromic. If the puncture

\* Hamartoma = a tumourous mass of superfluous tissue.

*Subarachnoid-ureterostomy.* The indications for this operation are obstructive adhesions in the basal cisterns and/or cerebral leptomeninges (i.e. "distal" to the posterior fossa). It may be performed when fluid accumulates in the posterior fossa after ventriculo-cisternostomy (Torkildsen's operation). The fluid is diverted through a thin polythene tube from the spinal subarachnoid space to one of the ureters, made available by nephrectomy. Before operation, the presence of two kidneys is checked by intravenous pyclography, and the urine is examined for organisms and abnormal chemical constituents. Nephrectomy is performed on the left side because the left kidney is higher than the right and therefore has a longer ureter. Laminectomy (lumbar 3 and 4) is next carried out. A thin polythene tube, fixed into an appropriate curve after soaking it in hot water, is inserted through a small incision in the dura and arachnoid so that several centimetres of the tube lie caudal to the incision among the roots of the cauda equina. A passage is made for the tube to the ureter through the sacrospinalis muscle. The ureter is disturbed as little as possible in order to conserve its blood supply. The polythene tube is inserted several centimetres into the ureter which is tethered to the lumbar fascia but *not* to the tube. Patients often need extra sodium chloride after operation. The tube does not need to be changed as the child grows.

*Ventriculo-ureterostomy.* This procedure is indicated when adhesions are present throughout the subarachnoid space.

A long polythene tube is inserted into one of the lateral ventricles through an occipital burr-hole and passed under the skin from one small incision to another down the back to join with a ureter made free by nephrectomy. The tube is passed several centimetres into the ureter as in the previous operation.

**Conclusions.** A child with a large head is not necessarily suffering from hydrocephalus. Hydrocephalus is a physical sign ; it is not a diagnosis and therefore rational treatment can be carried out only after accurate diagnosis of the site and pathology of the obstruction.

Homolateral exophthalmos occurs at first, but later the other eye is affected.

**Subarachnoid Carotid Aneurysms.** These aneurysms arise from the posterior aspect of the internal carotid between the point at which the artery pierces the *dura mater* and its bifurcation into anterior and middle cerebral arteries (Fig. 389). They are sometimes bilateral. The commonest manifestation is *subarachnoid hæmorrhage*. Sometimes homolateral oculomotor palsy, including *dilatation* of the pupil, occurs before, during or after subarachnoid hæmorrhage. It is due to pressure of the aneurysm on the third cranial nerve. In some cases, oculomotor palsy



FIG. 389. A larger than average subarachnoid aneurysm of the internal carotid artery. It gave rise to subarachnoid hæmorrhage.

and homolateral retro-ocular pain occur without subarachnoid hæmorrhage. Rupture of an aneurysm may cause arterial spasm and resultant hemiplegia; and when the dominant side is involved, aphasia also occurs. Widespread arterial spasm causes unconsciousness. Infarction of most of the affected cerebral hemisphere is found in fatal cases. Other factors contributing to unconsciousness are acute cerebral swelling occurring in some cases of infarction, and increased cerebrospinal fluid pressure produced by subarachnoid bleeding. Occasionally a subarachnoid aneurysm of the internal carotid causes unilateral blindness with a central or paracentral scotoma (scotoma = blind or partly blind area enclosed within the visual field) or diminished visual acuity and *bitemporal hemianopia* (Fig. 390).

**Aneurysms of the Anterior Cerebral Artery.** These aneurysms cause subarachnoid hæmorrhage, usually without localising signs. Occasionally,



is performed a few days after the hæmorrhage, the fluid is xanthochromic, but red cells are no longer present. Bleeding produced by lumbar puncture is more marked at the beginning of the procedure than at the end, and the centrifuged fluid is colourless.

**Intracavernous non-fistulous carotid aneurysms** (Fig. 388) present with pain in one or more divisions of the trigeminal nerve and/or homolateral ophthalmoplegia. An absent corneal reflex may be the only objective sign of involvement of the trigeminal nerve, or there may be sensory loss affecting any or all divisions of the trigeminal, and unilateral paralysis

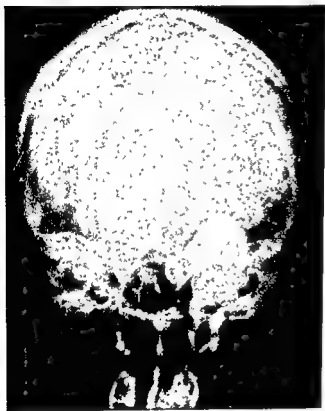


FIG. 388. Angiogram (Towne's view) showing a large left internal carotid aneurysm which had expanded the cavernous sinus causing very severe pain in the distribution of the trigeminal nerve. The pain, which was not controlled by drugs, was relieved by internal carotid ligation in the neck followed by intracranial clipping of the same artery above the aneurysm. Two years later the patient developed right hemiplegia and aphasia.

of the muscles of mastication. One or more of the nerves supplying the muscles of the eye may be paralysed. In oculomotor paralysis, the pupil is usually in the cadaveric position due to a combination of parasympathetic paralysis and stretching of the carotid sympathetic nerves by expansion of the aneurysm. Spontaneous remission of the syndrome sometimes occurs.

**Intracavernous Fistulous Aneurysms.** A fistula between the internal carotid artery and the cavernous sinus may occur *spontaneously*. It may also occur in closed head injuries, or in missile or other types of puncture wounds. Patients usually complain of an intracranial bruit which can be heard on auscultation of the skull or more distinctly by applying the stethoscope gently to the patient's closed eye on the side of the lesion.

of the brain ; and (d) *raised intracranial pressure* caused by subarachnoid hæmorrhage. The amount of blood lost is not sufficient to contribute to the patient's decline, but the presence of blood in the cerebrospinal fluid contributes substantially to the rise in intracranial pressure.

Even when localising signs are present, bilateral percutaneous common carotid angiography is always necessary to demonstrate the site of the aneurysm, to reveal the presence of a possible space-occupying hæmatoma and to give information about the arterial circulation. In some cases, a vascular malformation may be revealed ; in others no cause for the

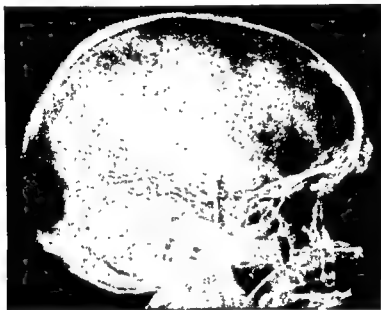


FIG. 301. Aneurysm of the upper end of the basilar artery revealed by vertebra angiography. The patient had a subarachnoid hæmorrhage.

bleeding is shown. When carotid angiography is negative (25 per cent. of cases of subarachnoid hæmorrhage), some neurosurgeons proceed to *vertebral angiography*, but the hazards of this procedure are considerable in comparison with the information likely to be revealed. It is safer to carry out ventriculography which may demonstrate the presence of an intracerebral hæmatoma. When a hæmatoma is evacuated, an aneurysm may be discovered in its wall. The filling defect caused by blood in the ventricles should not be mistaken for the deformity produced by an intracerebral hæmatoma.

**Surgical Treatment.** *Intracavernous non-fistulous aneurysms*, which cause trigeminal pain and ophthalmoplegia do not need surgery, unless the pain is intolerable (Fig. 388), for spontaneous regression sometimes occurs. Furthermore, this type of aneurysm rarely extends above the

however, when both anterior cerebral arteries are in spasm, the leg area of the cerebral cortex is involved bilaterally and paraplegia results. But in the presence of spasm of these two vessels, the patient is likely to be deeply unconscious so that paraplegia cannot be demonstrated. Un-

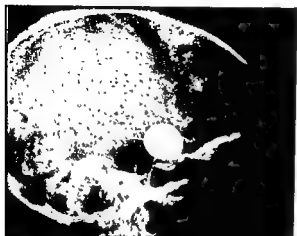


FIG. 390. A large aneurysm of the left internal carotid artery. The aneurysm arose from the medial side of the internal carotid artery and lifted the chiasm upward. The patient complained of declining visual acuity and was found to have bitemporal hemianopia.

sites are at the upper and lower ends of the basilar artery (Fig. 391) and on the vertebral arteries.

#### MANAGEMENT OF CONGENITAL INTRACRANIAL ANEURYSMS

The mortality of subarachnoid hæmorrhage treated conservatively is 50 per cent. Many patients who recover from the first hæmorrhage die from a recurrence within two to three weeks. The factors which, singly or in combination, cause death are : (a) Infarction of the brain ; (b) swelling which may accompany infarction ; (c) hæmorrhage into vital parts

consciousness may also occur as the result of intracerebral hæmorrhage involving the hypothalamic region. The site of the aneurysm is commonly at the point of origin of the *anterior communicating artery* which may be replaced by the aneurysm. The latter may be filled from either, or both anterior cerebral arteries.

**Aneurysms of the Middle Cerebral Artery.** These aneurysms often bleed without causing localising signs. Hemiplegia, however, sometimes occurs as the result of arterial spasm or intracerebral hæmorrhage, and when the dominant hemisphere is involved dysphasia may result. The commonest site for aneurysms on the middle cerebral artery is at the point where the main trunk divides into its terminal branches.

**Aneurysms of the posterior cerebral artery** are rare. Sometimes hemianopia is produced by arterial spasm or cerebral hæmorrhage.

**Aneurysms in the posterior fossa** are rare. The classical

*Posterior cerebral aneurysms* usually require vertebral angiography for their demonstration. They are somewhat inaccessible on the medial surface of the occipital lobe. The more peripheral ones can be trapped at the expense of hemianopia which is produced by the handling or amputation of the occipital lobe required in the exposure.

**Carotid Ligation.** Ligation anywhere along the course of the common or internal carotid artery may cause immediate or *delayed* neurological dysfunction. If ligation or temporary occlusion is contemplated, the common carotid is first compressed digitally in the neck through the intact skin for up to ten minutes (Matas' test). If consciousness is disturbed or contralateral neurological signs appear during this time, the pressure is relaxed and ligation not undertaken. If ten minutes (it is difficult to maintain pressure for a longer period) of digital pressure is tolerated, the common carotid artery is exposed under infiltration anaesthesia through an incision in a transverse skin crease and a clamp applied to the artery. If no abnormal signs appear after twenty minutes, a ligature is applied to the common carotid artery. Ligature of this artery still allows some blood to reach the homolateral hemisphere from its own internal carotid artery which receives blood from the external carotid artery, derived from the capillary anastomosis between the two external carotid systems. After two weeks' interval, during which intracranial anastomoses have been given the chance to adapt themselves, the internal carotid is exposed through an incision in the line of the inferior ramus of the mandible (again in a skin crease), and a temporary clamp applied to the artery. If no neurological signs appear in twenty minutes the artery is ligated. *Despite the above precautions, hemiplegia and, in the case of the dominant hemisphere, aphasia may occur at any time.* Carotid ligation should not be undertaken on unconscious patients, for their tolerance of this procedure cannot then be ascertained.

**Aids to Surgery.** Removal of cerebrospinal fluid by tapping both lateral ventricles relaxes the brain and thus facilitates the exposure of aneurysms. *Induced hypotension*, obtained by administration of *Arfonad*, helps to control the bleeding which may occur from aneurysms when they are being exposed or being clipped. *Induced hypothermia*, by reducing metabolic requirements, including those of the brain, enables clips to be applied to the feeding vessels of an aneurysm while it is being defined and clipped. *In dogs*, with the body temperature reduced below 25° C., cerebral vessels may be occluded for twenty minutes without producing histological or neurological signs of cerebral damage. Under normal conditions, infarction occurs if the period of occlusion exceeds four minutes.

**Operative Exposures.** A temporal flap is indicated for clipping the neck of an aneurysm of the internal carotid artery or the distal part of the middle cerebral artery. A bifrontal flap is preferred for operations on anterior cerebral aneurysms. Aneurysms on the posterior cerebral artery are

cavernous sinus and therefore is unlikely to rupture into the subarachnoid space.

*Intracavernous fistulous aneurysms* which cause an intolerable bruit may be treated by carotid ligation in the neck (*vide infra*). This procedure relieves the symptom considerably, although a slight murmur is still heard by the patient and on auscultation. After a variable interval, the bruit returns to its former intensity and intracranial clipping or ligation of the internal carotid artery (i.e. above the fistula) has to be considered. Even after this operation the bruit may return, for there is a reversed flow through the ophthalmic artery (from the external carotid anastomosis) to the internal carotid artery, below the intracranial ligation. It is unlikely that the ophthalmic artery could be clipped without causing homolateral blindness.

*Subarachnoid internal carotid aneurysms* (Fig. 389) may be treated by cervical carotid ligation with due regard to the hazards of this procedure (*vide infra*), or by the application of a clip across the base of the aneurysm. Exposure is facilitated by aspiration of cerebrospinal fluid from the ventricles. Ligation or clipping of the internal carotid artery must be carried out under infiltration anaesthesia so that the circulation through the artery can be re-established if neurological signs appear. Some neurosurgeons do not operate on internal carotid aneurysms on the dominant side for fear of producing aphasia.

*Anterior cerebral aneurysms*, especially those involving the anterior communicating artery, are difficult to treat because of the danger, at operation, of occluding both anterior cerebral arteries. If this accident occurs, death is inevitable, and while life lasts, paralysis of both legs can be observed if the level of consciousness permits. If it is certain that the aneurysm arises from one anterior cerebral artery, the latter may be clipped or ligated proximal and distal to the aneurysm (trapping), or the aneurysm may be wrapped in a piece of muscle or dura mater. Rarely, the aneurysm has a well-defined neck which can be clipped. Carotid ligation is valueless in the treatment of these aneurysms. Associated intracerebral haematomas should be evacuated.

*Middle Cerebral Aneurysms.* Ligation of the middle cerebral artery close to its origin causes severe contralateral hemiplegia and sensory loss by cutting off the blood supply of the internal capsule and nearby structures. On the dominant side, *aphasia* is also produced. Ligation of the middle cerebral artery just proximal to an aneurysm occurring at the point of division of the artery into its peripheral branches, causes a motor loss which varies from facial paresis to hemiparesis. Therefore, if all concerned are willing to accept this neurological deficit, an aneurysm on the non-dominant side may be trapped by the application of proximal and distal clips. On the dominant side, an aneurysm may be wrapped with a piece of muscle or dura mater. Coexistent intracerebral haematomas should be evacuated. Carotid ligation is of doubtful value for these aneurysms.

**Treatment.** If *epilepsy* does not respond adequately to drugs, excision of the lesion and surrounding epileptogenic cerebral tissue is considered. The resultant neurological deficiency has to be weighed against the disabling effect of the epilepsy. *Subarachnoid hæmorrhage* is frequently associated with an *intracerebral hæmatoma* which tends to penetrate deeply to involve vital structures. Clipping of feeding vessels and sometimes total removal of the lesion are possible. When *localised neurological defects* are produced by vascular malformations, there is no indication for surgery in the absence of hæmorrhage.

### SPONTANEOUS HÆMORRHAGE IN THE BRAIN

Spontaneous hæmorrhage may occur anywhere in the brain. It is very common in the cerebrum, but is relatively rare elsewhere. The causes are : (a) Congenital defects of the media of *arteries* within the brain, frequently associated with arterial hypertension (essential or malignant, and hypertension of chronic nephritis or coarctation of the aorta) ; (b) *hæmorrhagic infarction* (some authorities believe most cases of classical cerebral hæmorrhage affecting the internal capsule to be hæmorrhagic infarcts caused by occlusive arterial disease) ; (c) *congenital aneurysms* of the subarachnoid arteries ; (d) *vascular malformations* (hamartomas) ; (e) *mycotic aneurysms* in bacterial endocarditis ; (f) *blood disease*, especially acute leukæmia. Tumours of the brain rarely cause intracerebral hæmatomas, although malignant gliomas frequently contain hæmorrhagic infarcts.

The diagnosis of hæmorrhage in the brain is suggested by the sudden onset of the illness. Localisation is established by clinical means and the usual neurosurgical diagnostic procedures (burr-holes, angiography or ventriculography).

*Progressive cerebral hæmorrhage* calls for urgent operative treatment. Patients suffering from the rare but very lethal *cerebellar hæmorrhage* and surviving long enough to be admitted to hospital should *always* be operated on as soon as possible. The diagnosis is suggested by the sudden onset of vertigo, headache, and *persistent vomiting*, and the finding of a rising blood pressure, bradycardia and slow respiratory rate. Hæmorrhage in the brain stem is inaccessible to the surgeon.

**Surgical Management.** The diagnosis of hæmorrhage is confirmed by aspiration through a burr-hole over the site of the lesion. If the brain bulges through the incision made in the dura, a bone flap is elevated and the hæmatoma evacuated through an incision in a relatively silent part of the brain. The cause of the hæmorrhage is not usually found, except in those cases in which the hæmatoma is caused by an aneurysm or vascular malformation. These lesions should then be treated as previously described in this chapter.

rarely found and still more rarely attacked. An occipital flap would be used. Very occasionally, aneurysms in the posterior fossa, diagnosed by vertebral angiography or discovered accidentally, are amenable to surgery. Large ones may be mistaken for tumours and their true nature may not be discovered until torrential bleeding occurs on attempted removal. The hæmorrhage may be controlled by the application of muscle inside the aneurysm.

**Other Types of Aneurysm.** *Mycotic* aneurysms may occur in bacterial endocarditis and cause subarachnoid and/or intracerebral hæmorrhage. *Arteriosclerosis* may cause fusiform aneurysms, especially on the basilar, vertebral and internal carotid arteries. Various cranial nerve palsies may arise. Rupture does not occur. Neither *syphilis* nor *polyarteritis nodosa* causes intracranial aneurysms.

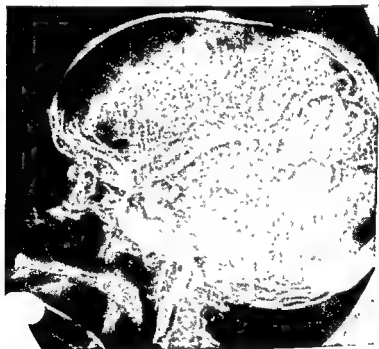


FIG. 392. A vascular malformation in the left parietal region giving rise to epilepsy. The attacks were almost completely controlled by drugs.

### VASCULAR MALFORMATIONS

(Syn. Hamartomas)

These terms apply to tangles of tortuous superfluous vessels, sometimes erroneously called angiomas (Fig. 392). Most of them have *arteriovenous* connections. Vascular malformations may cause : (a) Generalised or Jacksonian epilepsy ; (b) a localised neurological deficit, depending on the site of the lesion ; (c) subarachnoid hæmorrhage ; or (d) an intracerebral hæmatoma, with or without associated subarachnoid hæmorrhage.

## CHAPTER 57

### THE SURGERY OF EPILEPSY AND MENTAL DISEASES

**Epilepsy** is a spontaneous episodic discharge of neurons from a focus anywhere in the cerebral cortex, cerebral ganglia or brain stem in a susceptible brain. The discharge which is accompanied by characteristic electrical activity usually spreads to involve part or all of the cerebral cortex.

#### CLINICAL SYNDROMES

**Grand mal** (major epilepsy) is the term applied to attacks which spread very rapidly to involve the whole of the cerebral grey matter irrespective of the site of origin of the attacks. An aura indicating the origin of the discharge may or may not be reported by the patient. Loss of consciousness occurs. *Tonic* contraction of all voluntary muscles is followed by generalised *clonic* movements (convulsions). Cyanosis is produced by temporary arrest of respiration. Absence of swallowing causes saliva to collect in the mouth, and typical frothing occurs when breathing is resumed. The tongue may be bitten if a gag is not inserted promptly, and urine is passed if the bladder is full at the onset of the attack. After the convulsions have ceased, the patient may be in a confused state and may perform unwilled complicated acts (*post-epileptic automatism*), or sleep may supervene. If grand mal attacks follow each other in rapid succession without restoration of consciousness, the patient is described as being in *status epilepticus*. The electroencephalogram (E.E.G.) in grand mal attacks shows fast, approximately 20 per second, high voltage spikes. The normal resting brain, with the patient's eyes closed, has a relatively low voltage rhythm of 10 per second, known as alpha rhythm (Fig. 370).

**Jacksonian Epilepsy.** In this type of seizure, the focus of discharge is somewhere in the cerebral cortex, and, if the disturbance spreads, the rate of spread is slow enough to be observed clinically. Thus a focus of discharge in the facial area of the motor cortex on one side causes clonic movements of the opposite side of the face. The attack may then spread on the same side to involve successively the arm, the leg and perhaps finally the whole of the body. At this point the patient loses consciousness. If the discharge arises in the post-central cortex, the attack begins with tingling somewhere on the opposite side of the body. The brain of patients with Jacksonian epilepsy is more resistant to the spread of the discharge than that of patients with grand mal. The degree of resistance determines the extent of spread of attacks. The essential point about Jacksonian epilepsy is that the march of events is slow enough for the



## OCCLUSION OF THE INTERNAL CAROTID ARTERY

*Recurrent* hemiparesis, transient homolateral blindness and, on the dominant side, dysphasia, may occur as the result of partial or complete occlusion of the internal carotid artery. The causes are : (a) Atherosclerosis (atheroma) occluding the vessel, either in the neck just above the bifurcation of the common carotid artery or on the intracranial course of the artery ; (b) post-traumatic thrombosis following either open or closed cervical injuries.

Eventually death or permanent hemiplegia may occur from cerebral ischæmia. Therefore the following surgical procedures have been tried in *cervical* occlusion of the internal carotid artery : (a) Removal of the atheromatous plaque through an incision in the artery which is temporarily occluded by arterial clamps until the incision is sutured (Oliver) ; (b) excision of the occluded segment of the artery followed by anastomosis or arterial grafting (Rob).

suppressed by *tridione*, but may be increased by *phenobarbitone*. In some cases, the number of attacks is reduced by *caffeine* or *amphetamine*. There is electroencephalographic evidence that the focus of origin of these attacks is in the cerebral ganglia or nuclear structures of the brain stem, although no gross pathology has been discovered. These attacks, therefore, concern the neurosurgeon only in regard to differential diagnosis. *Pyknolepsy* refers to very frequent attacks of loss or clouding of consciousness; it is really a particularly severe type of juvenile petit mal and therefore there is no point in retaining this term.

**B. Small Grand Mal.** These seizures may occur at any age and from any focus, including the temporal lobe. Although there are no convulsions, the E.E.G. registers the same electrical changes as seen in grand mal. These attacks respond to the same drugs as used for grand mal (*phenobarbitone* and *hydantoins*).

The susceptibility of the brain to epilepsy depends on many factors, such as heredity, age (the young brain is more sensitive than the old), state of activity of the brain (the active brain is less sensitive than the inactive one; indeed temporal lobe epilepsy is often brought on by sleep), state of hydration (increased hydration may precipitate an attack), level of the blood sugar (hypoglycæmia may bring on attacks), and degree of oxygenation of the blood (anoxia may cause fits, e.g. as with the sudden fall of blood pressure in Stokes-Adams heart-block attacks and the carotid sinus syndrome). Some drugs known as convulsants (e.g. leptazol and picrotoxin), if given in sufficient doses, cause epileptic fits.

Some of the factors which influence the state of the brain and which have been cited above are variable. It is therefore not surprising that the seizures of a given patient may vary from time to time. When the brain is in its most receptive state a grand mal attack occurs, whereas if the brain is in a resistant state a Jacksonian attack is more likely.

**Todd's Paralysis.** After an epileptic fit transitory paresis (weakness) of a limb or limbs may be found. This sign is of localising value, but does not necessarily indicate tangible pathology. If paresis persists and still more if it increases, a recognisable lesion, especially a tumour, is likely to be the cause of the attacks.

### INVESTIGATIONS

All fits require careful investigation, for some are amenable to surgery, and even when full investigation is negative, diligent follow-up must be arranged as epilepsy may precede, sometimes by years, the more obvious signs of a tumour.

The term *idiopathic epilepsy* is frequently applied to attacks in which no tangible pathology can be discovered, and patients with this label tend to be neglected. They should therefore be periodically re-investigated.

The neurosurgeon first endeavours to discover the site of origin of fits

point of origin of the attacks to be revealed. It is somewhat misleading to refer to Jacksonian epilepsy as focal, for all epileptic attacks begin in a focus somewhere. During Jacksonian attacks, the E.E.G. shows the same type of spiking as in grand mal, but the origin of the discharge may be shown.

**Temporal Lobe Epilepsy** (Syn. "Uncinate" Epilepsy or Psychomotor Epilepsy). About a quarter of all cases of epilepsy have their focus of discharge in one or other temporal lobe. Again, attacks may spread so quickly that grand mal results. More often, however, the discharge is localised to the temporal lobe, or it extends slowly enough for the aura to be identified. There are several modes of origin of temporal lobe epilepsy, and each reflects one of the functions of this versatile lobe. The commonest aura is a sensation in the abdomen, chest or throat (visceral aura). The classical attack described by Hughlings Jackson begins with a sensation of a bad smell or taste accompanied by smacking of the lips and a "dreamy state." The origin of such attacks is in the uncus, hippocampus or amygdala, the location of olfactory and gustatory function. Other patients may complain of unpleasant noises or a disturbance of equilibrium, sometimes so severe that the patient is thrown to the ground (*gyratory epilepsy*). These attacks are related to the auditory and equilibratory functions of the superior temporal convolution. Some patients experience sudden fear, anger or pleasure. Included in the "dreamy state" of Hughlings Jackson is the sudden feeling of familiarity with the surroundings. Sometimes objects seem small and receding (*micropsia*) or large and near (*macropsia*). Sounds may seem unduly loud or soft. The patient may feel that what he is witnessing has taken place before (*déjà vu* phenomenon). A vivid recollection of the past with its associated emotions may force its way into consciousness. Patients may carry out complicated acts unrelated to present events, but sometimes related to the sensory phenomenon, and tending to be aggressive (not to be confused with post-epileptic automatism). *Depersonalisation* (a feeling of complete detachment from self), and sometimes the strange related phenomena of *autoscopy* (seeing an image of oneself in the external environment), are sometimes experienced. These latter phenomena of Jacksonian "dreamy states" are related to the highest functions of the temporal lobe. Many patients with temporal lobe epilepsy have an associated personality defect or may be suffering from a psychosis. The personality tends to decline still more when fits are brought under control by drugs.

**Petit Mal** (Syn. Minor Epilepsy). Short attacks of loss or clouding of consciousness without convulsions are known as petit mal. The term is a clinical one comprising two different types of epilepsy.

A. *Juvenile petit mal*, occurring usually in children and characterised by the great frequency of the attacks (sometimes as many as a hundred a day), and a unique E.E.G. (3 per second alternating spikes and waves—Fig. 370). Attacks tend to abate as adult life is reached. They are often

**Air Encephalography** (also Chap. 53). Sulci, cisterns and ventricles can be visualised radiologically by replacement of cerebrospinal fluid with air injected by lumbar or cisternal puncture. The amount of fluid replaced varies according to the requirements and the volume of the fluid spaces, but 60 to 100 ml. should give good outlines in most cases. The majority of epileptics without neurological signs have normal air encephalograms, although from time to time an area of cortical atrophy or a cerebral tumour may be revealed. Patients with neurological signs are more likely to have abnormal air encephalograms. This investigation should not be carried out if there is evidence of raised intracranial pressure, as dangerous and sometimes fatal coning of the cerebellum may occur. Instead, *ventriculography* (replacement of ventricular fluid with air by means of a brain cannula passed through parietal burr-holes—see also Chap. 53) should be carried out, but only if facilities are available for immediate operation, as patients with space-occupying lesions tend to deteriorate also after ventriculography. The radiological appearances in the various epileptogenic diseases are described later.

**Angiography** (also Chap. 53). Carotid or vertebral angiography may show the outline of a vascular malformation or tumour. Tumours may reveal themselves also by displacement of cerebral vessels.

### SYMPTOMATIC TREATMENT

All patients suffering from persistent epileptic attacks need anti-convulsant drugs whatever the causal lesion, for the attacks are a danger to the patients and to others. All patients suffering from epilepsy, except juvenile petit mal, may sooner or later pass into *status epilepticus* which, irrespective of the pathology, may end fatally. Adequate anticonvulsant therapy helps to prevent this dreaded condition. *Hydantoins* (e.g. Epanutin) act on the discharging focus, whereas *phenobarbitone* increases the resistance of the brain to the spread of the discharge. It is customary, therefore, to give phenobarbitone and hydantoins together for grand mal, small grand mal and Jacksonian epilepsy. If patients taking both these drugs together omit phenobarbitone, there is the danger that if an attack occurs it will be particularly violent and may develop into *status epilepticus*. Temporal lobe epilepsy is treated with hydantoins alone, for barbitone has a strong tendency to increase the frequency of the attacks in this type of epilepsy. Neither of the above drugs should be given for juvenile petit mal. *Tridione* is the specific drug for this disorder. *Primidone* (Mysoline) may be successful in any form of epilepsy and is given when other drugs fail.

### DOSAGE OF ANTICONVULSANTS

*Phenobarbitone*. Adult dose begins with tablets of 30 mg. ( $\frac{1}{2}$  gr.) t.d.s.

*Sodium diphenyl hydantoinate* (phenytoin sodium, B.P., Epanutin, proprietary name). Adult dose begins with capsules of 0.1 gm. ( $1\frac{1}{2}$  gr.) b.d.

and then to ascertain the underlying pathology. A careful history is taken from the patient and from witnesses. Particular attention is paid to the mode of onset of seizures. A complete clinical examination must be carried out although in the majority of cases *no* physical signs are elicited between attacks.

**Electroencephalography (E.E.G.).** When electrodes are applied to the scalp, electrical waves arising in the cerebrum can be amplified and recorded. When the eyes are closed normal waves are of relatively low voltage and have a rate of about 10 per second (alpha rhythm, originally known as Berger rhythm). In grand mal or small grand mal attacks, fast 20 per second high voltage waves or spikes are recorded from both cerebral hemispheres (Fig. 370).

During Jacksonian seizures, localised spiking may be found. Between attacks of epilepsy, the rhythm is often normal, especially in grand mal and small grand mal, but sometimes larval (sub-clinical) episodes may be recorded. In juvenile petit mal, the characteristic spike and wave complexes are recorded (Fig. 370). They are synchronous in all leads of both hemispheres suggesting that such fits originate in the basal ganglia or brain stem. In temporal lobe epilepsy it is necessary to employ sphenoid and midline electrodes (chin, nasopharyngeal and nasal) to pick up spiking from the hippocampal region. Sometimes the dysrhythmia can be recorded only on overbreathing or during natural or induced sleep (e.g. after administration of intravenous sodium thiopentone, or quinalbarbitone by mouth). Light general anaesthesia also tends to accentuate the dysrhythmia. Sometimes flat-topped waves are recorded instead of spikes. In patients with juvenile petit mal there is a good chance of recording the characteristic rhythm between attacks. Sometimes localised abnormal non-epileptic waves may indicate the site of the pathological process. Although tumours themselves are electrically inactive, slow high voltage delta waves (3 per second) frequently occur adjacent to tumours. It is frequently necessary to repeat E.E.G. recordings before a conclusion can be reached. *A normal E.E.G. does not exclude epilepsy.*

**Radiography.** Plain X-rays of the skull are taken as a routine. Separation of the sutures (diastasis), marked convolutional impressions (beaten-silver sign), and rarefaction or erosion of the posterior clinoid processes are signs of raised intracranial pressure. Failure of growth of the calvarium on one side with neurological signs on the opposite side suggests atrophy of the underlying hemisphere, whereas expansion of the calvarium on one side with neurological signs on the opposite side suggests a space-occupying cyst or tumour. A basal view may show a small middle fossa on the affected side in temporal lobe epilepsy. Excessive vascular markings may be associated with a subjacent meningioma. Intracranial calcification may indicate the presence of a tumour or vascular malformation.

of one kind or another together with associated changes in personality. Mental development may be retarded. Plain radiography may show the calvarium to be small on the affected side (the side opposite to the hemiplegia). The E.E.G. may show epileptic rhythms and air-encephalography may reveal a greatly enlarged lateral ventricle and convolutional atrophy. Hemispherectomy (devised by Krynauw) on the affected side relieves the epilepsy and improves the personality without increasing the hemiplegia or affecting speech (the speech centres are always in the normal hemisphere). A complete homonymous hemianopia, including half of each macular field, is produced.

(3) *Infantile Porencephaly.* Air-encephalography in a child having seizures may reveal a large cavity in the cerebrum in connection with one of the lateral ventricles. The cause is unknown, but the importance of these lesions is that the brain around them may be abnormal and give rise to epilepsy. Excision of the epileptic focus is indicated if the fits cannot be controlled by drugs.

**B. Traumatic Epilepsy.** After open head injuries in which penetration of the dura mater has occurred, approximately half the patients develop epilepsy. This type of injury is particularly common in warfare. After closed head injuries the incidence of epilepsy is about 2 to 5 per cent. Intractable attacks should be investigated along the lines already described, and epileptic foci if discovered and if accessible, should be excised. Dense scars adherent to the dura mater are sometimes found in the open head injury group (Fig. 393). The presence of sepsis in the wounds does not affect the incidence of persistent epilepsy. In many cases of post-traumatic epilepsy it may be impossible to demonstrate the focus.

**C. Cerebral Tumours.** The great majority of patients suffering from epilepsy do not have cerebral tumours, but patients suffering from cerebral tumours frequently present with epilepsy. Most patients with cerebral tumours presenting with epilepsy have gliomas, for these are the commonest tumours; but, nevertheless, meningiomas, because they affect the cerebral cortex, are very likely to cause epilepsy. Grand mal or Jacksonian epilepsy may be caused by cerebral tumours. The latter type of seizure indicates the exact site of the lesion. Treatment is carried out as described under intracranial tumours. Anticonvulsants are given after operation for at least a year, or indefinitely if the attacks are not relieved by removal of the tumour.

**D. Cerebral Abscess.** Epilepsy may follow at any time after successful treatment of a cerebral abscess. If drugs fail and the focus of origin of the attacks is accessible it should be excised.

**E. Vascular Malformation.** Tangles of large arteries and veins cause degeneration of cortical neurons and may thus give rise to epilepsy. Removal of the lesion and some of the affected brain may have to be considered if drugs do not control the attacks. Vascular malformations in

*Tridione* may be given in tablets. The doses are 0.1 gm. t.d.s. for infants, 0.2 gm. t.d.s. between two and four years, 0.3 gm. t.d.s. for children over five years, and 0.4 gm. t.d.s. for adults. This drug is also supplied in capsules containing 0.3 gm.

*Primidone (Mysoline)*. Tablets contain 0.25 gm. of the drug. The adult dose begins with 0.125 gm. (half a tablet) given once late in the evening for a week, as patients tend to be sleepy on this drug for the first few doses. The dose is then gradually increased as required, at weekly intervals, up to a maximum of 1.0 gm. total daily dosage. Patients get too sleepy if this drug is combined with phenobarbitone, although it can be used in combination with the other anticonvulsants.

*Bromides* are no longer used because they cause bromism, and they are the least effective of the anticonvulsants.

Drugs are continued after the removal of operable lesions for one year from the time of the last seizure, but, if seizures continue, drugs are given indefinitely.

#### EPILEPTOGENIC DISEASES AMENABLE TO SURGERY

A. *Cerebral Atrophy*. Methods of investigation previously described may lead to the demonstration of macroscopic atrophy of some part of the cerebral cortex, or the E.E.G. may show an epileptic focus which microscopically can be shown to be a zone of degeneration. There are several well-known examples of cerebral atrophy which cause epilepsy.

(1) *Temporal Lobe Epilepsy*. The pattern of the attacks has already been described. The commonest cause of epilepsy arising in this lobe is micro- or macroscopic atrophy affecting especially the hippocampus, uncus and amygdala. Clinical examination is usually negative. The diagnosis is based on a carefully taken history and repeated E.E.G. examinations including the use of sphenoid and midline electrodes. In some cases radiography (basal view) shows the temporal fossa to be small on the affected side. Air-encephalography may demonstrate enlargement of the temporal horn of the lateral ventricle. Temporal lobe epilepsy is difficult to control with drugs so that partial temporal lobectomy may be required (Penfield). It is performed through the standard temporal approach (Chap. 54). The lobe is removed as far as 6 cm. back from the anterior pole. The only neurological effect is an homonymous upper quadrantic visual field defect. Sometimes temporary dysphasia occurs following operations on the dominant side. Tumours (gliomas or meningiomas) and vascular malformations account for some cases. The majority of the latter lesions are demonstrated by angiography, but others are unexpectedly discovered during temporal lobectomy for suspected atrophy.

(2) *Infantile Hemiplegia*. The commonest cause of this syndrome is atrophy of part or all of one hemisphere, sometimes occurring after an attack of measles. Many of these children suffer from epileptic attacks

anxiety states in man might be relieved by procedures directed at the frontal lobes, and in the same year persuaded his surgical colleague Almeida Lima first to inject alcohol into the frontal lobes, and later, using a special instrument, to cut cores out of the frontal white matter. Walter Freeman (neurologist) and James Watts (neurosurgeon), of Washington, D.C., were impressed with the results, and quickly followed in Moniz and Lima's footsteps. Watts, in 1936, carried out his first prefrontal lobotomy (leucotomy), dividing the white matter in the mid-frontal region bilaterally through burr-holes (Fig. 394). Various modifications of the original lobotomy have been devised although the anatomical basis of all of them is the division of prefrontothalamic connections. Prefrontal lobotomy is frequently followed by severe blunting of personality, shown chiefly by apathy, lack of imagination, lack of foresight and absence of social sensitivity. Therefore the present-day tendency is to abandon the original lobotomy in favour of more conservative operations such as bilateral *selective undercutting* of the superior prefrontal convexity or the orbital surface of the frontal lobe (W. B. Scoville), *topectomy* (bilateral subtotal ablation of the superior prefrontal convexity—J. Lawrence Pool) and *anterior cingulectomy* (bilateral removal through a unilateral approach of the anterior part of the gyrus cinguli, based partly on the observation of tameness and placidity in monkeys after removal of these gyri—Jacques Le Beau).

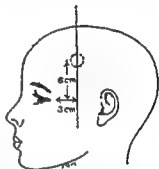


FIG. 394. The Watts' lobotomy or leucotomy. The centre of the incision is 6 cm. upward from the zygoma and 3 cm. posterior to the lateral rim of the orbit.

**Indications.** When all other methods of treatment fail, operation is indicated in the following disorders: Schizophrenia, manic-depressive psychosis, severe anxiety states, and intractable pain caused by incurable disease. In 1947, the Board of Control (England and Wales), reporting on a thousand cases of the original prefrontal leucotomy (lobotomy), recorded a discharge rate from mental institutions of 23 per cent. of schizophrenics and 50 per cent. of manic-depressives. In regard to mental symptoms, depression, agitation and obsession respond well to leucotomy. Intractable pain is not eliminated by leucotomy, but the patient's emotional reaction towards it is damped down and the demand for drugs ceases.

**Complications.** The commonest complication and commonest cause of death is intracerebral hæmorrhage. Persistent epilepsy occurs in 2 per cent. of cases of leucotomy (Board of Control report), and in about 15 per cent. of cases following cortical excisions (Lawrence Pool).



relation to cerebral and subarachnoid hæmorrhage are discussed in Chap. 56.

**Electrocorticography.** This term is used for E.E.G. carried out with electrodes in contact with the exposed cerebral cortex. It is much more sensitive, for the electrical resistance of the skull is eliminated. It is used at operation to define the limits of the epileptic focus. Whatever the causal pathology, the cerebral cortex, provided it has no vital function, is removed wherever epileptic waves are recorded. Epileptic discharges

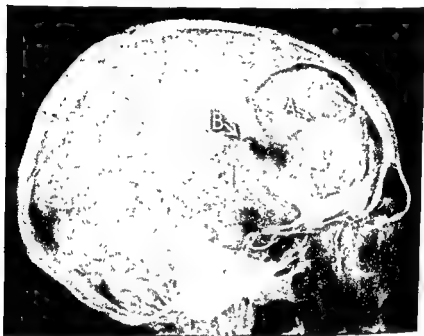


FIG. 303. Air encephalogram showing a diverticulum in the anterior horn of the right lateral ventricle produced by a cerebral scar which followed a penetrating head injury (traumatic porencephaly). A. Diverticulum from right ventricle. B. Anterior horn of the normal left ventricle. A roughly circular frontal skull defect, produced by removal of septic bone fragments, is also shown. The patient suffered from intractable *grand mal* which was relieved by excision of the scar and a wide margin of surrounding cortex.

do not arise from tumours or cerebral scars, but from compressed or degenerating cortex adjacent to these lesions.

Electrical stimulation of the cerebral cortex may reproduce the aura of seizures and is therefore also employed to define epileptic foci.

#### MENTAL DISEASES

Egas Moniz, of Lisbon, had for years pondered over the possibility of treating mental diseases by surgical means, when in 1935 he heard John Fulton, the eminent physiologist, describe the effects of *bilateral* frontal lobectomy in two chimpanzees, carried out during the course of investigations into the function of the frontal lobes. After operation, the animals had become devoid of emotional expression. Moniz at once realised that

or other frontal sinus are often seen in radiographs of the skull. Occasionally this type of osteoma grows very large, expanding and obliterating the sinus and causing an unsightly swelling of the forehead. It sometimes arises in one of the ethmoid sinuses, producing nasal obstruction and unilateral exophthalmos. These large osteomas of the sinuses are peculiar for they usually have no bony attachment to the skull. Whenever possible, removal of osteomas arising in the nasal sinuses should be avoided, as there is the risk of a cerebrospinal fluid fistula occurring, and reconstruction of the skull defect in a potentially septic field is likely to fail. Osteomas arising in the external auditory meatus are removed only when they cause deafness, or as part of radical mastoidectomy.

**Hæmangioma.** This tumour is usually *hard*, and therefore cannot be distinguished *clinically* from an osteoma. There is usually no bruit on auscultation, and visible pulsation is rare. Radiography shows a circumscribed rarefied area within which there is a honeycomb formation. In profile, there are fine spicules of bone at right-angles to the surface of the skull (sun-ray effect) suggesting a sarcoma. Histologically, these tumours are capillary or cavernous hæmangiomas supported by bone. The indications for removal are the same as for osteoma.

**Giant-cell Tumour (Syn. Osteoclastoma).** This tumour is excessively rare in the skull, and is restricted to bone formed in cartilage (Fig. 396). It has been found more often in the mandible than elsewhere in the skull. Clinically, it has the consistency of rubber, and cannot be distinguished from other non-osseous tumours of the skull. Radiography shows a sharply circumscribed area of absorption of bone. Histologically typical osteoclastic giant cells are observed among spindle and round cells. Very occasionally the tumour is malignant. Giant-cell tumours are completely removed and radiotherapy is given post-operatively.

**Eosinophilic Granuloma.** This tumour affects children and young adults, and is sometimes multiple. In the skull the vault only is affected. The tumour is soft or rubbery in consistence. At first there may be local pain and tenderness. In many cases examination of the blood shows an eosinophilia ranging from 4 to 10 per cent. Radiography demonstrates an area of absorption with an irregular outline and no surrounding bony reaction (Fig. 397). Microscopy shows masses of large foamy phagocytes (histiocytes), fibroblasts, eosinophils and eosinophilic myelocytes resem-

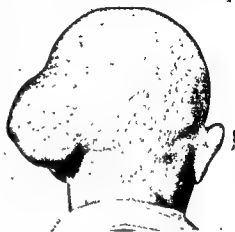


FIG. 396. A large osteoclastoma arising from the occipital bone. This one proved to be malignant.

## CHAPTER 58

### TUMOURS OF THE SKULL

#### PRIMARY TUMOURS

ALL types of primary tumours of the skull are rare—except in examinations!

**Osteoma.** This is the most frequently occurring primary tumour of the skull. The history of the tumour often dates back to childhood. It may be composed of cortical bone with a cancellous interior (*cancellous* or *spongy osteoma*) or entirely of dense bone (*ivory osteoma*). Osteomas may be clinically indistinguishable from the dense bony reaction caused by some meningiomas, but the cancellous type can be diagnosed with confidence by radiography (Fig. 395). Small osteomas projecting into one



(a)



(b)

FIG. 395. Osteoma of the left frontal region. (a) Radiograph showing the tumour to be a mixed spongy and ivory osteoma. (b) The tumour after removal.

circular punched-out area of erosion with a densely sclerosed margin—characteristic of this tumour (Fig. 398). After removal of an epidermoid, the cavity is treated with Zenker's fluid\* to destroy any remaining capsule. Sometimes an epidermoid grows in the petrous part of the temporal bone causing slowly progressive facial paralysis and deafness. Recovery of the seventh and eighth cranial nerves may not follow removal of the tumour. Facio-hypoglossal or facio-accessory anastomosis may then be required.

**Chondroma and Chondrosarcoma.** These tumours arise from bone formed in cartilage, i.e. the base of the skull. They may project into the middle or posterior fossæ or both. Microscopically, nodules of typical and atypical cartilage are found in a myxomatous stroma which may be focally calcified or ossified. The malignant variety is more likely to cause neurological effects. Chondromas very occasionally arise in the vault of the skull despite its membranous origin.

**Osteogenic Sarcoma.** This tumour very rarely occurs in the skull. It is characterised by its rapid growth and invasion of soft tissues. There is destruction of the two tables of the calvarium, but there is new formation of bone in the periosteum. Osteogenic sarcoma may occur in *Paget's disease* of the skull (osteitis deformans). *Periosteal fibrosarcoma* of the skull has been described. Patients with sarcomas usually die from metastasis. There is no effective treatment.

**Chordoma.** Notochord remnants may give rise to chordoma of the basisphenoid or basioccipital bones. Alveolar masses of large physaliphorous† cells of typical foamy appearance due to mucin are observed microscopically; there is also abundant extracellular mucin. These tumours are locally invasive and cause pressure on the pons and associated cranial nerves. The pituitary gland may be destroyed. Eventually they cause increased intracranial pressure. They are inaccessible to the surgeon.

## TUMOURS OF THE SKULL SECONDARY TO INTRACRANIAL TUMOURS

**Meningiomas.** These tumours arise from arachnoid cells in the dura mater to which they are almost always attached. They often have no effect on the overlying bone, but sometimes they cause an increase in the thickness of the bone, and the resultant "tumour" of the skull may be clinically and radiologically indistinguishable from the ivory type of osteoma. Neurological manifestations, and large or adventitious vascular channels shown in radiographs, indicate the presence of a subjacent meningioma. This increased bone formation caused by some meningiomas may occur with or without invasion of the bone by tumour

* Zenker's fluid :	Potassium bichromate	.	.	2.5 gm.
	Sodium sulphate	.	.	1.0 gm.
	Corrosive sublimate	.	.	5.0 gm.
	Water	.	.	100 ml.

† Physaliphorous = having globular cavities.



FIG. 397. Radiograph of a solitary eosinophilic granuloma in a girl age twenty-one years. Note the punched-out appearance and the lack of surrounding bony reaction.

heterotopia). It is composed of a capsule with an outer layer of dense acellular connective tissue having a pearly appearance and an inner layer of squamous epithelial cells *without dermal appendages* (i.e. hair follicles and sweat glands). The capsule encloses a mass of horny epithelial debris and cholesterol crystals. Clinically the tumour has a rubbery consistency. Radiography shows a roughly

bling the appearance seen in the deposits of Hand-Schuller-Christian disease. However, eosinophilic granuloma is benign and is cured by excision or radiotherapy.

**Epidermoid Tumour.** The alternative names are *tumeur perlée* of Cruveilhier and cholesteatoma (the latter is a bad name, for confusion with the inflammatory type of cholesteatoma occurring in otitis media and mastoiditis is possible). The tumour is thought to arise in epithelial cell rests (developmental

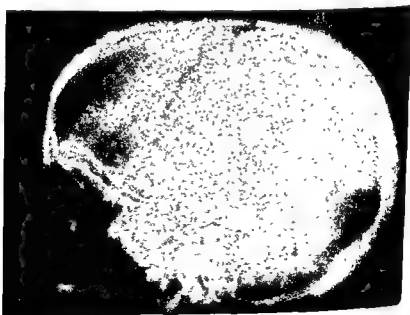


FIG. 398. Radiograph of an epidermoid tumour. Note the circular outline and ring of sclerosed bone around the tumour.

diseases affect the skull. Thus *myelomatosis* causes numerous deposits in the skull; in the radiograph many circular punched-out areas are seen (Fig. 400). In *Hand-Schüller-Christian* disease there are both punched-out areas and irregular patches of rarefaction giving the appearance of a map. In this disease, the deposits of cholesterol and its esters in the *pericranium* cause secondary erosion of the bone. The *leukemias* may also affect the skull.

### DIAGNOSIS

From the foregoing description of the various tumours of the skull it is evident that clinical examination of tumours, when accessible, seldom enables a correct diagnosis of their nature to be made. A bony hard tumour may be an *osteoma*, *hæmangioma*, or a bony reaction to an underlying *meningioma*, whereas a tumour of rubbery consistency may be a *giant-cell tumour*, an *eosinophilic granuloma*, an *epidermoid*, or a *secondary tumour*. Radiography helps to make the diagnosis in many cases, but biopsy may be necessary before a diagnosis can be made with certainty.

Other conditions which have to be considered in the diagnosis of solitary tumours of the skull are: (a) *Brodie's abscess* (chronic osteomyelitis); (b) *Pott's puffy tumour* (Chap. 51); (c) *traumatic skull defect* containing granulation tissue and blood pigment; and (d) *osteitis fibrosa cystica* which may affect the membranous bones of the skull without affecting the rest of the skeleton. *Cephalhæmatoma*, a collection of blood under the pericardium, occurring soon after birth in a new-born infant, is not likely to be confused with a neoplasm (*vide* Chap. 50).



FIG. 390. A hard tumour in the left frontal region producing no neurological signs. Biopsy showed it to be a meningioma.

ulcers, epitheliomas or sarcomas of the scalp, carcinomas of the antrum, nasopharyngeal tumours (Chap. 52), glomus jugulare tumours (Chap. 52), or carcinomas of the middle ear.

#### TUMOURS OF THE SKULL SECONDARY TO DISTANT PRIMARY TUMOURS

Primary tumours of the bronchus, breast, stomach, thyroid gland, kidney or suprarenal gland may give rise to single or multiple secondaries in the skull.

Various systemic

cells. Some meningiomas invade and destroy the bone and thus cause an external swelling of the skull resembling other non-osseous tumours. Such tumours can be neurologically silent (Fig. 399).

**Secondary carcinoma** of the dura mater, e.g. from the breast or bronchus, may grow through the bone to form a swelling of variable consistency on the surface of the skull. It is a very rare type of tumour. Neurological signs are likely to be present.

**Pituitary tumours** (chromophobe adenomas or the rare adenocarcinoma) very occasionally invade and destroy the base of the skull and may cause cranial nerve palsies.

#### TUMOURS OF THE SKULL SECONDARY TO EXTRACRANIAL TUMOURS

The skull may be invaded directly from the exterior by rodent



FIG. 400. Myelomatosis of the skull. (Radiograph by Dr. J. M. Stewart.)

sneezing, compression of the jugular veins in Queckenstedt's test, or lumbar puncture). Lower neuron effects corresponding with the level of the lesion may accompany the long tract manifestations below. The maximum bilateral long tract signs comprise spastic paralysis (paraplegia or quadriplegia), positive Babinski responses, increased tendon jerks,

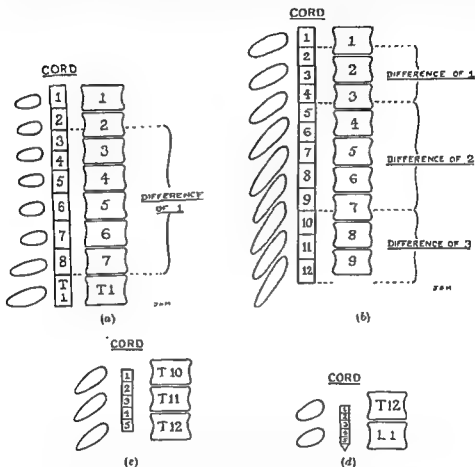


FIG. 402. Relationships between the segments of the spinal cord and the vertebral bodies: (a) The cervical segments. (b) The thoracic segments (here the tip of a spine is opposite the body of the vertebra below). (c) The lumbar segments. (d) The sacral segments. The lower end of the spinal cord terminates opposite the lower border of the first lumbar vertebra.

absent abdominal reflexes, and loss of all modalities of sensation up to the level corresponding with the site of the lesion (Fig. 401). The difference between the levels of the spinal cord segments and the vertebrae is shown in Fig. 402. The signs of compression are usually less than those given above. Sometimes there is a Brown-Séquard syndrome of "hemi-section" of the spinal cord (homolateral pyramidal paralysis and posterior column loss, with contralateral analgesia and thermæsthesia). With space-occupying lesions the Brown-Séquard syndrome is not usually pure,



## CHAPTER 59

### THE SPINAL CORD AND ITS NERVE ROOTS

**The Compression Syndrome.** The pattern of neurological disturbance varies with the level, conformation and pathology of the lesion. Unilateral or bilateral root pains with hyperalgesia may occur at the level of the lesion. In the thoracic and abdominal regions they have a characteristic girdle distribution. Root pains tend to be aggravated or brought on by mechanisms which alter the cerebrospinal fluid pressure (e.g. coughing,

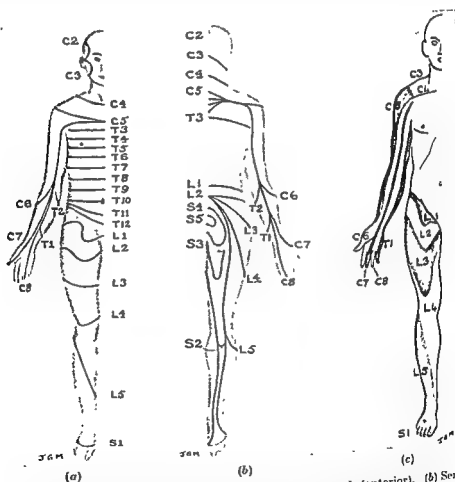


FIG. 401. Sensory levels and dermatomes : (a) Sensory levels (anterior). (b) Sensory levels (posterior). (c) Dermatomes. (There is apt to be confusion between sensory levels and the lines indicating the boundaries of the dermatomes ; a sensory level indicates the upper limit of sensory loss found on the skin with a transverse lesion at a given level in the spinal cord or cauda equina ; the outline of a dermatome on the skin indicates the area covered by one given sensory nerve root. The overlap of dermatomes is extensive. The neurosurgeon is particularly concerned with sensory levels in the diagnosis of spinal cord tumours and with dermatomes in the diagnosis of intervertebral disc protrusions.

necessary aspiration is attempted to demonstrate whether or not the needle has entered the cisterna magna, which in adults is normally 4 to 5 cm. from the skin surface. Sometimes the needle strikes the posterior margin of the foramen magnum. When this happens, a slight change of direction of the needle caudally is necessary.



FIG. 403. Myelogram demonstrating an extramedullary tumour (in this case a neuro-fibroma) opposite the sixth and seventh cervical vertebrae. The Myodil was injected by lumbar puncture. The curved lower border of the tumour is shown on the right (reader's left) of the spinal canal and some Myodil has settled on the upper end of the tumour. The left (reader's right) half of the Myodil column is less dense because of the presence of the spinal cord which is therefore separate from the tumour and has been displaced by it.

#### SYNDROMES OF THE VARIOUS LEVELS

*The Foramen Magnum Syndrome.* Tumours which arise in, or immediately above or below the foramen magnum and extend through it are uncommon. They may occur at any age, and although anatomically dangerous, are more often benign than malignant. *Meningiomas* are the most frequently encountered tumours in this situation. They tend to be missed in the early stages of their growth because the manifestations are vague, and the foramen magnum is a no man's land between ventriculography and myelography. A tumour in this situation should be considered when there is an unexplained increase in cerebrospinal fluid protein. Well-advanced tumours produce neurological effects which may be grouped as follows:—(a) *Long tract disturbances* (paresis of all limbs, paræsthesiæ and sensory loss, with exaggerated tendon jerks and positive

but is accompanied by some evidence of involvement of the other half of the spinal cord. The sensory level, which is of great localising significance, is the level at which *diminished* sensation changes to normal sensation (not the upper limit of *absent* sensation). Occasionally there is no sensory loss (thus sometimes patients who eventually have benign spinal tumours removed have previously been thought to have disseminated sclerosis!). Retention of urine with overflow and constipation occur in compression of the spinal cord or the cauda equina.

*Lumbar puncture* in the presence of spinal cord compression shows a normal or subnormal cerebrospinal fluid pressure. The oscillations of pressure caused by the pulse and respiration are frequently absent. Pressure on the jugular veins (Queckenstedt's test) produces no rise in pressure when there is a complete subarachnoid block. Either a slow rise and fall of pressure, or a normal rise followed by a slow fall, indicates an *incomplete* block. An increase in protein in the fluid is, however, a more delicate indication of spinal subarachnoid obstruction (normal protein up to 40 mg. per 100 ml.). The fluid caudal to a block is frequently yellow; it has an increased protein content, and may coagulate spontaneously due to the presence of fibrinogen. These phenomena constitute Froin's syndrome. There may be some increase in the amount of protein immediately above the block. A normal response to Queckenstedt's test and a normal protein level do not necessarily exclude a space-occupying lesion. Tests for syphilis are carried out as a routine on the blood and cerebrospinal fluid.

*Radiography* of the spinal column is carried out at the level corresponding with the neurological signs (Figs. 401 and 402). Often there is no radiological abnormality, but sometimes there is evidence of disease of the spinal column, or there are changes in the bone produced by tumours arising from the dura mater, the spinal cord or the nerve roots. The radiological appearances of the various lesions affecting the spinal cord and the nerve roots are described later.

*Myelography* is carried out before operations on space-occupying lesions in the spinal canal. Myodil (3 to 6 ml.) is injected into the subarachnoid space by lumbar or cisternal puncture; lumbar puncture is employed for lower thoracic and lumbar lesions, whereas cisternal puncture is preferred for upper thoracic and most cervical lesions. Negative myelography does not exclude a space-occupying lesion. This is particularly true of disc protrusions which may be situated lateral to the theca. Myelography demonstrates the exact site of a block and may also distinguish between intra- and extramedullary tumours (Fig. 403).

*Cisternal Puncture.* This is performed under infiltration anaesthesia with the patient's head flexed on the extended neck. A thin lumbar puncture needle is introduced in the midline just cephalad to the spine of the axis and directed upward towards the foramen magnum. Whenever the needle is felt to pierce any tissue plane, the stylet is withdrawn, and if

the third, fourth and fifth cervical segments or nerve roots and consequent paralysis of the diaphragm (confirmed by fluoroscopy). The patient may have to be placed in a respirator. Tumours in the upper cervical region may cause lower neuron signs in the arm. This is probably due to interference with the blood supply of the lower cervical segments of the spinal cord by compression of the anterior spinal artery above. Long tract motor and sensory signs are found below the level of the lesion.

*The Lower Cervical Syndrome.* Radiating pain, sensory loss, lower neuron paralysis and depressed or absent tendon jerks affect one or both upper limbs. At first patients may be thought to have *angina pectoris*. Horner's syndrome is sometimes observed on the side of the lesion (Fig. 404). Percussion of the radial styloid process with a jerk-hammer sometimes causes flexion of the fingers instead of the normal contraction of the brachio-radialis. There may be reversal of the triceps jerk, i.e. flexion of the elbow is produced instead of extension. Long tract motor and sensory effects are found below the level of the lesion.

*The Upper Thoracic Syndrome.* Compression at the level of the first thoracic segment of the spinal cord produces atrophy and paralysis of the intrinsic muscles of the hand and sensory loss along the ulnar border of the upper extremity, together with long tract signs. Again, the radiation of pain may cause confusion with *angina pectoris*. Interruption of the first thoracic segment or nerve root produces Horner's syndrome (Fig. 404).

*The Middle Thoracic Syndrome.* Root pains give rise to confusion with disease in the chest or upper abdomen. Lower motor neuron signs are not usually demonstrable at this level, but a band of hyperalgesia is commonly found. Signs of involvement of the spinal motor and sensory tracts are found below.

*The Lower Thoracic Syndrome.* Root pain may suggest the presence of disease in the lower half of the abdomen. The lower sensory level is the only other essential difference between this syndrome and that of the middle thoracic level.

*The Lumbar Syndrome.* Root pain may be felt in the groin, the front of the thigh and sometimes in the bladder and rectum. Lower motor neuron paralysis and wasting affects the muscles supplied by the lumbar nerves (adductors of the hip, L3; quadriceps, L4; and hamstrings, L5); and upper motor neuron paralysis affects muscles supplied by the sacral segments (calf muscles, S1; and peronei and anterior tibial muscle, S2). There are constipation and retention of urine with overflow (false incontinence). The abdominal reflexes are unaffected. Babinski's sign is positive. The knee jerks are absent whereas the ankle jerks are exaggerated.

*The Syndrome of the Sacral Segments and Cauda Equina.* Root pains occur in the leg. Intramedullary lesions first cause lower motor neuron paralysis of the muscles below the knee (S1 and S2) and the glutei (S1 and S2). There is sensory loss over the "saddle" area, back of the thigh

Babinski reflexes). When pressure affects the cord unilaterally, a Brown-Séquard syndrome may occur. Lumbar puncture is likely to reveal a block. (b) *Cervical Nerve Palsies*.—Thus pain and sensory loss in the distribution of the second cervical nerve and paralysis of the spinal accessory nerve are frequently found. (c) *Cranial Nerve Palsies*.—The vagus, glossopharyngeal, bulbar part of the accessory and the hypoglossal nerves are the first involved. Unilateral or bilateral absence of the *corneal reflex* and *analgesia* of the face is sometimes caused by pressure on the descending tract(s) of the trigeminal nerve(s). (d) *Cerebellar Signs* (incoordination, nystagmus and ataxia). (e) *Bulbar Signs* (dysarthria, dysphagia, Cheyne-Stoke's breathing). (f) *Lower neuron paralysis* of

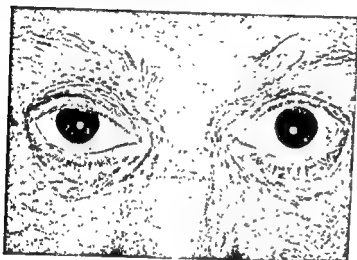


FIG. 404. Horner's syndrome (small pupil and narrowed palpebral fissure) caused by a meningioma at the level of the first thoracic spinal cord segment.

the upper limbs with the usual muscular atrophy. This paradox results from impairment of the blood supply of the lower cervical segments caused by occlusion of the anterior spinal artery. (g) *Hydrocephalic Signs* (headache, vomiting, papilloedema and sometimes diplopia). Any of these groups may occur alone, or there may be any combination of groups. Myelography is carried out by lumbar puncture; cisternal puncture is not indicated when a tumour is suspected at the foramen magnum. When there is raised intracranial pressure the tumour is demonstrated by positive contrast ventriculography (i.e. with Myodil). These tumours are exposed by a posterior fossa craniectomy combined with hemilaminectomy (i.e. laminae are removed only on the side of the tumour in order to maintain the stability of the spine). Tracheotomy may be required post-operatively if coughing and swallowing are absent.

*The Upper Cervical Syndrome.* Root pains radiate to the occipital region or the shoulder. There may be atrophy and paralysis of the trapezius and sternomastoid muscles caused by involvement of the spinal accessory nerve. *Horner's syndrome* (contracted pupil and narrowing of the palpebral fissure) may be caused by pressure on the descending autonomic tract in the spinal cord (Fig. 404). Dyspnoea may result from pressure on

retention of urine in a flaccid bladder. If there is no interference, overflow (false incontinence) follows. In mild injuries normal function may return. When irreversible damage has occurred, involuntary evacuation is eventually established after a variable interval, which is longer in cauda equina than in spinal cord lesions. Patients with cauda equina lesions have the advantage of being able to empty their bladders at convenient times by applying manual pressure to the suprapubic region. Contraction of the detrusor muscle is thus induced by means of reflexes mediated through intrinsic vesical ganglia. On the other hand, the entirely reflex or automatic "spinal bladder" is an intolerable nuisance, for which permanent cystostomy is required. Before cystostomy is performed, the effect of bilateral alcoholic injection or resection of the third sacral nerve roots should be ascertained, for by this means it is possible to convert the reflex "spinal bladder" into the more convenient autonomous "cauda equina bladder." Urinary sepsis, contraction of the bladder, atony of the detrusor, or intercurrent disease may prevent periodic micturition being established. In these circumstances, even patients with a cauda equina lesion are condemned to permanent cystostomy.

**Treatment of Retention.** *Carbachol* (0.25 to 0.5 mg. subcutaneously), a drug of the acetylcholine group, is used for retention which is likely to be transitory, or for relapse after a period of voluntary micturition. It is ineffective in the presence of spinal "shock," i.e. immediately following acute lesions. Use of the drug may avoid catheterisation and, therefore, the risk of urinary infection.

*Non-interference* is appropriate immediately after the onset of acute retention if the level of sensory loss is high enough to prevent appreciation of the pain of vesical distention. It is a means of avoiding catheterisation and therefore the danger of infection while cystostomy is awaited. The bladder is allowed to fill and overflow. The method then has to be abandoned in women unless a female incontinence bag is available, for otherwise the urine cannot be conveniently collected. There is no danger of the bladder rupturing provided manual expression is not attempted.

*Suprapubic aspiration* may be carried out once to relieve the pain of vesical retention if the urine is not infected. Repeated aspiration causes extravasation of urine, and aspiration of infected urine causes cellulitis of the abdominal wall. A thin lumbar puncture needle is employed for the aspiration.

*Manual expression* is mentioned to be condemned, for there is a definite risk of rupture of the bladder. The method aims at stimulating local detrusor reflexes. It is ineffective during the period of "spinal shock."

*Intermittent catheterisation* invariably causes urinary infection however carefully it is carried out. *Continuous perurethral drainage* tends to cause urethritis, and then infection spreads into the bladder. In the female, stretching of the urethra occurs and may need operative correction.

*Low suprapubic cystostomy* is a bad operation, for the opening is directly

and calf, and sole and outer border of the foot. At first there is difficulty in passing urine and later retention with overflow. Constipation is caused by paralysis of the rectum. Some of the worst bed sores are seen with lesions affecting this part of the cord. The plantar reflexes are absent. Priapism and "involuntary" ejaculation sometimes occur. Further expansion of intramedullary lesions of the sacral segments involves the lumbar nerves descending from the lumbar segments of the cord above to their neural foramina between the lumbar vertebrae below (reference to Fig. 402 helps in the understanding of this effect). Thus there is added to the syndrome: paralysis of muscles supplied by the lumbar nerves (adductors of the thigh, quadriceps and hamstrings), and further sensory loss which may extend up to the groin. *Extramedullary* tumours in this situation affect the lumbar nerves from the beginning.

**Extramedullary and Intramedullary Syndromes.** The majority of spinal tumours are extramedullary and benign, and their removal is usually followed by neurological recovery. Some intramedullary tumours are relatively benign (e.g. ependymomas), but their removal gives disappointing results. The spinal cord has a remarkable ability to recover after relief of external compression of slow development, but it has little or no tendency to recover after relief of internal compression. Therefore it would be desirable to be able to distinguish between intra- and extramedullary tumours. This, however, cannot usually be done with certainty. The occurrence of root pains, and displacement of the spinal cord as seen in myelography, favour the diagnosis of an extramedullary tumour. The encircling band of analgesia and thermanæsthesia, without loss of the other modalities of sensation (dissociated sensory loss\*), so characteristic of *syringomyelia*, is sometimes found with intramedullary tumours.

### THE BLADDER

*Slowly progressive lesions of the spinal cord or the cauda equina* cause difficulty in micturition and later retention of urine with overflow. The bladder is then very susceptible to infection, which, when it occurs, is difficult to eradicate. Pyelonephritis may follow and lead to death from renal failure. Therefore the strictest asepsis must be maintained in the treatment of retention of urine. In *chronic vesical retention*, the bladder must be emptied *slowly*. Emptying should take not less than twenty-four hours, otherwise suppression of urine may occur.

*Acute spinal and cauda equina lesions* (e.g. injuries, or collapse of vertebrae resulting from Potts' disease or tumours) cause immediate

\* This band of dissociated sensory loss, caused by interruption of decussating spinothalamic fibres in the centre of the spinal cord, must not be confused with the extensive area of analgesia and thermanæsthesia, without loss of other sensation, below some transverse lesions of the cord causing interruption of the spinothalamic tracts without affecting the posterior columns.

seldom causes dangerous instability of the spine. When laminæ are being removed wet patties (small squares of cottonoid each attached to a long piece of silk suture material) are inserted between the bone and dura mater to prevent bleeding from extradural veins. When these veins bleed they are coagulated with a *weak* diathermy current; a strong current cuts thin-walled vessels and therefore increases the bleeding. When coagulation fails to control *venous* bleeding *Gelfoam* or *gelatin sponge* is applied and a patty placed over it temporarily. The foam or sponge is left in place. Small "stamps" of pounded muscle may be used instead.

When the lesion is extradural (e.g. a protruded lumbar disc) the dura mater is left intact. Otherwise it is steadied by passing a sharp hook into it (not through it) and a small opening is made with a round-ended tenotome. A small wet patty is pushed through the opening to protect the arachnoid while the incision is extended with round-ended scissors. The margins of the incised dura mater are held apart by traction sutures.

Localisation is usually accurate, but if a space-occupying lesion is not immediately exposed, a fine rubber catheter is passed first upwards and then downwards in the *subdural* space in search of a block, and the laminectomy extended as required. Absence of pulsation of the dura and arachnoid suggests the presence of a space-occupying lesion at or above the region exposed, whereas the presence of pulsation suggests that the lesion is more caudal. Some posteriorly or laterally placed meningiomas can be removed without opening the arachnoid. When necessary the arachnoid is perforated with a sharp hook and then teased away from the cord with a blunt instrument. Small vessels lying in the *subarachnoid* space are avoided. Not infrequently the arachnoid is torn inadvertently when the dura mater is incised, although it is desirable to keep it intact at first to facilitate the passage of an exploratory catheter in the subdural space (*vide supra*) and also to protect the vessels on the spinal cord while the incision in the dura is being extended.

When the intradural part of the operation is finished the dura is closed with interrupted fine silk sutures. Gaps in the dura left by excision of meningiomas may be closed with pieces of deep fascia, although some neurosurgeons do not close the gaps. The muscle layer and skin are closed with interrupted sutures of waxed silk. A thickening of the superficial fascia allows the skin to be sutured in two layers. A cerebrospinal fluid fistula and consequent meningitis may result from inadequate closure.



FIG. 405. Standard double-action rongeur.



into the bladder. Urine consequently leaks around the catheter. The fistula may become adherent to the pubic crest, and in some cases osteomyelitis of the pubis develops. The low attachment of the bladder discourages its expansion and therefore prevents the development of periodic micturition. *High suprapubic cystostomy* creates the best conditions for the prevention or control of urinary infection and for the ultimate development of periodic micturition (Chap. 28). Patients with a high level of sensory loss need no anaesthetic.

In many cases bladder function returns to normal, either spontaneously or as the result of treatment of the causal lesion (e.g. spinal tumour). Until that time arrives the greatest care must be taken of the urinary tract.

### LAMINECTOMY

For cervical or upper thoracic laminectomy the patient is placed in the sitting position on an operating chair. This posture reduces both arterial and venous bleeding, but it is necessary to bandage the patient's legs to prevent too great a fall in blood pressure. More caudal laminectomies are performed with the patient in the lateral or prone position. The head of the table may be tilted slightly downward to minimise venous bleeding in lumbar laminectomy. *Suction, irrigation and diathermy are used as in cranial surgery* (Chap. 54). The skin incision is made longitudinally in the midline and may be extended upwards or downwards during the operation. The deep fascia is incised with cutting diathermy along the line of the spinous processes. When the tips of the spines have been denuded they are nipped off with rongeurs, for their bulk prevents a clear view of the sides of the spinous processes, against which the point of the diathermy needle must cut if bleeding is to be minimised; if the needle is allowed to wander laterally into the muscle, bleeding is sure to be produced. The laminae are denuded as far laterally as the diarthrodial joints of the vertebrae. Muscle attached to each ligamentum flavum is coagulated before being cut as it often contains vessels which penetrate the ligament. Haemostasis is strictly maintained to avoid shock, and to ensure a clear view when the dura mater is opened. Two self-retaining retractors are used to separate the spinal muscles. In the cervical and upper thoracic regions the laminae can easily be removed with a double-action rongeur alone (Fig. 405), but in the lower thoracic and lumbar regions the laminae are thick; therefore, it is easier to begin by making burr-holes in the laminae, but only very slight pressure should be exerted on the brace. Further removal of bone may then proceed with the rongeur. A modified sphenoid-punch is very useful for removing the most lateral parts of the laminae in the lumbar region. In the cervical region there is some tendency to instability of the spine after extensive laminectomy. Therefore, whenever possible hemilaminectomy is performed (i.e. only laminae on the side of the lesion are removed). Elsewhere extensive laminectomy

are affected much more often than women. Trauma, usually of minor degree, precedes the onset of symptoms in about half the cases.

*Cervical Disc Protrusion.* The common sites of protrusion in the cervical region are between vertebrae  $C_5$  and  $C_6$ , and between  $C_6$  and  $C_7$ . It should be noted that there are only six discs in the cervical region, for there is no disc between the occiput and atlas or between the atlas and axis. The clinical course frequently begins with repeated attacks of stiff neck. Pain occurs at the base of the neck and over the shoulder, and radiates down the arm. *Spurling's test*, which consists of applying downward pressure on the patient's head when it is rotated and inclined towards the affected side, brings on the pain, whereas traction on the head may relieve it. With laterally placed  $C_5/C_6$  protrusions pain is referred to the anterior axillary fold, the anterolateral aspect of the arm as far as the elbow, and along the radial side of the forearm as far as, but not beyond, the wrist.  $C_6/C_7$  protrusions compress  $C_6$  nerve root and therefore cause weakness and wasting of the deltoid and biceps. The biceps jerk may be diminished or absent. Sensory loss affects the skin over the first interosseous space and the base and dorsal aspect of the thumb.

With a laterally placed  $C_6/C_7$  protrusion, pain radiates down the posterior axillary fold, the posterolateral aspect of the arm and the dorsum of the forearm as far as the wrist. The  $C_7$  nerve root is compressed. Therefore there is weakness and wasting of the triceps and extensors of the wrist and fingers. The triceps jerk is diminished or absent. Sensory loss affects the skin of the terminal phalanx of the index finger, and sometimes of the middle finger and tip of the thumb. Localisation by clinical examination can be erroneous when there is pre- or post-fixation of the roots of the brachial plexus.

Sometimes *spinal cord compression* occurs, alone or in combination with root compression. As might be expected, the posterior columns are often spared. The cord may be involved bilaterally or unilaterally. When the motor tracts only are affected confusion with disseminated sclerosis may occur.

*Thoracic Disc Protrusion.* Protrusions occur less frequently in the thoracic region than elsewhere. They cause unilateral root-pain which may be mistaken for pain arising from disease of thoracic or abdominal viscera. The spinal cord may be affected with or without root compression.

*Lumbar Disc Protrusion.* Disc protrusion occurs more often in the lumbar region than in other parts of the spinal column. The lower lumbar discs are much more often affected than the upper, and the  $L_5/S_1$  disc more often than  $L_4/L_5$  disc. It is seldom possible on the neurological evidence alone to distinguish with certainty between  $L_5/S_1$  and  $L_4/L_5$  disc protrusions. Very rarely lumbar disc protrusion follows a difficult lumbar puncture.

*The Lower Lateral Lumbar Syndrome ( $L_5/S_1$  or  $L_4/L_5$ ).* Pain in the lumbar region ("lumbago") usually, but not invariably, precedes or

## EXTRADURAL LESIONS

**Spinal Injuries.** The orthopædic aspects of this subject are discussed in Chap 98. Only the neurological complications are dealt with here. The spinal cord may be injured in flexion injuries of the spine, and also, though less often, in extension injuries. Neurological effects range from brief transient paralysis of the limbs to permanent quadriplegia or paraplegia, with paralysis of the bladder and rectum. Paralysis may occur without a fracture, especially in cervical strains, and severe fractures may not be accompanied by paralysis. The association of dislocation with a fracture greatly increases the chances of neurological damage. Very occasionally the onset of the neurological disturbances is delayed for some hours. This phenomenon should not be confused with the syndrome to which Kümmel gave his name in 1921. He described a painful angular deformity of the spine, developing weeks, months or years after an injury. The original radiographs were normal whereas those obtained subsequently showed collapse of a vertebra or intervertebral disc. The existence of this syndrome has not been confirmed.

When neurological recovery does not occur spontaneously after spinal injury there is very rarely, if ever, anything to be gained by exploratory laminectomy. Factors favouring useful recovery are :—(a) *Careful handling of the patient after the accident* ; (b) the presence of some spinal cord function immediately or soon after the accident ; thus if sensation remains in the presence of paralysis there is a good chance of recovery ; (c) the avoidance of urinary infection ; and (d) the avoidance of bed sores, for bacterial toxins absorbed from the infected area delay neurological recovery.

**Missile Injuries.** The spinal cord may be injured directly by missiles, sharp objects, or bone fragments, and sometimes temporary or permanent paralysis is produced by the passage of high-velocity missiles through tissues adjacent to the spinal column. Missiles or other foreign bodies, when accessible, should be removed along with devitalised tissue and loose bone fragments. Hæmostasis is attained and the wound closed without drainage. Sulphadiazine is given by mouth as a prophylactic against meningitis. Penicillin may be given intrathecally (never more than 20,000 units in twenty-four hours) ; it is not passed from the blood stream into the cerebrospinal fluid.

**Intervertebral Disc Protrusion.** Backward protrusion of the *nucleus pulposus* through a defect in the *annulus fibrosus* may affect any intervertebral disc at any point from the midline to the most lateral limits of the posterior aspect of the annulus. Protrusion of the *nucleus pulposus* into the vertebral body, as described by Schmorl, has no neurological significance. Therefore the term disc protrusion will be used to indicate backward herniation of the *nucleus pulposus* only. Symptoms occur very rarely before the age of twenty years or after the age of sixty years. Men

and muscles supplied by all the sacral nerves. Urgency of micturition and later retention of urine and constipation may occur. Disturbances of sexual function are sometimes mentioned by male patients. Central or paracentral protrusions of  $L_2/L_3$  or  $L_3/L_4$  discs involve all the sacral nerves and also the last two or three lumbar nerves. Thus sensory loss over the front of both thighs; bilateral paresis of the quadriceps, hamstrings, and sometimes also of the adductors of the hips; and loss of knee jerks are added to the bilateral lower lumbar syndrome (*vide supra*). Sometimes the onset of the bilateral cauda equina syndrome is sudden and demands immediate operation if permanent paralysis is to be avoided.

**Radiography.** Narrowing of the affected disc space with increased density and "lipping" of the adjacent vertebral bodies is usually found in patients with cervical and thoracic disc protrusions, but only in the minority of patients with a lumbar disc syndrome. These appearances are sometimes found in patients with no outward manifestations of disc protrusion, and, furthermore, at operation the protrusion may be found at a different level from that of the radiological signs. The normal disc space between the fifth lumbar vertebra and the sacrum is different in outline and depth from those above and is therefore sometimes mistakenly reported as narrowed. *Myelography* is often negative, for many protrusions are too far lateral to indent the spinal theca. Some protrusions obliterate the corresponding nerve sheath or deform the column of contrast medium (Fig. 406).

**Treatment of Disc Protrusion. A. Cervical Disc Protrusion.** The majority of patients suffering from cervical disc protrusion are treated conservatively. The head and neck may be immobilised in a leather appliance (Fig. 407), but if the pain is very severe and intractable the patient is confined to bed and up to 10 lb. traction applied to the head by means of a leather halter. Operation is indicated for lateral protrusions when conservative treatment fails. Whenever possible *hemilaminectomy* rather than full laminectomy is performed, in order to preserve the stability of the spine. When lateral disc protrusions are removed, it is sometimes also necessary to remove the posterior wall of the neural foramen to decompress the affected nerve root. It is neither feasible nor necessary to remove the remains of the nucleus pulposus from between



FIG. 407. Leather appliance as used by the author for cervical disc protrusion. The posterior section must extend well up over the occiput.

accompanies pain radiating down the back of the thigh, outer side of the calf, and outer side of the foot ("sciatica"). The pain is aggravated by coughing or sneezing. Occasionally patients complain of weakness of plantar flexion of the foot.

On examination, there may be flattening of the lumbar lordosis, scoliosis and limitation of forward flexion of the spine. Firm pressure over the  $L_5/S_1$  interlaminar space on the affected side may cause pain to radiate into the leg when the protrusion is at this level. When positive, therefore, this sign does distinguish between  $L_4/L_5$  and  $L_5/S_1$  protrusions. Passive flexion of the hip with the knee extended causes pain at the back of the thigh and spasm of the hamstrings (the diagnosis of lower lateral lumbar disc protrusion is untenable if this sign is negative). Sometimes



FIG. 406. A large paracentral lumbar disc protrusion ( $L_4/L_5$ ) demonstrated by myelography. The patient had severe bilateral "sciatica" which was relieved by operation.

the sign is also positive, on the opposite side, but not to the same degree. There is often weakness of flexion of the knee ( $L_5$ ) and plantar ( $S_1$ ) and dorsiflexion ( $S_2$ ) of the ankle. The glutei ( $S_1$  and  $S_2$ ) may also be weak. The weakened muscles are often wasted and hypotonic. The ankle jerk may be absent, diminished or unaffected. Sensory loss varies considerably, but commonly there is diminished or absent sensation over the toes and outer side of the foot and calf.

*The Upper Lateral Lumbar Syndrome.* The discs involved are usually  $L_2/L_3$  or  $L_3/L_4$ . Pain and sometimes sensory loss occur in front of the thigh together with paresis and wasting of the quadriceps ( $L_4$ ). The knee jerk is diminished or absent. Sometimes upper and lower lateral lumbar syndromes occur together.

*Bilateral Lumbar Syndromes.* Central or paracentral lumbar disc protrusions cause bilateral compression of the cauda equina; protrusions of the  $L_4/L_5$  or  $L_5/S_1$  discs affect, to a variable extent, the sensory territory

severe that the patient is unwilling to submit to, or continue with conservative treatment; (c) paresis is severe; (d) the sphincters are affected; and (e) *central or paracentral protrusion causes an acute bilateral cauda equina syndrome (myelography should be carried out at once and be followed by urgent operation).*

**Operation for Lumbar Protrusions.** A longitudinal midline incision is made corresponding with the level of the protrusion and the muscles are separated and retracted as described under laminectomy. The spinous processes of the vertebrae above and below the affected disc are removed. The ligamentum flavum on the affected side is excised and sufficient bone is nibbled away from the contiguous laminae, with a modified sphenoid punch, to obtain a clear view of the disc region. Sometimes the  $L_5/S_1$  laminae can be left intact, and it is never necessary to do a complete laminectomy for *lateral* lumbar protrusions. In lateral protrusions, a nerve root ( $L_5$  or  $S_1$ ) lies in its dural sheath stretched over the protrusion. This root must be retracted to reveal the glistening yellowish-white dome of the protrusion, which is then incised with a pointed tenotome. The cutting edge is directed away from the nerve and the theca. The protrusion and the remainder of nucleus pulposus between the vertebrae are removed. The best instrument for this purpose is a small pituitary rongeur which can easily be inserted between the two vertebrae. Paracentral and even central protrusions can be removed extradurally, but when this approach is found to be difficult, the dura mater is opened. The nerves of the cauda equina are then separated, and the anterior wall of the theca is incised over the disc protrusion. Sometimes the nucleus pulposus is pulled out whole (Fig. 409), but usually it has to be removed piecemeal.

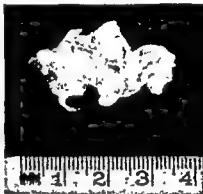


FIG. 409. A large disc sequestrum removed in one piece.

Occasionally at operation when a disc protrusion is not discovered, the dural sheath of the affected nerve is found to be fixed to the posterior longitudinal ligament by dense adhesions. In these cases, there is usually a long history of pain without remission. There is a good prospect of relief from pain if the nerve is liberated (neurolysis). This condition is probably the aftermath of a disc protrusion. A return of symptoms after operation is caused by a recurrent protrusion or by a protrusion at another level.

“**Sciatica.**” By far the commonest cause of sciatic pain is protrusion of a  $L_5/S_1$  or  $L_4/L_5$  disc, and, even when other pathology is discovered by investigations, there is always the possibility of a coexistent disc protrusion. Sciatica during pregnancy was once thought to be caused by

the vertebral bodies, as in the case of lumbar protrusions. It is extremely dangerous to operate on central or paracentral cervical disc protrusions, although it is sometimes impossible to avoid doing so when the syndrome is indistinguishable from that of a spinal cord tumour. Laminectomy, even without attempted removal of the disc protrusion, may cause complete and permanent quadriplegia. If such a protrusion is discovered accidentally at operation, the most that should be done is to relieve pressure on the cord by dividing the attachments of the denticulate ligaments in the neighbourhood of the compression.

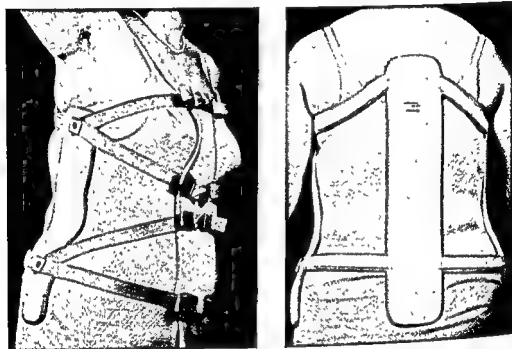


FIG. 408. The Fitzgerald brace for lumbar disc protrusion. The appliance restricts lumbar movements and thus encourages spontaneous recovery.

**B. Thoracic Disc Protrusion.** If it is possible to establish the diagnosis with certainty, treatment should be conservative, for, as with central and paracentral cervical protrusions, there is a serious risk of permanent damage to the spinal cord if laminectomy is performed. No satisfactory explanation of this phenomenon has so far been offered. When a thoracic disc protrusion is discovered at operation, performed for a suspected spinal cord tumour, no attempt should be made to remove it. The surgeon should do no more than divide adjacent attachments of the denticulate ligaments.

**C. Lumbar Disc Protrusion.** The majority of patients are treated conservatively. The lumbar spine is immobilised in its natural position by means of an appliance (Fig. 408). Operation is undertaken when (a) conservative treatment fails after a reasonable trial; (b) pain is so

complete interruption of spinal cord function, for a limit must be set to the powers of endurance of the cord; (c) paralysis of sudden onset caused by collapse of a vertebra or displacement of an intervertebral disc.

The object of operation is to relieve any compression of the cord without weakening the spine. Laminectomy is likely to cause instability of the spine when vertebral bodies are diseased. Therefore exploration must be carried out with the minimum removal of bone, and the spine should then be strengthened by a bone graft.

An operation (*costotransversectomy*) which preserves the stability of the spine has been devised by Dott, Alexander and Capener. The spinal cord and the diseased bone are approached from the side without interference with the laminæ. A semicircular skin incision is made on the side of the compression based on the midline. The superficial muscles are detached and retracted laterally, and the erector spinæ muscles are retracted medially. The angles of the ribs are thus exposed. The medial extremities of several ribs, together with the corresponding transverse processes, are removed. In the presence of diseased tissues and distorted anatomy, the intercostal nerves guide the surgeon to the pedicles and the spinal theca. Pedicles are nibbled away until a good view of the site of the compression is obtained. Then all tissue which indents the dura is removed, together with the carious bone of the vertebral bodies. This usually results in the restoration of dural pulsation. The operation is performed with the patient on the anterior section of his plaster shell.

**Epidural Abscess** (Syn. Extradural Non-tuberculous Abscess or Acute Spinal Pachymeningitis). There is a sudden onset of a rapidly progressive transverse lesion of the spinal cord accompanied by pyrexia. The patient may complain of pain in the affected region of the spine. The causes are:—(a) Blood-borne infection (organism usually *Staphylococcus aureus*), occurring with or without a known focus of infection, but without surrounding osteomyelitis—this is the usual type; (b) acute osteomyelitis of a vertebra (again blood-borne); (c) spread of infection from the chest; and (d) contamination of open spinal injuries.

Lumbar puncture reveals a manometric block, an increase in protein and a slight or moderate increase in cells in the cerebrospinal fluid. The exact site of the block is demonstrated by myelography. As soon as the diagnosis is made, the patient is given a wide "spectrum" antibiotic, and laminectomy is performed over the site of the block to relieve compression, caused by inflamed dura mater and pus. Normal-looking dura mater should be exposed above and below the lesion. The pus is examined for organisms, and their sensitivity to the various antibiotics is ascertained. The laminectomy wound is powdered with a mixture of penicillin and a sulphonamide and closed without drainage. There is a poor prospect of neurological recovery when paralysis below the lesion is complete. A good prognosis therefore depends on early diagnosis and prompt treatment. Epidural abscess is a surgical emergency.



pressure of the foetal head on the sacral plexus, but there is now no doubt that disc protrusion is the cause in the majority of cases. The other causes of sciatica are classified as follows :

**A. Sacro-iliac Lesions.** Subluxation of the sacro-iliac joint (rare); Marie's spondylitis ankylopoietica which commonly begins in the sacro-iliac joints ; tuberculosis of the sacro-iliac joint (rare), which radiologically shows irregular rarefaction of adjacent bone ; and metastatic carcinoma which commonly occurs in the ilium or sacrum from carcinoma of the prostate or other pelvic organs.

**B. Lesions of the Vertebrae.** Spondylolisthesis is sometimes associated with sciatic pain which is caused either by a disc protrusion or by nerve compression between the normal disc and the laminae of the two vertebrae involved in the displacement ; metastatic carcinoma of the lumbar vertebrae, from a primary tumour of the prostate, may cause sciatic pain ; chordomas invade the sacral plexus, but they are rare tumours.

**C. Intrapelvic Disease.** The sacral plexus may be infiltrated in advanced carcinoma of the rectum, cervix or ovary.

**D. Tumours of the Cauda Equina.** Severe unilateral sciatic pain is one of the characteristics of some cauda equina tumours. More physical signs are likely to be found with tumours than with disc protrusions, and tumours nearly always produce bilateral effects. There is usually a manometric block and a considerable increase in cerebrospinal fluid protein.

**Potts' Disease of the Spine (Syn. Tuberculous Spondylitis).** Usually two adjacent vertebrae and the intervening disc are affected. The resultant destruction of the bone and disc causes the characteristic angular deformity or gibbus. Less often several contiguous vertebrae are involved or very occasionally the disease begins in a lamina. The following are the causes of involvement of the spinal cord :—(a) Extradural abscess ; (b) sudden collapse of a vertebra, spontaneous or after laminectomy ; (c) displacement of a sequestrum or intervertebral disc ; (d) involvement of the spinal cord in the inflammatory process, i.e. tuberculous transverse myelopathy.

The spinal cord is frequently affected in the active stage of Potts' spondylitis. Thus weakness of the lower extremities may be the presenting symptom of the disease. Sometimes paraplegia (quadriplegia when the cervical spine is the site of the disease) appears years after apparent cure. As marked angulation is frequently seen with healed lesions without neurological effects, it therefore seems unlikely that angulation of the spine alone can cause paralysis. There is evidence that reactivation of the disease is also necessary.

**Treatment** is conservative when neurological signs are slight or moderate in degree. Retention of urine is treated as described earlier in this chapter. The indications for operation on neurological grounds are :—(a) Failure of response to conservative treatment ; (b) signs of marked or

hausen's disease, although there may be present the same kind of cutaneous *café au lait* patches seen in this disease. Less often spinal cord neurofibromas are associated with fully developed von Recklinghausen's neurofibromatosis. Neurofibromas are thought to arise either from the epineurium or from Schwann cells. They are firmly attached to the nerve of origin. Radiography may show erosion of the pedicles at the site of the tumour and an increase in the horizontal distance between them. There is no calcification in these tumours. Some neurofibromas extend through the neural foramen along the affected nerve (*dumb-bell tumours*), and enlargement of the neural canal can be



FIG. 411. Left cervical swelling (reader's right) deep to the lower end of the sternomastoid muscle. The tumour was a neurofibroma which extended into the spinal canal causing paraplegia and lower neuron manifestations in the left upper limb. The paraplegia was relieved by removal of the spinal part of the tumour. The cervical part had to be left in place because of its intimate attachment to the brachial plexus.

demonstrated radiologically (Fig. 410). If the extraspinal part is large, it may be palpable when the tumour arises in the cervical or lumbar region (Fig. 411); intrathoracic extensions may be seen in X-rays of the chest. Unimportant nerve roots may be sacrificed in the removal of these tumours, but important motor and sensory nerves should be preserved.

**Cauda Equina Ependymomas.** The *relatively* benign character of ependymomas is outweighed by their arising in awkward situations (e.g. the floor of the fourth ventricle, within the spinal cord, or in the cauda equina). Ependymomas of the cauda equina grow from the filum terminale or the conus medullaris. As with other tumours in this spacious region, they tend to grow to a large size before producing disabling neurological effects. Radiography may show widening of the lumbar

Hodgkin's disease sometimes spreads from the mediastinum through the neural foramina into the spinal canal and causes compression of the spinal cord. The extradural plaque should be dissected off the posterior and lateral surface of the dura mater. There is a good prospect of neurological recovery after early operation. *Leukæmia* may also cause extradural deposits.

**Metastatic Tumours.** A transverse spinal cord lesion may be caused by vertebral metastasis, or by spread of a tumour along neural sheaths into the extradural space.

### INTRADURAL EXTRAMEDULLARY LESIONS

**Meningiomas** (Syn. Meningeal Fibroblastomas, Endotheliomas or Psammomas). Meningiomas are benign tumours which are thought to arise from arachnoid cells. The tumours are always attached to the dura mater from which they obtain their blood supply. They may arise from any part of the spinal dura mater and may therefore give rise to any of the spinal cord syndromes previously described. Spinal meningiomas are slowly growing, and therefore cause *slowly progressive* neurological effects. Radiography may demonstrate:—(a) The calcification which is nearly always present in *spinal* meningiomas (hence the term psammoma\*); (b) erosion of pedicles at the site of the tumour and an increase in the horizontal distance between them. Myelography is usually employed to

verify the level of the tumour (Fig. 403). The dural attachment is excised with the tumour to prevent recurrence. In the region of the cauda equina, giant tumours may be encountered because of the large space available for growth. Intradural tumours in this situation tend to become adherent to nerve roots. Intracapsular removal may therefore be advisable in some cases if permanent damage to the cauda equina is to be avoided.

**Neurofibromas** (Syn. Perineural Fibroblastomas, Schwannomas, Neurinomas, Neurilemmomas). These tumours are also benign and therefore cause a *slowly progressive* syndrome. Some pathologists consider solitary neurofibromas to be different from the multiple neurofibromas of von Recklinghausen's disease (Chap. 61). Spinal cord compression is frequently caused by solitary neurofibromas, occurring without evidence of von Reckling-



FIG. 410. Enlarged neural foramen of the vertebra, marked by the point of a paper-clip, caused by a dumb-bell neurofibroma.

\* Psammos = sand.

**Dermoid Cysts.** The capsule of dermoid cysts contains both epidermis and dermis, and therefore, unlike epidermoids, they contain hair and sebaceous material. They may be found at any level of the spinal cord and have a tendency to involve many segments. There may be an associated dermal sinus or fistula. The contents of the cyst should be evacuated, but it may be impossible to remove the capsule without damaging the spinal cord or nerve roots.

**Secondary Tumours.** Meningeal metastasis may arise from intracranial tumours (notably from medulloblastomas and ependymomas), or from primary tumours outside the central nervous system.

### INTRAMEDULLARY LESIONS

**Ependymomas.** These are the commonest intramedullary tumours, although all varieties of glioma may be found in the spinal cord. Intramedullary tumours produce swelling of several or many segments of the spinal cord. Clinically they cannot usually be distinguished with absolute certainty from extramedullary ones. Nothing is to be gained by removing intramedullary tumours.

**Secondary tumours** in the spinal cord may arise from primary malignant intracranial tumours, especially medulloblastomas and ependymomas, or from primary malignant tumours outside the central nervous system (rare).

### SPINA BIFIDA

Spina bifida results from failure of complete closure of the spinal canal—and sometimes of the spinal cord—during foetal development. It is found most frequently in the lumbar region, which is normally the last part to close. The presence of hydrocephalus (Fig. 413), or its appearance after operative treatment of spina bifida is well known. The lesion, past or present, responsible for the hydrocephalus is also the cause of the spina bifida; both result from raised intracranial pressure. Therefore those patients with a spina bifida which is considered operable should have a complete neurosurgical investigation, including ventriculography (see under hydrocephalus). The ventricles are usually already dilated even when the skull is not enlarged. The failure of the skull to enlarge in some cases is due to the decompression effect of the spina bifida.

### TYPES OF SPINA BIFIDA

**Spina Bifida Occulta.** There is no protrusion of the membranes or nervous structures, but neurological signs are associated with defects of the posterior arches of the vertebrae. The cauda equina may be entangled in a mass of fibrous tissue, or compressed by a dermoid or epidermoid cyst. The diagnosis is sometimes suggested by the presence of a subcutaneous lipoma, a tuft of hair, or a dermal sinus. Note.—Defects in the arches of the vertebrae are frequently revealed by radiography when no neurological signs are present.

and sacral spinal canal, concavity of the posterior surfaces of the vertebral bodies and erosion of the pedicles (Fig. 412). Lumbar puncture may produce no cerebrospinal fluid, and it may cause severe pain because nerve roots cannot be pushed aside by the needle. At operation, great care is necessary in opening the dura mater, for the nerve roots are held against it by the tumour. Therefore it is best to begin the incision in the dura above or below the level of the tumour. The interior of the tumour is gently evacuated with the aid of a pituitary rongeur. The capsule is

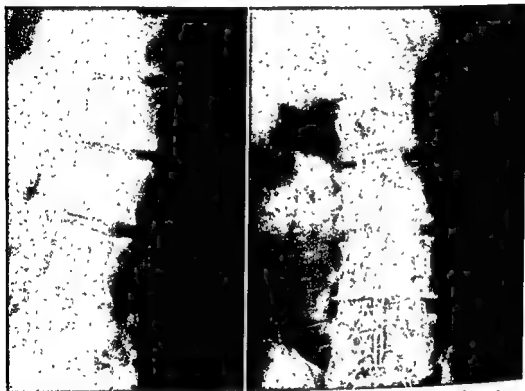


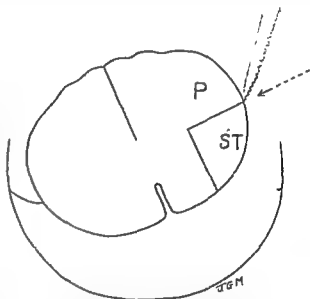
FIG. 412. On the right, the pedicles of the second lumbar vertebra have become wedge-shaped, and the horizontal distance between them increased. On the left, in the lateral view, marked erosion of the posterior aspects of the bodies of the second and third lumbar vertebrae is shown. These appearances were caused by a giant ependymoma.

left in place if it is adherent to the nerve roots. The cauda equina is a delicate structure which is liable to be permanently damaged by compression and rough handling. The removal of a large tumour may delay or arrest the development of further neurological signs, but those already present may not regress.

**Epidermoid Cysts** (Syn. Cholesteatomas or Cruveilhier's Tumeurs Perlées). These rare benign tumours are of developmental origin, and consist of a mass of epithelial debris and cholesterol crystals enclosed by a capsule of epithelium without dermal structures. These tumours occur also in, and within, the skull. Intracapsular removal is carried out when the capsule is intimately adherent to the spinal cord and nerve roots.

*Operation for Spina Bifida.* A longitudinal elliptical skin incision is made. Care must be taken that enough skin is left for closure. The muscles are stripped from the spines and laminae above and below the deformity. The sac is opened, and if there are no nervous structures attached to its wall, it is amputated and the opening sutured. Pedicles of deep fascia are turned towards the midline to complete the repair. When there is no sac (*spina bifida occulta*) the theca may be explored in an attempt to relieve the neurological defect. Sometimes a dermoid or epidermoid cyst is found. The contents of these cysts should be evacuated.

FIG. 414. Spinothalamic tractotomy. The position and extent of the cut in the spinal cord is shown. P = the crossed pyramidal tract and ST = the spinothalamic tract.



In other cases, nerve roots are found adherent to the region of the bone defect and intermingled with fibro-lipomatous tissue. In these circumstances, the neurological deficit would be increased by an attempt to free the affected nerves.

#### SPINOTHALAMIC TRACTOTOMY

Nerve fibres conveying sensation of pain and temperature enter the spinal cord through the posterior nerve roots and then cross in the grey matter of the spinal cord to join the opposite lateral spinothalamic tract. Division of the tract may be performed for the relief of intractable pain caused by incurable disease. When the pain is unilateral the tract on the *opposite* side is divided (Fig. 414). Occasionally the tract is divided on both sides for bilateral pain caused by malignant tumours. Only pain and temperature sensation are interrupted, for other forms of sensation are conveyed in the posterior columns. The depth of the incision varies with the level of the cordotomy (upper cervical 6 mm., lower cervical 7 mm., and upper thoracic 5 mm.).

**Meningocele.** This is a protrusion of membranes without nervous structures. Expansion usually occurs when the child cries. The connection with the theca is often quite narrow. Cauda equina signs are minimal or absent.

**Myelomeningocele.** The protrusion contains part of the spinal cord and some nerve roots. Marked cauda equina and spinal cord signs are invariably present.

**Syringomyelocele.** This is a rare form of spina bifida in which the herniation is occupied by a cystic expansion of the spinal cord.



FIG. 413. Spina bifida (myelomeningocele) associated with hydrocephalus. There was a complete cauda equina syndrome.

**Myelocele.** In this deformity the two sides of the spinal cord, as well as the more superficial tissues, have failed to fold together. The open spinal cord appears on the surface of the back as a red granular area with cerebrospinal fluid flowing from its centre.

**Management of Spina Bifida.** When cauda equina signs are present, little or no improvement follows operation. The surgeon has therefore to ask himself whether it is justifiable to save the paraplegic and incontinent child from the lethal effects of ulceration of the overlying skin. If there are no neurological signs, or if those present are considered compatible with a useful life, the child is first investigated for evidence of hydrocephalus, which, if present, is treated before the spina bifida. The investigation and management of hydrocephalus are described in Chap. 55.

fibres arising in the spinal cord are *preganglionic* and have medullated sheaths which give the white rami their name. At operation, however, the surgeon cannot distinguish the white rami from the grey ones which contain the non-medullated postganglionic fibres.

Sympathectomy produces vasoconstrictor paralysis in the skin and anhidrosis (loss of sweating), but there is little or no direct effect on voluntary muscles, although their blood supply may be improved distal to arterial occlusion by the increase in collateral circulation resulting from sympathectomy.

The parasympathetic system consists of medullated efferent nerve fibres in the third, seventh, ninth and tenth cranial nerves, and in the pelvic nerves (S.2, S.3 and S.4).

The hypothalamic nuclei and the frontal lobes of the cerebrum are the higher centres of the autonomic system, and exercise control through incompletely charted efferent pathways in the spinal cord.

Nerve impulses are transmitted across synapses and end-organs by chemical means. Thus *acetylcholine* mediates all parasympathetic function, the transmission of nerve impulses across synapses in sympathetic ganglia, the secretion of sweat glands, the function of the suprarenal medulla and the activation of *voluntary* muscle fibres. *Sympathin* (probably noradrenaline, i.e. adrenaline minus a methyl group) is the chemical agent responsible for the transmission of impulses between sympathetic nerve endings and effector organs, with the exception of sweat glands which respond to acetylcholine. Thus injections of adrenaline do not cause sweating although all other sympathetic effects occur. Nerves which liberate acetylcholine are termed *cholinergic* and those which liberate *sympathin* are termed *adrenergic*.

Visceral afferent (sensory) nerve fibres, although not now regarded as a functional part of the autonomic nervous system, run in sympathetic and parasympathetic nerves and plexuses, without synaptic interruption. Those sensory fibres which traverse the sympathetic nerves enter the spinal nerves through *white rami communicantes* (i.e. from T.1 to L.2), and, like all sensory fibres, pass into the *posterior* roots to their nerve cells in the posterior root ganglia. No sensory fibres have been demonstrated in the superior cervical sympathetic ganglia. Therefore there is no sensory downflow from the brain in the sympathetic chain.

### INVESTIGATIONS

The likely response to sympathectomy may be judged by temporarily blocking the nerve supply of the region concerned, and measuring the temperature of the skin with a skin thermometer or thermocouple. These investigations are, if possible, carried out in an air-conditioned room in which the patient is exposed to constant conditions for an hour beforehand. The methods employed are :—(a) Peripheral nerve block with local anaesthetic—the best results are obtained if more than one nerve is



## CHAPTER 60

### THE AUTONOMIC NERVOUS SYSTEM

THE autonomic nervous system is concerned with the innervation of involuntary muscle and glands. It is composed of two main parts:— (a) The *sympathetic* (thoraco-lumbar outflow, T.1 to L.2) ; and (b) the *parasympathetic* (cranio-sacral outflow). These two parts have different and sometimes opposing functions. The sympathetic system consists of two ganglionated trunks with their rami, branches and associated plexuses. Every ganglion is connected with a spinal nerve by a grey

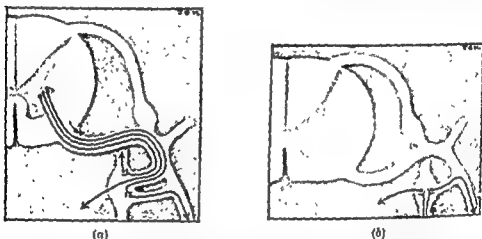


FIG. 415. (a) The course of sympathetic fibres at levels where there are both white and grey rami (T<sub>1</sub> to L<sub>2</sub>). (b) The course of sympathetic fibres above the first thoracic sympathetic ganglion. Below the second lumbar ganglion there are also no white rami, but the sympathetic fibres run caudally in the chain.

*ramus communicans*. From T.1 to L.2 each ganglion is also connected with the corresponding spinal nerve by a white *ramus communicans*. Thus there are no white rami in the neck, lower lumbar region or sacral region. The efferent sympathetic fibres of the spinal cord originate in the lateral column of grey matter from segments T.1 to L.2 inclusive and pass out along all thoracic anterior nerve roots and the first two lumbar anterior nerve roots. These efferent fibres then leave the anterior primary divisions of the spinal nerves in the white rami and enter the corresponding sympathetic ganglia where they do one of three things (Fig. 415):—(a) Form synapses with neurons which send their axons back into the spinal nerve through the grey ramus ; (b) pass upward or downward in the chain to form synapses with neurons in other ganglia ; or (c) pass through the ganglia without interruption to one of the autonomic plexuses. The

the sternal head of the sternomastoid. The external jugular vein is divided if small, and retracted if large. The clavicular head of the sternomastoid and the omohyoid are divided. The pad of fat deep to the latter is dissected free from below and retracted upward. The phrenic nerve is deliberately freed from scalenus anterior and retracted medially with a wet tape. Scalenus anterior is completely divided piecemeal with blunt-ended scissors as close as possible to the first rib. The subclavian artery is retracted downwards (sometimes upward when the artery has a high curve) with a wet tape. Some upgoing branches of the artery may have to be divided between ligatures. For safety, Sibson's fascia is separated from the first rib with the index finger because the T.1 contribution to the brachial plexus crosses the first rib. At first, the sympathetic chain is easier to feel than to see. *A good headlight is essential.* The stellate ganglion (the inferior cervical joined by an isthmus to the first thoracic ganglion) is a grey structure lying behind the vertebral artery and partly buried in longus cervicis in front of the transverse process of the seventh cervical vertebra and the head of the first rib. On the left side the thoracic duct lies close to the medial border of scalenus anterior, but it is not usually seen. The chain is caught with a long nerve hook, and the rami of the stellate ganglion and the thoracic ganglia down to and including T.3 are cut with scissors. Then the chain is divided above the stellate ganglion and below the T.3 ganglion, and the segment of chain removed. This amount of the chain is removed to discourage regeneration which so readily occurs in the peripheral sympathetic system.

Removal of the stellate ganglion causes the somewhat unsightly Horner's syndrome because the spinal sympathetic supply to the eye originates in the first thoracic segment of the spinal cord and flows outward through the first thoracic nerve and its sympathetic ganglion (part of the stellate ganglion). This effect may be avoided by removal of the sympathetic chain from just below T.1 ganglion to below T.3 ganglion, for the sympathetic efferents to the arm originate in spinal segments T.2 to T.10, and flow out of the cord through the corresponding anterior nerve roots and white rami, and then run upward in the chain to the middle and inferior cervical ganglia which supply the brachial plexus level (Fig. 416). Some surgeons, however, prefer to remove the stellate ganglion (i.e. postganglionic section) because they believe the white ramus of T.1 also contains fibres for the upper limb, and there is also less chance of regeneration. After postganglionic operations, the arterioles become abnormally sensitive to circulating noradrenaline, although the degree of this effect is not now regarded as sufficient to affect the choice between pre- and postganglionic section. Both types of operation may also be carried out through a posterior extrapleural or an axillary transpleural approach.

**Thoraco-lumbar Sympathectomy.** Bilateral removal of most of the thoracic and lumbar sympathetic chain together with the splanchnic nerves for essential hypertension is an obsolete operation. The fall in

injected to allow for the considerable overlap in sensory and therefore sympathetic supply ; (b) injection of paravertebral sympathetic ganglia with local anæsthetic ; (c) spinal anæsthetic for the lower extremities ; and (d) reflex dilatation of arterioles by heating the body in a cabinet or by immersion of the opposite limbs in hot water (110° F. or 43° C.). The position of the limbs in regard to gravity should be identical. All these tests are applicable when an improved blood supply is required by sympathectomy, and test (b) when the object is the relief of pain. Failure to obtain a normal rise of temperature after nerve blocking indicates inadequate infiltration of the local anæsthetic or obliterative disease of the vessels.

Too much reliance should not be placed on these tests, for the results of operation may be better than the tests would indicate. Many surgeons do not employ them.

### OPERATIONS

**Resection of the Superior Cervical Ganglion.** The face, the eye and the cerebral vessels on the corresponding side are among the structures deprived of their sympathetic supply by this operation. Horner's syndrome (contracted pupil and partial closure of the eyelid) is produced.

*Indications.* (a) Hyperhidrosis (excessive sweating) of the face ; and (b) to improve the cerebral blood flow after cerebral apoplexy (of doubtful value).

*Operation.* The patient's neck is extended and turned to the opposite side. An incision is made through the skin and platysma in a transverse crease at the level of the upper border of the thyroid cartilage. Dissection is carried out in the carotid sheath beginning along the anterior border of the sternomastoid. The internal jugular vein and the sternomastoid are retracted laterally. The vagus nerve which tends to adhere to the posterior aspect of the internal carotid artery is identified. The sympathetic trunk lies deep to the carotid sheath and is recognised by the unmistakable widening produced by the superior cervical ganglion. All connecting rami are cut and the ganglion is excised.

**Stellate Ganglionectomy.** This operation produces sympathetic denervation of the corresponding half of the head and neck, and the homolateral upper limb, with the exception of the axilla. Horner's syndrome also results.

*Indications.* (a) Raynaud's disease ; (b) hyperhidrosis of the hands ; (c) acrocyanosis ; (d) angina pectoris (the chain must be removed down to and including the fifth sympathetic ganglion) ; (e) late vascular disturbances of frostbite and other types of cold injury ; (f) trophic ulceration of the skin ; (g) gangrene of the skin ; and (h) as an aid in conservative amputations for gangrene of the fingers.

*Operation.* An incision is made through the skin and platysma, a finger's breadth above the clavicle, from the lateral border of trapezius to

the sternal head of the sternomastoid. The external jugular vein is divided if small, and retracted if large. The clavicular head of the sternomastoid and the omohyoid are divided. The pad of fat deep to the latter is dissected free from below and retracted upward. The phrenic nerve is deliberately freed from scalenus anterior and retracted medially with a wet tape. Scalenus anterior is completely divided piecemeal with blunt-ended scissors as close as possible to the first rib. The subclavian artery is retracted downwards (sometimes upward when the artery has a high curve) with a wet tape. Some upgoing branches of the artery may have to be divided between ligatures. For safety, Sibson's fascia is separated from the first rib with the index finger because the T.1 contribution to the brachial plexus crosses the first rib. At first, the sympathetic chain is easier to feel than to see. *A good headlight is essential.* The stellate ganglion (the inferior cervical joined by an isthmus to the first thoracic ganglion) is a grey structure lying behind the vertebral artery and partly buried in longus cervicis in front of the transverse process of the seventh cervical vertebra and the head of the first rib. On the left side the thoracic duct lies close to the medial border of scalenus anterior, but it is not usually seen. The chain is caught with a long nerve hook, and the rami of the stellate ganglion and the thoracic ganglia down to and including T.3 are cut with scissors. Then the chain is divided above the stellate ganglion and below the T.3 ganglion, and the segment of chain removed. This amount of the chain is removed to discourage regeneration which so readily occurs in the peripheral sympathetic system.

Removal of the stellate ganglion causes the somewhat unsightly Horner's syndrome because the spinal sympathetic supply to the eye originates in the first thoracic segment of the spinal cord and flows outward through the first thoracic nerve and its sympathetic ganglion (part of the stellate ganglion). This effect may be avoided by removal of the sympathetic chain from just below T.1 ganglion to below T.3 ganglion, for the sympathetic efferents to the arm originate in spinal segments T.2 to T.10, and flow out of the cord through the corresponding anterior nerve roots and white rami, and then run upward in the chain to the middle and inferior cervical ganglia which supply the brachial plexus level (Fig. 416). Some surgeons, however, prefer to remove the stellate ganglion (i.e. postganglionic section) because they believe the white ramus of T.1 also contains fibres for the upper limb, and there is also less chance of regeneration. After postganglionic operations, the arterioles become abnormally sensitive to circulating noradrenaline, although the degree of this effect is not now regarded as sufficient to affect the choice between pre- and postganglionic section. Both types of operation may also be carried out through a posterior extrapleural or an axillary transpleural approach.

**Thoraco-lumbar Sympathectomy.** Bilateral removal of most of the thoracic and lumbar sympathetic chain together with the splanchnic nerves for essential hypertension is an obsolete operation. The fall in

blood pressure caused by visceral vasodilatation is not maintained, although some authors have claimed relief of headache.

**Lumbar Sympathectomy.** This operation causes sympathetic paralysis of the lower limb. The spinal sympathetic efferents originate in the grey matter in the spinal segments T.11 to L.2 (Fig. 416). They enter the corresponding anterior nerve roots and leave the anterior primary division of the spinal nerves through white rami and then enter the sympathetic chain. The postganglionic fibres leave the chain in grey rami from ganglia opposite the origin of the lumbar and sacral plexuses to join the anterior primary division of the nerves of these plexuses (Figs. 415 and 416). Thus removal of the lumbar chain produces postganglionic denervation of that part of the lower limb supplied by the lumbar plexus and preganglionic denervation of that part supplied by the sacral plexus.

**Indications.** (a) Raynaud's disease (rare in the toes); (b) thromboangiitis obliterans; (c) arteriosclerotic gangrene; (d) to permit conservative amputations for gangrene of the toes; (e) trophic ulceration of the feet; (f) in the after-treatment of circulatory stasis following frost-bite and other forms of cold injury; (g) as an aid in the treatment of chronic ulceration of the leg; and (h) for vasospasm in poliomyelitis and pyramidal disease.

**Operation.** The patient is placed on the side. An incision is made from the lateral border of sacrospinalis almost to

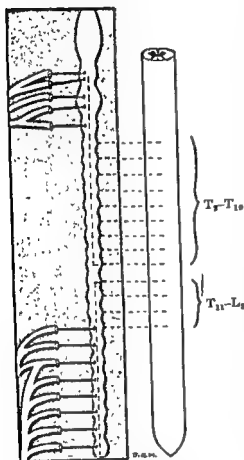


FIG. 416. The origin and distribution of the sympathetic supply to the limbs.

the umbilicus. The muscles are divided down to the lumbar fascia and the latter is incised. The peritoneum is separated from the parietes with the fingers. The sympathetic chain lies on the vertebral bodies running along the medial border of the psoas. On the right side the chain is covered by the inferior vena cava and on the left by the abdominal aorta. At first, the chain is often more easily felt than seen. The first lumbar ganglion lies under cover of the crus of the diaphragm and the fourth ganglion beneath the bifurcation of the common iliac artery. The part of the chain including the second, third and fourth ganglia is usually removed, but the first lumbar ganglion must also be removed when denervation is intended to include the skin of the groin.



blood pressure caused by visceral vasodilatation is not maintained, although some authors have claimed relief of headache.

**Lumbar Sympathectomy.** This operation causes sympathetic paralysis of the lower limb. The spinal sympathetic efferents originate in the grey matter in the spinal segments T.11 to L.2 (Fig. 416). They enter the corresponding anterior nerve roots and leave the anterior primary division

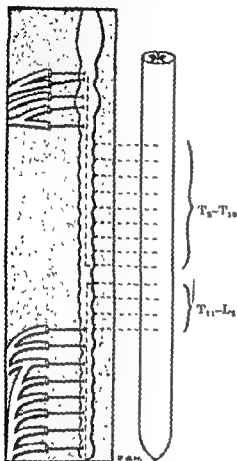


FIG. 416. The origin and distribution of the sympathetic supply to the limbs.

of the spinal nerves through white rami and then enter the sympathetic chain. The postganglionic fibres leave the chain in grey rami from ganglia opposite the origin of the lumbar and sacral plexuses to join the anterior primary division of the nerves of these plexuses (Figs. 415 and 416). Thus removal of the lumbar chain produces postganglionic denervation of that part of the lower limb supplied by the lumbar plexus and preganglionic denervation of that part supplied by the sacral plexus.

**Indications.** (a) Raynaud's disease (rare in the toes); (b) thromboangiitis obliterans; (c) arteriosclerotic gangrene; (d) to permit conservative amputations for gangrene of the toes; (e) trophic ulceration of the feet; (f) in the after-treatment of circulatory stasis following frost-bite and other forms of cold injury; (g) as an aid in the treatment of chronic ulceration of the leg; and (h) for vasospasm in poliomyelitis and pyramidal disease.

**Operation.** The patient is placed on the side. An incision is made from the lateral border of sacrospinalis almost to

If both L.1 ganglia are removed, there is a 50 per cent. chance of *sterility* in the male from paralysis of the seminal vesicles.

### POST-OPERATIVE COURSE

After sympathectomy of the limbs there is vasodilatation and consequent rise in temperature of the skin, but on the third to the fifth post-operative day the temperature falls abruptly and sweating returns. Then again sweating ceases and the skin temperature rises, although not to its immediate post-operative level.

*Post-operative Tests.* After sympathectomy of the limbs, sweating is paralysed (sudomotor paralysis). Therefore the quinizarin sweat test (see Chap. 61) may be employed to demonstrate the extent of the sympathectomy. It should be carried out after the normal "relapse" described above. Measurement of the electrical resistance of the skin is a delicate test of sympathetic denervation. It is based on the fact that the resistance is increased when the skin is dry.

### DISEASES AMENABLE TO SYMPATHECTOMY

Raynaud's disease, Buerger's disease, atherosclerosis and chronic ulcer of the leg are discussed under Peripheral Vascular Surgery.

**Cervical Rib Syndrome** (also Chap. 61). If the vascular phenomena of this syndrome, including ulceration of the fingers, are not relieved by removal of the rib and its fibrous band, sympathectomy should be considered, provided nerve block with procaine produces a satisfactory rise of temperature.

**Hyperhidrosis.** Excessive sweating of the hands and feet and sometimes of the face occurs most frequently in the male sex. In the hands it is treated by pre- or postganglionic sympathectomy of the upper extremities. If the axilla is involved the chain must be removed down to and including the T.5 ganglion. A posterior extrapleural or transpleural axillary approach must be employed to divide the chain at this level. After operation compensatory sweating may occur in other areas.

**Carotid Sinus Syndrome.** Attacks occur in which vagal inhibition, resulting from nerve impulses arising in an abnormally sensitive carotid sinus, causes slowing of the pulse rate and arterial hypotension. Consciousness is lost and convulsions may occur. Periarterial denervation of the carotid bifurcation is curative, for the afferent fibres of the reflex are thereby removed.

**Angina Pectoris.** The pain fibres from the heart travel by two routes:— (a) Along the middle and inferior cardiac nerves which go to the middle cervical and stellate ganglia respectively; and (b) directly to the upper five thoracic sympathetic ganglia. The neurons which enter the middle cervical and stellate ganglia descend in the sympathetic chain to enter the same spinal cord segments as the direct cardiac fibres (*viz.* T.1 to T.5) through the corresponding posterior nerve roots. These pathways may



be interrupted by:—(a) Injection of the upper five thoracic ganglia with alcohol ; (b) intrathoracic removal of the inferior cervical and upper five thoracic ganglia ; or (c) intradural section of the upper five posterior thoracic nerve roots. Some success has been claimed when these procedures have been carried out on the left side only, but relief of all cardiac pain is assured only by a bilateral procedure. Operation is indicated only in the most intractable cases, for the mortality has been estimated at 10 per cent. Operation does not take away the warning of attacks for patients still become breathless and have a feeling of malaise.

**Diseases of the Pancreas.** The pain of chronic relapsing calculous pancreatitis is relieved by bilateral removal of the splanchnic nerves with as much as possible of the sympathetic chain that can be removed through a supradiaphragmatic approach (resection of the eleventh ribs bilaterally). This operation is unsuitable for carcinomas of the pancreas as pain in this disease does not occur until surrounding structures (supplied by somatic nerves) are invaded.

## NEUROBLASTOMA AND GANGLIONEUROMA

by

VALENTINE A. J. SWAIN

Both these tumours arise from primitive elements of the sympathetic nervous system. Whereas neuroblastomas are one of the commonest malignant tumours in infancy, ganglioneuromas are rare and arise in older children and adults.

By neuroblastoma is meant a tumour consisting of immature undifferentiated neuroblasts ; and by ganglioneuroma is meant one consisting of well-differentiated nerve cells and fibres. The two types of growth are not sharply distinct, however ; many tumours of transitional or mixed structure occur and the two names denote merely the poorly differentiated and the highly differentiated members of the same species, the former rapidly growing and malignant, the latter slowly growing and relatively benign. All intermediate gradations of structure and behaviours are observed.—(WILLIS, 1948.)

**Neuroblastoma** (Syn. Undifferentiated Sympathicoblastoma). This tumour arises from the medullary part of the adrenal gland, or from the abdominal, thoracic or cervical sympathetic chain, and grows rapidly. It has a firm irregular nodular surface, and the cut surface shows whitish tissue interspersed with areas of hæmorrhage, necrosis and sometimes calcification. Histologically, it is composed of masses of round cells, resembling small lymphocytes, in a scanty stroma. In places, the cells are arranged in clusters termed rosettes. Neurofibrils can be seen after special staining. Mitosis is often marked. Surrounding structures soon become directly invaded and metastasis occurs in the liver (*Pepper's*

*syndrome*), the skeleton and the peripheral lymph nodes. The skull and long bones, especially the femur and humerus, are common sites for secondary deposits (many "Ewing's tumours" are thought by some pathologists to be secondaries from neuroblastomas). Proptosis (exophthalmos) associated with a secondary deposit in the orbit is known as *Hutchison's syndrome*. Not infrequently, cervical glands on the left side of the neck are affected and sometimes pulmonary metastasis also occurs.

Neuroblastomas affect male and female infants equally, and most commonly occur within the first four years of life; about half occur before the age of two years. The majority of children with this tumour have secondary deposits when first seen. Extensive adenopathy may be the first thing noticed. Sometimes abdominal manifestations are discovered on routine examination of a child presenting with vague symptoms such as malaise, wasting, anæmia, abdominal discomfort, fever or cough. Occasionally the mother notices an abdominal swelling. Locomotor disorders due to spinal compression may be the first manifestation. Examination reveals a swelling in the upper abdomen, usually indistinguishable from the liver, lying deeply across the midline. If the tumour is mostly unilateral, it may be difficult to distinguish from a renal or splenic swelling. The tumour surface is firm and nodular and fades deeply into the abdomen. The outline of the neoplasm is demonstrated by plain radiography of the abdomen; the liver shadow is enlarged and often shows calcification, which is thought to result from hæmorrhage within the hepatic deposits. Intravenous pyelography may show distortion of the renal pelvis or ureters; displacement of the kidneys downwards and laterally suggests the presence of a tumour originating in the adrenal region. All possible sites of metastasis should be examined. Sometimes radiography of the chest shows the primary tumour to be lying in the posterior part of the mediastinum. Liver biopsy may be positive.

*Differential Diagnosis.* A *Wilm's tumour* has a smoother, more coarsely lobulated surface compared with the finer nodular neuroblastoma. Furthermore, a renal tumour is felt in the loin. It rarely passes across the midline, and is separate from the liver above. A *Wilm's tumour* metastasizes in the lungs rather than in the bones.

*Treatment.* Excision of the tumour is followed by radiotherapy. Although neuroblastomas are usually widespread when the patients are first seen, exploration is carried out in case the mass can be removed. If this is impracticable a biopsy is done. Although neuroblastomas are radiosensitive, the degree of sensitivity varies inversely with the differentiation of the cells. Preoperative radiotherapy does not produce sufficient regression of the tumour to warrant delay in operating. Irradiation is, however, given to inoperable tumours and post-operatively to operable ones. A careful watch is kept on the leucocyte count which tends to decrease rapidly.

*Prognosis.* Figures for survival over three years vary from 17 per cent.

(R. Phillips, 1953) to 29 per cent. (Gross, 1953). As there is a natural tendency for *some* of these tumours to regress, the effects of any particular line of treatment are difficult to assess.

**Ganglioneuroma.** This is a rare relatively benign tumour, usually found in the paravertebral region of the abdomen, chest, or neck. Like the neuroblastoma, it arises from the sympathetic chain or the adrenal medulla. It is spherical, and the cut surface is homogenous. Microscopically, it consists of well-differentiated ganglion cells and nerve fibres. Occasionally a ganglioneuroma is dumb-bell shaped, partly lying inside and partly outside the spinal canal. It is thought that this bilobed distribution results from neoplasia developing in primitive sympathetic nervous tissue before the ganglion cells have completely migrated laterally from their site of origin in the neural crest, rather than by growth of the tumour into the spinal canal.

Clinically, a ganglioneuroma may present as an abdominal swelling near the midline, or may be observed in radiographs, lying in the posterior mediastinum. They are occasionally calcified. Enlargement of the neural foramen occurs with the dumb-bell-shaped tumour, and neurological manifestations may arise from pressure on the spinal cord.

Excision is the best treatment. Both the intra- and extraspinal parts of dumb-bell tumours should be removed. Recurrence is infrequent.

## CHAPTER 61

### THE PERIPHERAL NERVES

#### INJURIES

**Modes of Injury.** Missiles are responsible for the large majority of nerve injuries in wartime. Nerves may be damaged directly by missiles, or indirectly by the disturbance created in surrounding tissues by high velocity missiles. Fractures and dislocations are responsible for the majority of injuries in peacetime. Incised wounds (by stabbing, accidental cuts with knives or glass) also occur. Less common injuries to nerves arise from:—(a) Traction; (b) deep burns; (c) long continued minor trauma; (d) persistent pressure; (e) vascular occlusion.

**Effects of Injury (Seddon's Classification).** *Neurapraxia* is the term applied to temporary cessation of function without anatomical interruption of axons, as may result from the application of a tourniquet to a limb (myelin sheath only damaged). The sensory disturbance is subjective (tingling, pain or feeling of numbness) with little or no objective sensory loss and no effect on autonomic function. *Axonotmesis* applies to interruption of axons without damage to their connective tissue sheaths. *Neurapraxia* and *axonotmesis* are known as lesions in continuity. *Neurotmesis* is the term for complete disruption of a nerve including its sheath; sometimes there is disruption of only part of a nerve. There is no clinical means of distinguishing between *axonotmesis* and *neurotmesis*.

**Sequels of Injury.** *Perfect* recovery occurs only after *neurapraxia*. *Wallerian degeneration* follows in part of, or in the whole of the nerve, in all other injuries:—At first there is a profuse increase in the number of neurilemmal Schwann cells in an attempt to bridge the gap. Then, from the site of division of the nerve to the periphery and also for a short distance along the proximal segment of the nerve as far as the first nodes of Ranvier, the myelin sheaths break up into globules; the neurilemmal Schwann cells proliferate, and the axons degenerate. The Schwann cells assist in the phagocytosis of disintegrated myelin and axons. Motor and sensory fibres go through the same process. The nerve cells from which degenerating fibres arise undergo Nissl's degeneration (the cells become swollen and rounded and the nuclei eccentric; the Nissl granules disappear). This process is reversed in about three weeks in injury of lower motor neurons, but is permanent in injury of upper motor neurons. *Wallerian degeneration* can be demonstrated histologically by Marchi's method which stains degenerating myelin black. Collagen is deposited in the muscles, obliterating the sheaths of the nerve fibres and causing

(R. Phillips, 1953) to 29 per cent. (Gross, 1953). As there is a natural tendency for *some* of these tumours to regress, the effects of any particular line of treatment are difficult to assess.

**Ganglioneuroma.** This is a rare relatively benign tumour, usually found in the paravertebral region of the abdomen, chest, or neck. Like the neuroblastoma, it arises from the sympathetic chain or the adrenal medulla. It is spherical, and the cut surface is homogenous. Microscopically, it consists of well-differentiated ganglion cells and nerve fibres. Occasionally a ganglioneuroma is dumb-bell shaped, partly lying inside and partly outside the spinal canal. It is thought that this bilobed distribution results from neoplasia developing in primitive sympathetic nervous tissue before the ganglion cells have completely migrated laterally from their site of origin in the neural crest, rather than by growth of the tumour into the spinal canal.

Clinically, a ganglioneuroma may present as an abdominal swelling near the midline, or may be observed in radiographs, lying in the posterior mediastinum. They are occasionally calcified. Enlargement of the neural foramen occurs with the dumb-bell-shaped tumour, and neurological manifestations may arise from pressure on the spinal cord.

Excision is the best treatment. Both the intra- and extraspinal parts of dumb-bell tumours should be removed. Recurrence is infrequent.

blocking intact nerves with procaine (deeply placed nerves are first located by means of a needle electrode).

**Vasomotor and Trophic Changes.** Such changes commonly follow injuries of those nerves giving a large cutaneous supply to the hands and feet (e.g. the median and internal popliteal nerves). For the first three weeks after injury, the part is always warm because of vasodilatation, but later, patients may complain of coldness of the affected area which may or may not show a corresponding change in temperature. The effect is not accurately related to the area of sensory loss, and may even involve

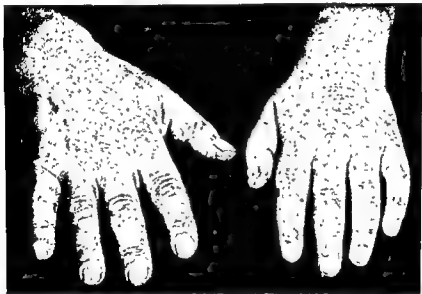


FIG. 417. The patient's left hand was smooth, glossy and swollen; the curvature of the nails was increased and their growth had ceased. These effects were caused by a long-standing total brachial plexus injury.

the whole of the hand or foot. Cyanosis and redness is very common following nerve injuries and usually corresponds exactly with the area of analgesia (loss of sensation to pain). Pallor occurs infrequently. After long-standing denervation the skin usually becomes smooth and in some extreme cases has a glossy appearance (Fig. 417). Ulceration of the skin may affect the fingers or the toes, especially under the first metatarsal head where the bone may become exposed. Thus a classical *perforating ulcer* is produced. Obvious precipitating factors exist such as unconscious burning of the fingers with cigarettes, or constant pressure of footwear. Nevertheless, the absence of sensory supply is the factor responsible for the chronicity of these lesions. There is often atrophy of the subcutaneous tissue of the fingers and toes; brittleness, increased curvature, and slow growth of the nails; and paronychia (infection of the nail bed).

atrophy of the muscle spindles. After about a year, this process is so advanced that the chances of a successful nerve repair are greatly diminished.

**Signs of Nerve Injury.** A detailed neurological examination must be carried out on all injured limbs (the physical signs may be misleading if the limbs are cold). This examination is important legally as well as surgically. Nerve injuries are recognised by loss of motor activity in the muscles supplied by the affected nerve. Examination of individual muscles or groups of muscles is required in much greater detail than for suspected upper motor neuron lesions. Muscles should be both observed and palpated during attempted contraction. The same special care is also necessary in testing for sensory loss. The variation and overlap of the sensory supply of adjacent nerves is often considerable so that the area of sensory deficiency is frequently smaller than might be expected. *Tinel's sign* is now restored to favour although it is not reliable during the first three to four months after injury; digital percussion along the regenerating nerve causes a tingling sensation referred to the peripheral distribution of the nerve when the point of the advancing sensory axons is reached. Tendon jerks may be diminished or absent in nerve injuries (e.g. the ankle jerk after damage to the sciatic nerve). The power of each muscle or group of muscles is assessed according to the *Medical Research Council's* scheme: 0 = total paralysis; 1 = slight contraction; 2 = contraction with gravity eliminated; 3 = contraction against gravity; 4 = contraction against gravity and slight resistance; and 5 = normal contraction against powerful resistance. It is most important to have this base line for comparison of muscular power following either conservative or operative treatment.

**Anatomical Anomalies.** Variations in the nerve supply of the muscles of the hands are frequent. Thus flexor pollicis brevis is just as often supplied by the ulnar nerve as by the median, and may be supplied by both. Occasionally all the thenar muscles are supplied by the ulnar nerve; or the adductor pollicis, with or without the first dorsal interosseous muscle is innervated, together with the other thenar muscles, by the median nerve. The clinician should be on his guard against "trick movements" which may develop within a few weeks of injury. No attempt at deceit on the part of the patient is implied by this term. Thus full flexion of the wrist in a patient with radial nerve palsy causes some extension at the metacarpophalangeal joints due to tension created in the extensor tendons. The force of gravity may aid or produce trick movements. Sometimes the function of paralysed muscles is partially taken over by other normal muscles, e.g. when the usual flexors of the wrist are paralysed in median and ulnar nerve paralysis, abductor pollicis longus may flex the wrist; or the flexors of the forearm may flex the elbow when the brachioradialis, biceps and brachialis are paralysed. Anomalous nerve supplies and trick movements may be checked by

is dusted over the skin with a wad of cotton wool which should be pressed firmly on to the skin so that the sweat-ducts are filled with powder. The normal sweating skin becomes purple, leaving the denervated area uncoloured. The powder can then be washed off with warm water and soap. Care must be taken to avoid soiling the patient's clothes and the bed covers.

The validity of an area of sensory loss from a peripheral nerve lesion may also be tested by intradermal injection of histamine.\* Skin deprived of its peripheral nerve supply does not give the spreading flare (part of the triple response of Lewis) if the test is done three weeks or more from the time of injury—i.e. after Wallerian degeneration has taken place and the axon reflexes on which the triple response depends therefore no longer function.

**Muscle Biopsy.** This diagnostic method is sometimes employed to aid the surgeon in deciding for or against exploration of an injured nerve. A piece of muscle, about a cubic centimetre in size, is excised well away from the point of entry of the nerve supply of the muscle, and fixed on a piece of cork with pins. The specimen is then immediately fixed in formol-saline. Later, various stains are used to demonstrate muscle spindles, connective tissue, blood vessels, and nerves and their terminal branches. If the muscle is thus shown to be completely degenerated, operation on the injured nerve would be futile, whereas if there are well-preserved muscle spindles and open nerve sheaths, operation may be successful. The presence of normal muscle and intact nerve fibres indicates regeneration; this state of affairs may exist in the absence of *functional* recovery. Abnormal muscle with normal nerve fibres is found in *muscle dystrophies*. Muscle biopsy is not a routine procedure, but is indicated when nerve repair has been delayed longer than two years.

**Nerve Biopsy.** This procedure may be necessary when inspection of the site of injury fails to indicate the nature of the lesion. An unimportant branch is excised or a small length of nerve fibres may be removed from the main trunk. Myelin sheaths are best demonstrated in transverse sections, and axons in longitudinal sections. The presence of axons distal to the injury will influence the surgeon against further action. Some injuries in *continuity*, e.g. those caused by ischaemia, traction, or the accidental injection of injurious drugs, may have little or no palpable swelling to indicate the extent and severity of the injury. It is in these circumstances that nerve biopsy is of value.

### TREATMENT OF NERVE INJURIES

**A. Closed injuries** (e.g. tourniquet palsy; paralysis in closed fractures and dislocations; paralysis following injection of drugs into or close to a nerve; and paralysis due to prolonged posture on the operating table).

\* Histamine acid phosphate = available in ampoules containing 0.5 mg. in 0.5 ml. solution; 0.1 ml. of this solution (i.e. 0.1 mg.) is injected intradermally.



**Causalgia.** Severe, persistent, burning pain in the palm of the hand or the sole of the foot may follow within twenty-four hours of a peripheral nerve injury. The pain is aggravated by emotional stimuli, and the threshold to other stimuli, especially heat, is lowered. It is often relieved by wet applications. The patient is not kept awake by the pain. Causalgia is usually caused by missile wounds under the conditions of warfare. The nerve injuries are usually *incomplete*. Causalgia has been described following injuries to the brachial plexus, and the median, sciatic, medial popliteal and posterior tibial nerves. The injury to the nerve occurs above the elbow or above the knee and the syndrome is more likely to develop when more than one nerve is injured. It is rarely produced by sharp instruments. The pain comes on too quickly for infection to be regarded as a causal factor. Sympathectomy (cervico-thoracic for the upper limb, and lumbar for the lower) is often effective. There is a tendency, however, towards spontaneous recovery.

**Reaction of Degeneration (R.D.).** Nowadays a current of *short duration* and *high intensity* is used instead of faradism and a current of *long duration* and *low intensity* is used instead of galvanism. The terms "faradism" and "galvanism" have, however, been retained to indicate roughly the intensity and duration of the current employed. The currents now used are produced by electronic apparatus. Muscle with an intact nerve supply responds to "faradism" and "galvanism" whereas muscle with its nerve supply interrupted long enough for Wallerian degeneration to have occurred (two to three weeks) does not respond to "faradism" of tolerable intensity, and its response to "galvanism" is slow and localised around the point of stimulation as compared with the quick diffuse response of normally innervated muscle. These electrical phenomena observed in denervated muscle are known as the reaction of degeneration (R.D.). The classical polarity formula (C.C.C. > A.C.C.\* for normal muscle, and A.C.C. > C.C.C. for denervated muscle) has been discarded along with the electrical apparatus formerly used as stimulators.

**Electromyography.** Action-currents in muscles (detected on a loud-speaker and cathode-ray oscilloscope by means of implanted electrodes and an amplifier) are the earliest signs of arrival of regenerated axons in muscle-end plates.

**The Sweat-test.** The area of sensory loss may be checked by the sweat-test which is based on the fact that sudomotor paralysis exists in the area of cutaneous sensory loss. Quinizarin powder is nowadays used for this purpose instead of Minor's starch and iodine mixture.

The patient takes 10 gr. of aspirin with a hot beverage, and is then placed under a radiant heat cradle for half an hour. Quinizarin powder †

\* The muscle reaction to the cathode is greater than to the anode when the circuit is closed or, in other words, the cathode closing current (C.C.C.) response of the muscle is greater than the anode closing current (A.C.C.) response.

† The hygroscopic sodium salt of quinizarin (35 gm.) is mixed with powdered sodium carbonate (30 gm.) and rice starch (60 gm.). The sodium carbonate helps to keep the mixture dry.

cutaneous bony prominences. The incision for exploring nerves high in the upper limb avoids the axilla and follows the lateral border of the pectoralis major, so that when adduction of the shoulder is necessary to bring the nerve ends together they are still accessible. The incision for exploring the upper reaches of the sciatic nerve follows the course of the nerve in the thigh, but at its proximal end curves well laterally over the buttock to allow easy access to the insertion of gluteus maximus which may have to be detached. Dissection of the nerve is first carried out through normal tissue proximal and distal to the injured region. Before it is freed, the nerve is stimulated with a weak faradic current on both sides of the site of injury to verify the location of the nerve lesion and to determine the presence or otherwise of intact axons in the distal segment.

**Appearances at Operation.** (a) The nerve may look and feel normal. This indicates complete or incomplete *axonotmesis* (division of axons with intact sheaths)—not *neurapraxia*, for this would recover too quickly for exploration to be indicated. (b) The nerve may be trapped between the fragments of a fracture. (c) There may be a *fusiform neuroma*. The larger and harder the *neuroma*, the greater is the likelihood of serious damage. More often than not, the information obtained before and during operation leads to no interference. In borderline cases, *trial section* is carried out. The hardest part of the neuroma is incised transversely until any nerve bundles are identified. If more than half the cross-section of the nerve is replaced by scar tissue, resection is performed. (d) The affected segment of a nerve may feel hard although it is of normal or diminished width. This state is caused by *intra-neural fibrosis* which may be localised, as after damage from an injected drug, or diffuse, as after a traction injury. The motor axons suffer more than the sensory ones so that a sensory response does not necessarily indicate a good prognosis. A positive motor response does, however, contraindicate resection. If there is no motor response, *trial section* (*vide supra*) is indicated, and muscle and nerve biopsy may help. (e) A *lateral neuroma* may be discovered. If two-thirds of the nerve are affected, or if the remaining intact portion supplies relatively unimportant muscles or skin, total resection is indicated. (f) The injured nerve may present a dumb-bell appearance, i.e. the swelling on the proximal segment of the nerve (the "*neuroma*") is connected by a central strand to the swelling at the end of the distal segment of the nerve (the "*glioma*"). This central strand is almost certainly entirely fibrous. Resection and anastomosis should be carried out. (g) The nerve may be completely divided. Again resection and anastomosis should be done. (h) Paralysis due to compression of a nerve by fibrous tissue is a theoretical possibility, but in practice is very rare. Freeing such a nerve is termed *neurolysis*. When a nerve is adherent to, and compressed by, a traumatic aneurysm, good results may follow treatment of the latter. (i) A large gap may be found between the ends of a nerve. One of the following methods is then used to approxi-

If a response to "*faradism*" is obtained after three weeks from the time of injury, *neurapraxia* (damage to myelin sheath only) has occurred, and a rapid spontaneous recovery is to be expected. No response to "*faradism*" after three weeks, with an altered response to "*galvanism*" (reaction to degeneration—R.D.) indicates nerve degeneration. Regeneration will occur if there is *axonotmesis* (degeneration of axons with intact nerve sheaths), but it will not occur if the nerve has been completely divided (*neurotmesis*). In closed injuries it is usual to wait several months before exploration because spontaneous recovery commonly occurs. However, the longer the delay in cases of complete division (*neurotmesis*) the less the chance of successful suture. Pain and progressive weakness are indications for exploration. After one year the chance of success is very much reduced. After two years it is negligible, for muscle degeneration has almost certainly occurred, and nerve sheaths have become obliterated. The rate of regeneration of axons (allowing three weeks for it to begin) varies from 2 to 4 mm. a day, and is one factor to be taken into account. Tinel's sign is useful after the third to fourth month from the time of injury, although *electromyography* gives the earliest evidence of recovery. Explorations should not be delayed more than six months; they are usually carried out earlier in cases of doubt.

**B. Open injuries** (e.g. missile, knife and machine wounds). In open injuries the nerve is usually completely or partly divided. If paralysis persists after a wound has healed—unless, of course, satisfactory suture has been previously carried out—exploration should be undertaken.

**Splinting.** Splints are used to:—(a) Compensate for paralysis and thus to encourage the use of the limb; (b) correct deformities; (c) obtain immobilisation; (d) elevate a limb to prevent œdema and therefore fibrosis. Splints in common use are described under specific nerve injuries.

### OPERATIONS ON PERIPHERAL NERVES

*At the primary operation on an open injury, divided nerve ends should be approximated by a suture through the whole thickness of the nerve to prevent retraction and therefore facilitate later repair. The latter is usually carried out as an early secondary operation when the wound is completely healed (usually in three to six weeks from the time of injury). According to Seddon, this delay is of fundamental importance, for even when an injury is produced by a sharp knife, damage is inflicted on the nerve ends, although it is not apparent immediately, but is revealed by subsequent fibrosis observed at a secondary operation. Furthermore, mobilisation of the nerve ends to bridge a gap can be done with greater safety when the risk of sepsis is eliminated by treatment, or the passage of time.*

**Exploration.** An incision is made along the course of the nerve extending well above and below the site of injury and avoiding any sub-

Therefore the plasma clot technique is employed:—A trough is formed around the graft and nerve ends with fibrin foam, and then a freshly prepared solution of fibrinogen and thrombin is run on to the junction. While the clot is forming the graft is held in contact with the recipient nerve with fine forceps. If, however, a main trunk nerve has been used as a graft, it is sutured in the usual way. Grafting should be used also for gaps in partially divided nerves. Grafting of gaps in the radial nerve is not required because of the good results of tendon transplantation.

**Physiotherapy and Rehabilitation.** One of the main objects of splinting is to enable the patient to make the maximum use of the remaining function of the limb. Occupational therapy and games should be organised to this end and also to maintain the active interest of the patient. *Joints that cannot be moved actively are moved passively several times a day to prevent stiffness.* Galvanism is useful for maintaining the blood supply of small muscles, and faradism for stimulating recently re-innervated muscles. Results of nerve suture and grafting offer a 50 per cent. chance of useful recovery provided the above measures are conscientiously carried out.

### LESIONS OF SPECIFIC NERVES

**The Phrenic Nerve (C.3, C.4, C.5).** Crushing this nerve to paralyse one half of the diaphragm in certain cases of pulmonary tuberculosis is not recommended (Chap. 41). It may be accidentally injured during operations on the neck. Sometimes it is affected by malignant glands in the neck or mediastinum. *Intrathoracic tumours and aneurysms may paralyse the phrenic nerve.* The site of origin of the phrenic nerve in the spinal cord may be affected in poliomyelitis or by tumours of the spinal cord. Unilateral paralysis of the diaphragm usually causes some degree of dyspnoea; bilateral paralysis causes serious dyspnoea. Radiography shows a high diaphragm on the paralysed side, but screening (fluoroscopy) is necessary to confirm that the diaphragm is paralysed. Sometimes X-rays taken for other purposes reveal an unsuspected paralysis of half of the diaphragm (usually on the left) without apparent cause. This phenomenon is termed *eventration of the diaphragm*. *Hiccup* is caused by spasm of the diaphragm and glottis. In addition to toxic states (e.g. uræmia), local irritation may be the cause (e.g. excessive gas in the stomach, inflammation above or below the diaphragm, or malignant glands in the mediastinum).

**The Brachial Plexus.** The normal origin is from the anterior rami of spinal nerves C.5 to T.1. Sometimes the origin is prefixed (C.4 to C.8), or postfixed (C.6 to T.2). The anterior rami of the nerve roots are behind scalenus anterior; the trunks of the plexus lie between the lateral border of scalenus anterior and the clavicles; the divisions of the trunks are behind the clavicle; and the cords are in the axilla.

*Missiles* may cause a partial or complete injury of the brachial plexus.

mate the ends:—(1) The nerve ends are mobilised by extensive dissection along the limb in both directions ; (2) *moderate flexion* of joints is maintained by plaster of Paris and then gradually released to stretch the nerve ; (3) changing the course of the nerve, e.g. transposition of the ulnar nerve in front of the elbow ; (4) *shortening bones* (rarely) ; (5) nerve grafting ; (6) *bulb-suture* in which the proximal "neuroma" and distal "glioma" are attached by sutures with the limb in flexion and gradual extension of the joint is carried out, followed later by excision and suture of the nerve ends.

### TECHNIQUE OF NERVE REPAIR

Nerves must be handled with the utmost care. Therefore fine dissecting forceps are required, and their use should be confined to holding the nerve sheath (*epineurium*). Retraction of nerves is accomplished with wet tapes. Very sharp instruments are required for excising neuromas and trimming nerve ends. A new safety-razor blade is commonly employed ; it may be held between the thumb and the index finger of the operator or in a Spencer-Wells' forceps. After a neuroma has been excised, or when a gap in a nerve has been closed by one of the methods listed in the previous paragraph, thin slices are cut off the nerve ends until all fibrous tissue has been removed and nerve bundles are clearly seen. A magnifying glass is useful at this stage. Care is taken to avoid turning the nerve in its long axis so that corresponding nerve bundles are brought as near together as possible. The nerve ends are approximated by fine sutures placed through the nerve sheath (*epineurium*)—*not* through the nerve. A suitable suture material is 0000 silk or 00 thread, supplied attached (swaged) to curved atraumatic needles. While the nerve ends are cut and sutured, they are steadied on a wooden tongue depressor. Whenever possible, the dissection and suture are done in a bloodless field obtained with the aid of a tourniquet. The pressure must be released after the nerve has been sutured, in order to avoid the risk of a hæmatoma at the site of suture. It is unwise to keep the tourniquet in place for longer than two hours.

**Nerve Grafting.** A gap of more than 6 cm. should not be closed by flexion of a joint and subsequent stretching by extension, for, however slowly this is carried out, the nerve will be severely damaged. *Autogenous* nerve grafts should be used. Results of grafting now closely approach those of simple suture (Seddon). The following are suitable donor nerves:—(a) The medial cutaneous nerve of the forearm in its course *above* the elbow ; (b) the posterior cutaneous nerve of the forearm ; (c) the sural nerve ; or (d) the saphenous nerve *in the thigh*. Larger nerves tend to undergo necrosis when grafted. The graft, whether composed of one or multiple strands (in the latter case it is known as a *cable graft*), must equal in diameter the recipient nerve and must lie in healthy tissue. The graft should also be somewhat longer than the gap to be filled. The sheaths of the above donor nerves are not thick enough to take sutures.

Therefore the plasma clot technique is employed:—A trough is formed around the graft and nerve ends with fibrin foam, and then a freshly prepared solution of fibrinogen and thrombin is run on to the junction. While the clot is forming the graft is held in contact with the recipient nerve with fine forceps. If, however, a main trunk nerve has been used as a graft, it is sutured in the usual way. Grafting should be used also for gaps in partially divided nerves. Grafting of gaps in the radial nerve is not required because of the good results of tendon transplantation.

**Physiotherapy and Rehabilitation.** One of the main objects of splinting is to enable the patient to make the maximum use of the remaining function of the limb. Occupational therapy and games should be organised to this end and also to maintain the active interest of the patient. *Joints that cannot be moved actively are moved passively several times a day to prevent stiffness.* Galvanism is useful for maintaining the blood supply of small muscles, and faradism for stimulating recently re-innervated muscles. *Results of nerve suture and grafting offer a 50 per cent. chance of useful recovery provided the above measures are conscientiously carried out.*

### LESIONS OF SPECIFIC NERVES

**The Phrenic Nerve (C.3, C.4, C.5).** Crushing this nerve to paralyse one half of the diaphragm in certain cases of pulmonary tuberculosis is not recommended (Chap. 41). It may be accidentally injured during operations on the neck. Sometimes it is affected by malignant glands in the neck or mediastinum. Intrathoracic tumours and aneurysms may paralyse the phrenic nerve. The site of origin of the phrenic nerve in the spinal cord may be affected in poliomyelitis or by tumours of the spinal cord. Unilateral paralysis of the diaphragm usually causes some degree of dyspnoea; bilateral paralysis causes serious dyspnoea. Radiography shows a high diaphragm on the paralysed side, but screening (fluoroscopy) is necessary to confirm that the diaphragm is paralysed. Sometimes X-rays taken for other purposes reveal an unsuspected paralysis of half of the diaphragm (usually on the left) without apparent cause. This phenomenon is termed *eventration of the diaphragm*. *Hiccup* is caused by spasm of the diaphragm and glottis. In addition to toxic states (e.g. uræmia), local irritation may be the cause (e.g. excessive gas in the stomach, inflammation above or below the diaphragm, or malignant glands in the mediastinum).

**The Brachial Plexus.** The normal origin is from the anterior rami of spinal nerves C.5 to T.1. Sometimes the origin is prefixed (C.4 to C.8), or postfixed (C.6 to T.2). The anterior rami of the nerve roots are behind scalenus anterior; the trunks of the plexus lie between the lateral border of scalenus anterior and the clavicles; the divisions of the trunks are behind the clavicle; and the cords are in the axilla.

*Missiles* may cause a partial or complete injury of the brachial plexus.

High-velocity missiles injuring part of the plexus always cause a *temporary* paralysis of the whole limb. Injury of the motor root of T.1 produces Horner's syndrome (contracted pupil and narrowing of the palpebral fissure), for the efferent sympathetic fibres for the eye run in this root. Sometimes, presumably due to irritation of the nerve, the opposite syndrome (dilatation of the pupil and widening of the palpebral fissure) is found.

*Erb-Duchenne Palsy (C.5 and C.6).* This is produced by injury to the uppermost trunk of the brachial plexus. The following muscles are affected:—Supra- and infraspinatus, deltoid, biceps, brachialis, brachioradialis and supinator. If the plexus is postfixed, pronator teres and the radial extensors are also affected. Sensation is lost over the deltoid and the lateral aspect of the forearm. The posture of the limb is characteristic. It lies limply at the side, pronated and medially rotated with the hand directed backward, as though about to receive a surreptitious tip. Erb-Duchenne palsy may be found in the new-born infant. It is caused by traction on the head to deliver the shoulders, or traction on the shoulders to deliver the after-coming head in a breech delivery. This type of injury of the plexus may be caused by any mechanism producing forceful separation of the neck and shoulder. The clavicle may be fractured in the process, but the fracture is not itself the cause of the plexus injury. The high Trendelenburg position on an operating table may paralyse the upper part of the brachial plexus. Sometimes an upper trunk injury is associated with a traumatic false aneurysm or arteriovenous fistula. Compression by the aneurysm may be partly or completely responsible for the paralysis.

*Treatment.* Exploration is never required for birth palsy of the plexus, and is very rarely indicated for other injuries of the upper part of the brachial plexus, for the recovery rate is excellent in the whole group provided the upper limb is suitably splinted (the shoulder in abduction and flexion, and the elbow halfway between flexion and extension). Contracture of the biceps and loss of external rotation of the shoulder are thus prevented. In the presence of a traumatic aneurysm, paralysis may not be due directly to the missile, but to the pressure of the aneurysm, and can therefore be relieved by treatment of the latter.

*Klumpke's Palsy (C.8 and T.1).* This is caused by injury to the lowest trunk of the brachial plexus. The clinical picture resembles a combination of median and ulnar nerve palsies. Thus *all* the small muscles of the hand are affected and there is sensory loss along the ulnar border of the arm, forearm and hand. Horner's syndrome may also be present, for the sympathetic fibres to the eye pass out of the spinal cord in the motor root of the first thoracic nerve root. Klumpke's paralysis may be caused by traction on the upper limb or missile wounds. In some of the latter, there is an associated traumatic aneurysm which may be responsible for part of the neurological syndrome.

*Middle trunk palsy (C.7)* is similar to a radial nerve palsy, but there is sparing of the brachioradialis which is innervated by C.6. Sensation is impaired on the back of the forearm and radial part of the dorsum of the hand. This type of injury is usually caused by missiles. *Treatment.*—The patient should wear a radial nerve splint (Fig. 418). Those patients who do not recover are likely to be unsuitable for transplantation of flexor carpi radialis and flexor carpi ulnaris into the extensors, for the former muscles are innervated mainly by C.7.

All or any part of the plexus may be involved in injury, or in invasion

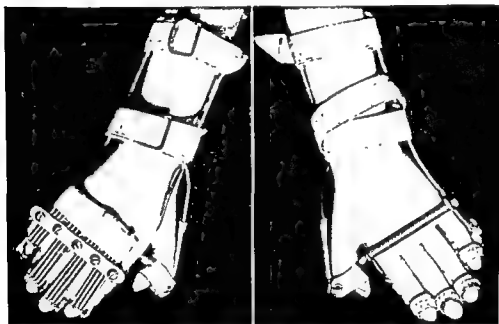


FIG. 418. The splint used for radial nerve or middle trunk paralysis. The springs on the back of the splint extend the digits and the transverse bar in the palm corrects the wrist drop.

by malignant glands. Tuberculous glands do not usually affect the plexus, but it is in danger during dissection of glands when they are adherent to surrounding structures. Large neurofibromas arising from cervical nerve roots can cause pressure on the brachial plexus. *Acute brachial "neuritis"* causes paralysis of the whole or part of the upper limb. It occurs suddenly with pain in the neck and shoulder, and later radiates to the periphery. The tendon jerks are absent, but there are no sensory signs. The cause is unknown. Recovery is usual.

**Cervical Rib.** Accessory ribs attached to the seventh cervical vertebra are fairly common. Sometimes they cause symptoms. The extra ribs may be complete and attached anteriorly to the sternum or the first costal cartilage. More often they are incomplete anteriorly where the pointed end is connected to the first rib by a *fibrous cord*. It is this fibrous



cord which is responsible for pressure effects on the brachial plexus. Hence the well-known paradox that when bilateral cervical ribs are present, symptoms are more likely to occur on the side of the smaller rib (i.e. the rib with the longer fibrous cord). The surgeon should be reluctant to attribute neurological effects to a complete cervical rib. Patients complain of paræsthesiæ in the fingers or pain radiating from the shoulder into the arm and along the ulnar border of the forearm. There is usually *some* paresis of all muscle groups of the upper limb, but those muscles supplied by the lowest trunk (C.8 and T.1) of the brachial plexus are the most severely affected. Thus *all* the small muscles of the hand are involved. Sensory loss may be found along the ulnar border of the forearm and on the ring and little fingers. The tendon jerks may be normal, diminished or absent. Trophic changes may affect the skin of the fingers. Sometimes, although there are sensory symptoms, there are no objective neurological signs. Pressure or friction of the fibrous cord on the subclavian artery may cause thrombosis of the vessel or, rarely, an aneurysm. Cervical ribs do not usually produce symptoms until adult life is reached.

**Operation.** The fibrous band is divided and part of the supernumerary rib lying behind the brachial plexus is nibbled away. The essential steps of the operation are as follows. An incision is made through skin and platysma 1 inch above and parallel to the clavicle from  $\frac{1}{2}$  inch medial to the lateral border of the sternomastoid to the trapezius. The external jugular vein is retracted or ligated. The omohyoid muscle is divided and the underlying pad of fat is dissected away to expose the brachial plexus lying behind the deep cervical fascia. The trunks of the plexus are freed from the fascia and retracted with wet tapes. The subclavian artery is exposed and retracted by the same method. The cervical rib and its fibrous band can be felt running caudally behind the brachial plexus in the lateral border of the scalenus medius. The band is divided and any part of the cervical rib lying in contact with the plexus is removed.

**Scalenus Medius Syndrome.** The effects produced by a cervical rib are sometimes caused by a fibrous band in the lateral border of scalenus medius. Recovery follows division of the lateral border of this muscle.

**Scalenus Anterior Syndrome.** It was formerly thought that the scalenus anterior could compress the brachial plexus, and that division of this muscle relieved the symptoms. Few surgeons nowadays believe in the existence of this syndrome.

**The Nerve to Serratus Anterior (C.5, C.6 and C.7).** This nerve passes downward behind the brachial plexus to supply the serratus anterior. It is in danger during radical mastectomy when the lymph glands are being cleared from the axilla. It may also be involved in lesions of the brachial plexus, including acute brachial neuritis. Paralysis of serratus anterior causes slight winging of the scapula at rest, and it is drawn towards the midline by the trapezius, latissimus dorsi and the rhomboids. During the



FIG. 419. Paralysis of the right serratus anterior muscle. The scapula is accentuated by the patient pushing against a wall. Note the scapula is drawn towards the midline by the unopposed trapezius, latissimus dorsi and rhomboid. (*J. G. Bonnin's patient.*)

movement of pushing forward, the winging is greatly increased (Fig. 419). In total paralysis, abduction of the shoulder is limited to 90 degrees, for when the movement is attempted, the scapula moves instead towards the vertebral column. Winging of the scapula is also caused by paralysis of the trapezius muscle (see Chap. 52).

**The Median Nerve** (C.6, C.7, C.8 and T.1). The following muscles are supplied by this nerve:—Pronator teres, flexor carpi radialis, palmaris longus, flexor digitorum sublimis, flexor digitorum profundus (also supplied by the ulnar nerve), flexor pollicis longus, pronator quadratus, abductor pollicis brevis, opponens pollicis, flexor pollicis brevis (also supplied by the ulnar nerve—sometimes entirely), and the first two lumbricals. The sensory distribution is shown in Fig. 420. The median nerve has no branches above the elbow. In median nerve palsy, the hand has a characteristic appearance: there is atrophy of the thenar eminence, and the thumb lies in the same plane as the palm (the simian hand). The median palsy splint (Fig. 421) allows adduction of the thumb and relaxes the paralysed thenar muscles. No splint is required if the thenar muscles happen to be supplied by the ulnar nerve.

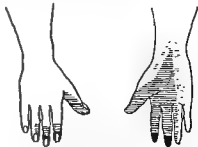


FIG. 420. The sensory distribution of the median nerve.



FIG. 421. Highet's splint for median nerve paralysis.

**The Carpal Tunnel Syndrome.** The median nerve may be subject to friction in the carpal tunnel. This may follow a fracture of the scaphoid bone (navicular), or fracture of the lower end of the radius; it may also occur without known cause. Symptoms appear in middle or old age and consist of burning or tingling in the hand and fingers, and weakness of abductor pollicis brevis and opponens pollicis. There is variable sensory loss in the median field of the hand. When the palmar cutaneous branch passes superficial to the transverse carpal ligament there is no sensory loss in the palm. Muscular wasting is confined to the *outer half* of the thenar eminence (the medial part is occupied by flexor pollicis brevis which is partly or wholly supplied by the ulnar nerve). There are no neurological signs above the wrist. The neuroma can sometimes be felt proximal to the flexor retinaculum (transverse carpal

ligament). **Operation.**—An incision  $1\frac{1}{2}$  inches long is made along the ulnar border of palmaris longus under which the median nerve is easily found. The distal end of the incision extends  $\frac{1}{2}$  inch into the palm so that the transverse carpal ligament can be completely divided along the course of the median nerve. If the diagnosis is correct, a swelling is observed on the median nerve partly beneath and partly proximal to the carpal ligament. The swelling may appear slightly inflamed. The median nerve *normally* widens considerably in this situation, but in the carpal tunnel syndrome the swelling is three-dimensional.

**The Ulnar Nerve (C.8 and T.1).** The muscles supplied by this nerve are as follows:—Flexor carpi ulnaris, flexor digitorum profundus (part also supplied by the median nerve), palmaris brevis, abductor, opponens and flexor digiti minimi, the third and fourth lumbricals, the palmar and dorsal interossei, adductor pollicis and flexor pollicis brevis. A few months after interruption of the ulnar nerve, wasting is visible between the metacarpals on the dorsum of the hand, and there is flattening of the hypothenar eminence. Abduction and adduction of the digits are lost and the fingers assume the characteristic posture of *main en griffe* or *claw hand* (Fig. 422). The posture of the hand results from the unopposed action of the antagonists of the paralysed muscles (the extensors extend only the metacarpophalangeal joints; the interphalangeal joints are extended by the interossei and lumbricals, and the lumbricals also flex the metacarpophalangeal joints; thus the ring and little fingers are extended at the meta-



FIG. 422. *Main en griffe* or claw hand.

carpophalangeal joints and flexed at the phalangeal joints, whereas the middle and index fingers are only slightly flexed at the phalangeal joints for the radial two lumbricals are usually supplied by the median nerve). The sensory loss in ulnar palsy is shown in Fig. 423. The "knuckle-duster" splint shown in Fig. 424 permits flexion and extension of the metacarpophalangeal joints.

**Traumatic Ulnar "Neuritis."** At the elbow the ulnar nerve lies in a bony groove between the medial epicondyle and the olecranon. In this situation it is sometimes subject to excessive friction. This may result from the following causes:—(a) The valgus position of the elbow resulting from faulty union of a fracture of the lateral condyle of the humerus—the



FIG. 423. Maximum sensory loss in ulnar nerve palsy.



FIG. 424. The "knuckle-duster" splint for ulnar nerve palsy.

ulnar nerve syndrome occurs some years after the injury; (b) an excessive valgus position of the elbow found in some people without a history of fracture (Fig. 425); sometimes recurrent dislocation of the nerve occurs. Symptoms may appear at any time; (c) fracture of the medial epicondyle of the humerus—ulnar nerve involvement occurs at the time of or soon after injury. In traumatic ulnar neuritis, there is burning or tingling (paræsthesiæ) along the ulnar border of the forearm and inability to perform fine movements, resulting from paralysis of the ulnar group of hand muscles. A strip of diminished sensation may be found along the ulnar border of the forearm. **Operation.**—Transposition of the nerve to the front of the elbow is carried out through an incision which follows the course of the nerve 3 inches above and below the elbow. The incision deviates forward a little to avoid the medial epicondyle. The slip of origin of flexor carpi ulnaris from the medial epicondyle is divided, and the nerve mobilised and transferred to the front of the elbow. The medial intermuscular septum of the humerus is divided if it kinks the nerve. Care is taken to preserve the branch to the flexor carpi ulnaris given off behind the epicondyle.

**Injury of the Deep Branch of the Ulnar Nerve.** Recurrent trauma to the palm, such as may follow the use of a spade, sometimes causes paralysis of the small muscles of the hand supplied by this branch of the ulnar

nerve. The branch tends to be injured after it has given off the supply to the muscles of the hypothenar eminence, which is therefore not wasted.



FIG. 425. Right-sided congenital cubitus valgus which was the cause of traumatic ulnar "neuritis."

### MUSCULAR WASTING OF THE HAND

Several syndromes have now been described in which wasting of the small muscles of the hand is a cardinal sign:—Brachial plexus injury of the Klumpke type (C.8 and T.1), acute brachial neuritis, cervical rib syndrome, scalenus *medius* syndrome, median nerve injury, carpal tunnel syndrome, ulnar nerve injury, traumatic ulnar neuritis and repeated trauma of the deep branch of the ulnar nerve. It remains to complete the picture by mention of other conditions which produce wasting of these muscles, and may therefore have to be considered in the differential diagnosis.

**Tumours of the Cervical Spinal Cord.** Intra- or extramedullary tumours in this situation frequently cause lower motor neuron effects in the upper extremity (Chap. 59).

**Syringomyelia.** This chronic disease is characterised by gliosis and cavity formation in the centre of the spinal cord (not an expansion of the

central canal as is sometimes thought). It begins most frequently in the cervical enlargement, and classically interrupts the spinothalamic fibres (conveying pain and temperature sensation) as they cross in the spinal cord. The other sensory fibres pass into the homolateral posterior column and escape destruction. Thus a *band of dissociated sensory loss*, corresponding to the extent of the lesion in the spinal cord, is produced on the surface of the body, and usually includes all or part of the upper limbs. The lesion also encroaches on the anterior horn cells and the pyramidal tracts, thereby causing muscular wasting in the upper extremities and eventually pyramidal signs in the lower.

**Amyotrophic Lateral Sclerosis** (Syn. Progressive Muscular Atrophy). The cause of this disease of the presenium is unknown. Degeneration affects principally the anterior horn cells and the cells of origin of the pyramidal tracts. The disease commonly begins in the cervical segments of the spinal cord and therefore causes wasting of the small muscles of the hand. Signs of involvement of the pyramidal tracts appear sooner or later. The classical physical sign is *fasciculation* of the muscles. There are rarely any sensory signs. Diagnosis is aided by electromyography. Chronic lead poisoning is sometimes the cause of progressive muscular atrophy (not to be confused with lead neuropathy).

**Occlusion of Neural Foramina.** Nerve roots may be compressed in their course through the intervertebral foramina of the cervical vertebrae by osteophytes growing from adjacent vertebral bodies. These changes may be localised to one or several intervertebral foramina without apparent abnormality of the rest of the vertebral column. Removal of the posterior wall of the affected canals and the osteophytes gives good results. Dental burrs and chisels are essential for this operation. Osteophytic occlusion of the intervertebral foramina may occur as part of spondylosis of the cervical vertebrae. The prominent transverse osteophytic ridges in this condition may also affect the spinal cord. Cervical disc protrusions (Chap. 59) do not usually affect the small muscles of the hand for the common sites of protrusion are between C.5 and C.6, and C.6 and C.7.

**Pancoast's Syndrome.** Pain in the shoulder radiating into the upper limb, wasting of the muscles of the hand and Horner's syndrome are sometimes caused by a bronchial carcinoma involving the apex of the lung. The upper ribs may be eroded, but there are usually *no* pulmonary symptoms.

**Neuropathy Associated with Carcinoma of the Bronchus.** Patients complain of numbness and ataxia. The limbs may show loss of all modalities of sensation and loss of tendon jerks, but the motor neurons are only rarely affected. Regression of the neurological syndrome may follow pneumonectomy.

Wasting of the small muscles of the hand may occur in many other neurological diseases, e.g. peripheral neuritis with its many causes, and

anterior poliomyelitis, but it tends to be overshadowed by the widespread effects of these diseases which are therefore not likely to cause difficulty in the differential diagnosis of surgical lesions. In leprous neuritis and progressive hypertrophic polyneuritis the nerves are enlarged and diagnosis can be confirmed by biopsy. *Muscular dystrophy* is not likely to cause difficulty in diagnosis because the proximal muscles are usually affected in this disease, but Gowers described a type in which the wasting begins in the small muscles of the hand. The muscle dystrophies occur mostly in children and run in families. Electromyography and muscle biopsy confirm the diagnosis. Wasting of the interossei is common in old age and also in association with rheumatoid arthritis. Wasting of these muscles on the opposite side of the body is sometimes found in lesions of the parietal lobe.

**The Radial Nerve (C.5 to T.1).** This nerve supplies the following muscles:—Triceps, anconeus, brachioradialis, extensor carpi radialis longus, extensor carpi radialis brevis, supinator, extensor digitorum, extensor digiti minimi, extensor carpi ulnaris, abductor pollicis longus, extensor pollicis longus, extensor pollicis brevis and extensor indicis. The sensory nerves are:—The posterior cutaneous nerves of the arm and forearm, the lower lateral cutaneous nerve of the arm and the dorsal digital nerves to the radial  $3\frac{1}{2}$  digits (variable). *Wrist-drop* is produced by interruption of the radial nerve. The sensory loss is often very slight; it may be confined to the skin on the dorsum of the hand between the first and second metacarpals, for there is overlap in the areas supplied by adjacent nerves. The radial nerve is most often injured in the spiral groove *after it has given off its branches to the triceps*. It may be injured by missiles, fractures of the shaft of the humerus, or inadvertent injection of noxious drugs. The *posterior interosseous nerve* passes round the neck of the radius in the substance of the supinator to reach the dorsal aspect of the inter-osseous membrane. It may be injured by missiles or by fractures of the *head of the radius*. The brachioradialis and extensor carpi radialis longus are unaffected and there is no sensory loss. *Wrist-drop* is again the characteristic posture. It is noteworthy that lesions of the middle trunk of the brachial plexus (C.7) and lead neuritis cause *wrist-drop*, also with sparing of the brachioradialis (C.6).

**Treatment.**—A radial palsy splint (Fig. 418) is fitted as soon as possible. Nerve injuries are explored according to the principles already discussed. Flexion of the elbow helps to approximate the nerve ends, but if this fails the nerve may be re-routed across the front of the humerus. Large gaps in the nerve are better treated by tendon transplantation than by nerve grafting (transplant pronator teres into extensor carpi radialis longus and brevis; flexor carpi radialis into abductor pollicis longus and extensor pollicis brevis; palmaris longus into extensor pollicis longus; and flexor carpi ulnaris into extensors digitorum communis, indicis proprius and digiti minimi).

**Digital nerves** are frequently injured in incised wounds and should be sutured. If a gap exists, grafting is carried out.

**The Lateral Cutaneous Nerve of the Thigh (L.2 and L.3).** This nerve passes to the thigh deep to the lateral end of the inguinal ligament; it penetrates the fascia lata and supplies the anterolateral aspect of the thigh. Friction at the point of penetration of the fascia sometimes causes numbness and pain in the region, and diminished or absent sensation. The condition is known as *meralgia paræsthetica*. Recovery follows incision of the fascia lata from the neural opening in the fascia to the inguinal ligament.

**The Femoral Nerve (L.2 to L.4).** The muscles supplied are iliacus, pectineus, sartorius and the quadriceps. It gives origin to the intermediate and medial cutaneous nerves and the saphenous nerve. Few lesions of the femoral nerve are seen, for missile wounds in this region usually cause fatal injury of the femoral artery.

**The Sciatic Nerve (L.4, L.5, S.1 to S.3).** The main trunk gives off the nerve supply to the hamstrings and to the adductor magnus (also supplied by the obturator nerve) and then divides into:—(a) The *medial popliteal nerve* (tibial nerve) supplying the muscles of the posterior compartment of the leg and the muscles of the sole of the foot through the medial and lateral plantar nerves; (b) the *lateral popliteal nerve* (common peroneal nerve) which divides into (1) the *musculo-cutaneous nerve* (superficial peroneal) supplying peroneus longus and peroneus brevis, and (2) the *anterior tibial nerve* (deep peroneal) supplying tibialis anterior, extensor digitorum longus, extensor hallucis longus, peroneus tertius, extensor digitorum brevis and the first dorsal interosseous muscle. The sensory areas are illustrated in Figs. 426 and 427. Injuries of the main trunk of the sciatic nerve are almost always incomplete. Total sciatic paralysis however occurs immediately, but after a week or two paralysis is found to be largely confined to the *lateral popliteal* distribution. Paralysis of the *lateral popliteal* nerve causes foot-drop with a steppage gait. There is loss of eversion and loss of sensation on the outer side of the leg and dorsum of the foot (Fig. 426). The lateral popliteal nerve is frequently injured because of its exposed position. The causes of injury are:—(a) Missile wounds in wartime; (b) fracture of the neck of the fibula in which an adduction strain may cause a serious traction injury of the nerve—a considerable length of the nerve is injured in this way; (c) incised wounds; (d) *tight plaster*.

**The Anterior Tibial Syndrome.** Acute painful swelling of the muscles of the anterior compartment of the leg sometimes occurs in healthy young men after unaccustomed exercise (e.g. in a recruit after his first route march). Arterial spasm is thought to be the cause. Loss of function is due to necrosis of muscles and ischæmia of the anterior tibial nerve (deep peroneal nerve). The extensor digitorum brevis which is outside the compartment is therefore paralysed, and there is loss of sensation in the





FIG. 426. Injury of the lateral popliteal nerve (cut with a piece of glass just above the head of the fibula). Note the foot drop and the wasting of the muscles of the anterior compartment. The area of sensory loss is due to interruption of the lateral cutaneous nerve of the calf (lined area in diagram), a branch of the lateral popliteal nerve; and interruption of the musculocutaneous nerve and the anterior tibial nerve (dotted area in diagram).

sible of both divisions of the obturator nerve are identified by faradism, and then divided. The origin of the adductor longus on the pubis is detached. *Flexor Spasm.*—An incision is made down the back of the thigh veering laterally at its distal end. The upper end of the incision reaches the inferior border of gluteus maximus. The sciatic nerve and its branches are found in the fat between the muscles. The branches are verified with a stimulator, and all those to the hamstrings are divided. Tendon lengthening is also usually necessary. The knee is immobilised in a padded plaster cast for several weeks. *Spasm of the Calf Muscles.*—A longitudinal incision is made in the popliteal fossa. About two-thirds of the nerve supply

first interdigital cleft (Fig. 427). Decompression of the muscular compartment by incision of the fascia has been suggested as a method of treatment. Injuries of the other nerves of the lower limb call for no special comment.

**Stöffel's Operation or Neurotomy.** This operation was designed to overcome adductor and flexor spasms in chronic spastic paraplegia (e.g. in Little's disease and disseminated sclerosis). Stöffel carried out partial division of the nerve supply of muscles in the main nerve trunks where, he claimed, the nerves of individual muscle groups could be identified by electrical stimulation. Nowadays the motor nerves are identified and divided after they leave the parent nerve trunks. *Adductor Spasm.*—An incision is made in the length of the adductor muscles. The adductor longus is retracted laterally, and as many branches as pos-



FIG. 427. The small area of skin supplied by the anterior tibial nerve (deep peroneal).

to gastrocnemius and one-third of the supply to soleus are divided. If there is restricted dorsiflexion of the ankle when the knee is extended, lengthening of the Achilles tendon should be carried out. Stabilising operations on the ankle and tarsal joints may also be required. The knee and ankle are immobilised in plaster for several weeks.

### TUMOURS OF PERIPHERAL NERVES

**Neurofibroma** (Syn. Schwannoma, Neurinoma and Neurilemmoma). This benign encapsulated tumour is well known as the acoustic nerve tumour and as one of the common types of extramedullary spinal tumours; it also occurs sometimes on peripheral nerves. This neurofibroma is not the tumour found in von Recklinghausen's disease, for it usually occurs singly, has a different histological appearance, and practically never becomes malignant. Microscopically, it consists of long slender cells with rod-shaped nuclei arranged in roughly parallel bands (pallisading). The site of origin is thought to be the fibrous part of the nerve sheath (epineurium) or the Schwann cells. Opinion is divided on this point. Radiating pain and paralysis of the muscles supplied by the affected nerve may occur when the tumour arises in a neural canal. Removal of the tumour is indicated for the relief of pain. The more peripheral symptomless tumours may be removed for æsthetic reasons, but there is some risk to function if the tumour is attached to an important motor or sensory nerve.

**Neurofibromatosis** (Von Recklinghausen's Disease). This disease is often familial. There are numerous tumours growing from small cutaneous nerves. These tumours are subcutaneous; they are often visible or can be palpated through the skin. Microscopically, they show no pallisading, and nerve fibres are distributed throughout the tumours. Thus they differ in at least two important respects from the solitary neurofibroma. Furthermore, the nerve tumours of Von Recklinghausen's disease may become malignant (fibrosarcoma). Neurofibromatosis may take place along the course of large nerves (*plexiform neurofibroma*), especially in the upper part of the neck where the affected nerves may be found in a pouch of skin. If this process is very extensive, it takes the name of familial hypertrophic "neuritis." Occasionally, a generalised thickening of a limb or part of a limb may occur (*elephantiasis neuromatosa*—one form of local gigantism). The grotesque appearance of some patients with Von Recklinghausen's disease is due to multiple intradermal tumours (*molluscum fibrosum*), which on histological examination often prove to be simple fibromas with no neural elements or connections.

Other types of tumours tend to occur in association with this disease, e.g. meningiomas, gliomas of the brain, gliomas of the optic nerves and chiasm, acoustic neurofibromas, spinal neurofibromas. Many patients have patches of brown pigmentation of the skin (*café au lait patches*).

**The Glomus Tumour.** Glomuses are microscopic structures found in the

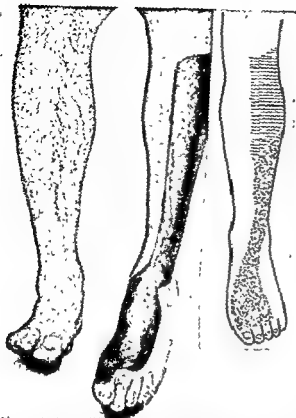


FIG. 426. Injury of the lateral popliteal nerve (cut with a piece of glass just above the head of the fibula). Note the foot drop and the wasting of the muscles of the anterior compartment. The area of sensory loss is due to interruption of the lateral cutaneous nerve of the calf (lined area in diagram), a branch of the lateral popliteal nerve; and interruption of the musculocutaneous nerve and the anterior tibial nerve (dotted area in diagram).

sible of both divisions of the obturator nerve are identified by faradism, and then divided. The origin of the adductor longus on the pubis is detached. *Flexor Spasm.*—An incision is made down the back of the thigh veering laterally at its distal end. The upper end of the incision reaches the inferior border of gluteus maximus. The sciatic nerve and its branches are found in the fat between the muscles. The branches are verified with a stimulator, and all those to the hamstrings are divided. Tendon lengthening is also usually necessary. The knee is immobilised in a padded plaster cast for several weeks. *Spasm of the Calf Muscles.*—A longitudinal incision is made in the popliteal fossa. About two-thirds of the nerve supply

first interdigital cleft (Fig. 427). Decompression of the muscular compartment by incision of the fascia has been suggested as a method of treatment. Injuries of the other nerves of the lower limb call for no special comment.

**Stöffel's Operation or Neurotomy.** This operation was designed to overcome adductor and flexor spasms in chronic spastic paraplegia (e.g. in Little's disease and disseminated sclerosis). Stöffel carried out partial division of the nerve supply of muscles in the main nerve trunks where, he claimed, the nerves of individual muscle groups could be identified by electrical stimulation. Nowadays the motor nerves are identified and divided after they leave the parent nerve trunks. *Adductor Spasm.*—An incision is made in the length of the adductor muscles. The adductor longus is retracted laterally, and as many branches as pos-



FIG. 427. The small area of skin supplied by the anterior tibial nerve (deep peroneal).

## SECTION VI

### THE FACE, MOUTH, LIPS, TONGUE AND JAWS

by

VALENTINE A. J. SWAIN

#### CHAPTER 62

#### DISEASES OF THE FACE

**Congenital Abnormalities.** *Congenital Cysts and Sinuses of the Face.*—These are liable to occur along the lines of fusion of the elements of the face. An *external angular dermoid* is the commonest. This smooth, cystic swelling lies under the skin in the region of the outer canthus of the eye against the floor of the temporal fossa, where it makes a small depression on the bone (Fig. 428). The cyst generally increases in size, but never involves the skin and never becomes inflamed (cf. sebaceous cyst). Excision of the cyst completely cures the condition. The cyst is usually thin-walled and contains white sebaceous material within it. A *preauricular cyst* characteristically appears as a round swelling in front of the ear near the crus of the helix, and is believed to be caused by the inclusion of dermal elements during the fusion of the tubercles of the developing ear. A small sinus opening is also usually seen on the helix and may extend into the cyma conchæ. Recurrent infection is prone to occur and give rise to an abscess which may discharge on the surface leaving a chronic sinus (Fig. 429). Excision of the tract including the cyst wall cures the condition. If infection is present, it is advisable to wait for this to subside before undertaking radical treatment. Incomplete removal is followed by recurrence. A *preauricular cyst*



FIG. 428. External angular dermoid cyst.

skin and are composed of connecting channels between arterioles and venules together with long bundles of nerve fibres. Their function is unknown. A glomus occasionally enlarges to form a benign tumour which causes spasms of agonising pain. The tumours are rarely larger than a pea, and occur only in the extremities, especially under the finger nails. The pain is relieved by excision of the tumour.

**Neural Fibrosarcoma.** This is a rare malignant tumour arising from nerve sheaths. It is composed of spindle-shaped cells with darkly staining nuclei. The cells are surrounded by variable amounts of collagen and reticulin fibres. The tumour tends to spread along the affected nerve trunk and separates the nerve bundles. It can be moved freely at right-angles to the nerve, but not longitudinally. Paresis, sensory loss and diminished or absent tendon jerks may be produced by these tumours when they occur in the limbs. (Note.—The peripheral neurofibroma—a benign tumour—does not produce neurological changes unless it arises in a neural canal.) The majority of the reported fibrosarcomas of nerves have occurred in patients with von Recklinghausen's neurofibromatosis. *Treatment.*—As soon as the diagnosis is established the limb should be amputated. These tumours do not respond to radiotherapy.

common familial deformity in which the ears protrude away from the sides of the head. It should be corrected by a plastic procedure on the pinnae.

**Inflammatory Lesions.** *Acute pyogenic infections* of the face vary in severity from a trivial lesion, such as a *boil*, to a large *carbuncle*. The oedema produced by the inflammation depends largely on the laxity of the subcutaneous tissue; thus marked and almost painless swelling occurs with infections in the periorbital region, where the cellular tissue is loose. In contrast, the swelling produced by inflammation on the back of the neck is tense and painful, for here the subcutaneous tissue is tight.

Infected abrasions and furuncles usually cause enlargement of the regional lymph glands either at the height of the infection or several days later (Fig. 431). Mostly, the lymphadenitis subsides with healing of the primary source of infection, but occasionally the glands suppurate. Treatment depends on the nature of the infection; small lesions should be kept clean and undisturbed, for they usually subside within a few days. More extensive infections should be treated by antibiotics, if there is little response to conservative measures (rest, sedatives and the application of local heat). Short-wave diathermy is of value. The possible association of a carbuncle with diabetes mellitus demands routine examination of the

urine for the presence of sugar. Pyogenic infections in the nose and upper lip can give rise to phlebitis of the angular vein leading to the serious complication cavernous sinus thrombosis (see Chap. 51). Occasionally, an apparently trivial lesion on the face or elsewhere may give rise to a systemic infection and cause a perinephric abscess (see Chap. 26), osteomyelitis (see Chap. 79), or septicæmia. Since the introduction of chemotherapy and antibiotics severe complications are rarely seen. *Chronic infections of the face* are more of dermatological interest than surgical. *Tuberculous ulceration*—often due to contamination of an abrasion with *M. tuberculosis*—is occasionally seen, and usually causes enlargement and suppuration of regional lymph nodes (Tuberculous Cervical Adenitis, Chap. 67). *Syphilitic infection* of the face may present as an extra-genital primary chancre, or as a secondary rash with lymphadenitis. Inflammation of the mucous membrane of the lips occurring in congenital or secondary syphilis may result in scarring at the angles of the mouth



FIG. 431. Showing lymphadenitis of the submandibular region secondary to an infected lesion on the upper lip.



FIG. 429. The scarring in the preauricular region is the site of a previous cyst. The sinus opening on the helix can be seen.

may be mistaken for a sebaceous cyst, or, if infected, for a suppurating preauricular lymph gland. The finding of a primary focus of infection favours the latter diagnosis. An *infra-auricular cyst* may arise in relation to a sinus which opens into the external auditory canal, and tracks downwards behind the angle of the jaw. It is liable to become infected and give rise to a chronic purulent discharge, either from the ear or below it. The anomaly is thought to arise from a vestige of the first branchial cleft. If the condition causes troublesome symptoms, the tract should be excised with due regard to the facial nerve and parotid gland to which it is closely

related. A *median nasal cyst* arises in the nasal septum and presents as a swelling over the bridge of the nose (Fig. 430). It occurs in childhood or early adult life. Infection of the cyst and the discharge of pus leaves a sinus on the crest of the nose, whence it can be traced downwards into the nasal septum. Radical treatment is necessary to cure the condition. The cyst wall should be exposed by splitting the nose in the midline and dissecting it from within the septum. Curetting of the sinus alone only allows temporary healing. This cyst must not be confused with a *meningocele* which also occurs in the midline, but a *meningocele* in this region usually lies over the inferior aspect of the frontal bone where a defect may be felt.

*Congenital lesions of the auricle*, such as absence or malformation of the pinna, due to agenesis of the cartilaginous tubercles, are occasionally seen. Auricular tags usually occur in the preauricular region as irregular, hard cartilaginous tufts covered with skin; they may occur anywhere along a line connecting the ear to the sternoclavicular joint. These outgrowths are thought to be vestiges of branchial cartilage. They should be removed for cosmetic reasons. "Bat's ears" are a fairly



FIG. 430. Congenital median cyst of the nose.

*Seborrhæic warts* occur on the scalp and face, and less commonly on the trunk, usually after middle age. These brown papillomas have a rough, greasy surface, and are attached at the base to normal skin. They are liable to injury, and so are best excised (under local anæsthesia). These warts are basal-celled papillomas ; they are benign and do not recur after removal.



FIG. 433. A classical illustration of rhinophyma as seen in the "Portrait of an Old Man and his Grandchild," by Domenico Ghirlandaio (1449-94). By permission of The Louvre, Paris (cliché Agraci).

*Rhinophyma* is a familial condition. It is caused by hyperplasia of the sebaceous elements of the skin, and gives rise to a cauliflower-like thickening of the nose (Fig. 433). The unsightly appearance can be remedied by plastic surgery. The affected skin is shaved off and the raw areas allowed to heal. A skin graft will be necessary if the nasal cartilage is exposed.

A *turban tumour* is a curious, rare, familial condition. It originates as a number of raised "tomato-like" masses on the scalp ; these slowly



(*rhagades*). *Actinomycotic infection* of the face is a rare condition and usually results from spread of inflammation from the jaw (see Chap. 65).

**Non-inflammatory Lesions.** A *sebaceous cyst (wen)* commonly occurs on the face, but more often arises on the scalp or back of the neck; they may be multiple. It presents as a smooth, rounded swelling covered with normal skin, the centre of which is partially attached to the cyst beneath (cf. dermoid cyst). Its lining consists of squamous epithelium and the cyst contains sebaceous material. It grows slowly. The cyst should be



FIG. 432. Multiple virus warts which were treated subsequently by electrocautery.

removed under local anaesthesia. A small incision is made over the cyst and the plane of cleavage in the subcutaneous tissue is opened up; in some cases it is possible to dissect around the cyst wall and to remove it intact; whereas in others, after opening the cyst and expressing its contents, its lining can be partly squeezed out and then separated from the surrounding tissue. Removal of the cyst is easier when the subcutaneous tissue is loose; thus those cysts situated on the back of the neck where the cellular tissue is dense are often difficult to remove. Sebaceous cysts are liable to become infected and ultimately discharge a mixture of sebaceous and purulent material. In these cases, the contents should be curetted out, and excision should be postponed till the inflammation has subsided. Then

it is important to remove all the lining lest a recurrence occurs. Occasionally malignant change occurs in a sebaceous cyst, and this should be treated as an epidermal carcinoma (*vide infra*). A *sebaceous horn* is a curious condition which arises by the slow protrusion and hardening of the sebaceous contents of a cyst. Removal of the underlying cyst cures the condition. *Simple virus warts (verrucae)* commonly occur on the face, mostly around the mouth, and are seen most commonly in childhood or early adult life (Fig. 432). They are thought to be due to a virus infection, and are commonly associated with warts on the fingers, and so may be spread by direct contact. They are squamous-celled papillomas. If they do not disappear spontaneously they are best removed by the electric cautery under local anaesthesia. Recurrences in the neighbourhood sometimes arise. Occasionally a pigmented wart may be mistaken for a melanoma.

of the regional glands. It may also resemble a basal-cell carcinoma, or keratosis of the skin. As molluscum sebaceum usually resolves without any treatment, radical methods are unnecessary. Resolution is, however, assisted by shaving off the swelling at its base. Radiotherapy and cauterization may also be used.

**Keratosis.** Prolonged exposure to sunlight is liable to produce hyperkeratosis of the skin. This appears as thick, roughened crusted areas on the exposed skin of the face, back of the hands and forearms. No special treatment is required, but areas showing infiltration or ulceration should be regarded with suspicion, for malignant change is liable to develop, therefore a biopsy examination may be necessary. A squamous-celled epithelioma or rodent ulcer may thus be found.

**Malignant Diseases of the Skin.** Next to the epithelioma, the commonest cancerous lesion on the face is the rodent ulcer. Malignant melanomas are also seen, and more rarely cancerous growths arise from sweat or sebaceous glands.

**Epitheliomas (Squamous-cell Carcinomas).** This condition is commonly seen on the face and arises in middle-aged and elderly people. It may occur in a pre-existing lesion (e.g. a chronic ulcer) or be aggravated by exposure to chemicals, such as tar or acids. Usually, however, epitheliomas develop *de novo*. They start as a small nodule which gradually breaks down leaving a shallow ulcer with heaped up edges around. At first the lesion is unattached to underlying tissues, but eventually deep infiltration occurs. Less often a papilliferous growth may develop (Fig. 435). Enlargement of the regional lymph nodes is common (cf. rodent ulcer). Radiotherapy is very effective in most cases, but lesions situated near cartilage or bone should be excised, for there is a risk of necrosis of these structures following irradiation. Lesions situated near the eye are also best excised, for the eyesight may be affected by radiotherapy. Skin defects resulting from excision of carcinomas of the skin are immediately skin-grafted. If the regional glands are involved, block dissection is necessary. The results of treatment by surgery or radiotherapy are good (90 per cent. are cured).

**Rodent Ulcers (Basal-cell Carcinomas).**—This condition commonly arises in old people, and is seen on the cheek especially in the region of the angle of the nose or eyes (Fig. 436). First a small pearl-like nodule appears and slowly breaks down, leaving an ulcer. Ulceration spreads very slowly and the lesion is commonly covered with a scab. Its margin is characteristically hard and rolled. Spread of the disease, if unchecked, may reach large proportions (terebant type) and gradually deep structures may be infiltrated, such as the eyelids and nasal cartilages. Occasionally a diffuse cicatricial type of lesion is seen, especially on the scalp. Rodent ulcers should be treated early, for they are curable by radiotherapy or excision; the former treatment is preferable and causes less inconvenience to the patient. Irradiation cures the majority of lesions, for 95 per

coalesce and ultimately envelope the head with an irregular growth. The tumour is derived from the sweat gland elements of the skin and spreads locally. It neither infiltrates nor ulcerates. The tumour is best removed. Afterwards, split-skin grafts are applied to the bare pericranium.

Other tumours, such as *hamangiomas*, *melanomas*, *neurofibromas* are commonly seen on the face; these conditions are discussed elsewhere in the book. *Lipomas* are occasionally seen on the face, or forehead, and are

characterised by their soft consistency and puckering of the skin when it is lifted away from the lobulated fatty tumour. This wrinkling is due to fibrous strands stretching between the fatty nodules below to the overlying skin. Lipomas may be solitary or diffuse; they should be excised if unsightly. *Lymphangiomas* may also occur.

*Molluscum Sebaceum* (syn. kerato-acanthoma, molluscum pseudocarcinomatousum). Since the description of this condition by MacCormack and Scarff in 1936, it has become recognised as a definite entity. It affects the elderly (sixty to seventy years old) of both sexes, in about equal proportions, and occurs mostly on the face, and



FIG. 434. Molluscum sebaceum. (Dr. J. Martin Beare's case, published in *The Lancet*.)

especially on the cheek (Fig. 434). Occasionally, this lesion appears on the ear, or rarely on the extremities. A single nodule develops. This is characteristically covered by a thin layer of skin through which friable keratinous material appears. It is usually about 1.5 cm. across. Regional lymph glands are not involved. The tumour is benign, and grows steadily for about eight to ten weeks to reach its maximum size, when it gradually resolves and finally disappears within six months or so of its onset, leaving a small, pale scar. The cause of the condition is unknown. It consists of a central mass with a keratinous core and is surrounded by a covering of normal skin, except at its summit. In most instances the condition cures itself spontaneously, but exceptional recurrences have been observed (M. Beare, 1955). In cases of doubt, a biopsy examination should be carried out, for it may resemble a squamous-celled epithelioma (*vide infra*), but the malignant lesion grows more slowly and may cause enlargement

of treatment and prognosis can best be decided. If the tumour is small, adequate local excision is preferable to "taking a piece for section."

### MELANOTIC SKIN LESIONS

These lesions commonly occur almost anywhere in the skin, and they are both common and conspicuous on the face. The majority are benign but the few which become malignant are invariably fatal. As the mode of pigment production in normal skin is obscure, their histogenesis is uncertain. It is thought that melanin, an iron-free pigment, is produced by specific epidermal cells called melanoblasts which lie just beneath the Malpighian layer of the skin. The melanoblasts give a positive "DOPA" reaction.\* Other pigmented branching cells lying in the dermis, called chromatophores, are melanophages; they contain melanin, but do not produce the pigment, and so give a negative "DOPA" reaction. Cells of many different origins are capable of producing melanin (e.g. in the choroid coat of the eye and the meninges). Therefore all melanin-producing tumours cannot be placed in one histogenetic group.

**Pigmented Nævi** (*Syn. Moles or Benign Melanomas*). They are usually brown, but may be black. They vary in size from a very small mole to an extensive pigmented area. The surface is usually flat, but may be irregular or warty. Some are hairy (Fig. 437). They continue to grow in childhood, but remain quiescent throughout the greater part of adult life. The pigmented nævus consists of collections of nævus cells (melanoblasts) which are small round, polygonal or oval cells with dark nuclei resembling the basal cells of the epidermis, and lie either at the junction of the epidermis and dermis (junctional nævus) or in the dermis (intradermal nævus). A lesion in which the pigment cells show junctional and intradermal distribution is termed a compound

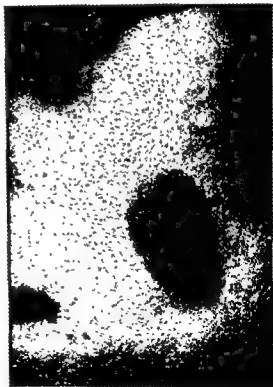


FIG 437. Hairy mole on face subsequently treated by excision.

\* The DOPA reaction: Dihydroxyphenylalanine (abbreviated to DOPA) reacts with an oxydase in certain cells in pigmented regions to form a dark substance chemically related to melanin. This reaction indicates the presence of cells which are true melanoblasts, i.e. containing the oxydase.



(a)



(b)

FIG. 433. (a) A squamous-celled epithelioma of the forehead before treatment. (b) After X-ray therapy. (Dr. H. Clive Sims' patient.)

cent. remain healed if the lesion has not involved bone or cartilage when first treated. Regional lymph glands are rarely involved.

**Diagnosis.** In the early stages, it is often difficult to distinguish between the various malignant lesions of the face, for it is only in the later stages that the rate of growth, the characteristic ulcer margin, and glandular involvement become apparent. Thus, early biopsy should be performed in cases in which the diagnosis is doubtful. Thereby the mode



FIG. 436. A rodent ulcer.

warning, "Beware the person with a glass eye and enlarged liver." Although the rate of spread is variable, the disease disseminates relentlessly and usually causes death within two to three years of its onset. The advance is more rapid in the younger age groups. Survivals of twenty to thirty years after removal of a primary tumour have been recorded, but such cases are extremely rare. Spontaneous regression of secondaries has also been recorded.

*Differential Diagnosis.* A seborrhœic wart, a pigmented basal-celled carcinoma or a sclerosing angioma may resemble a malignant lesion. A subungual hæmatoma closely simulates a melanoma. Biopsy is contra-indicated for it would cause metastasis. In doubtful cases, however, wide local excision followed by immediate examination of a frozen section can be used to confirm the diagnosis of malignancy before proceeding to removal of the regional lymphatic glands.

*Pathology.* The primary tumour is never large. Pigmentation is variable; some are not pigmented macroscopically, although melanin may be demonstrated by special staining with silver (Fontana's silver method). Microscopically the picture is most variable. There is proliferation and inequality in size of the nævus cells. Mitosis is frequently seen and there is often invasion of surrounding tissue. The histological distinction between a benign and malignant lesion may be difficult, and the decision places a great onus on the pathologist. The histological pattern is no guide to estimating the time of survival. Microscopy, as well as confirming the diagnosis, is also useful in assessing the thoroughness, or otherwise, of excision, and in demonstrating the presence of tumour cells in the regional lymph glands, although the absence of such cells does not necessarily indicate a good prognosis.

*Treatment.* When a melanoma is suspected it should be treated urgently and be widely excised, together with the regional lymphatic glands and, when possible, the deep fascia proximally and distally to the tumour. When the site of the lesion permits, the excision should be at least 1 inch (2.5 cm.) clear of the tumour and the deep fascia should be excised for 2 inches all round the tumour. The bare area is skin grafted if necessary. Damage inflicted on the tumour by frequent palpation and instruments tends to increase the danger of metastasis. Amputation of digits or part of the limb is necessary in peripheral lesions in addition to block dissection of the regional lymph glands. More extensive amputations, such as removal of the fore and hind quarter, are not recommended, for there is no evidence that the grave prognosis is influenced by such radical methods. Melanomas are relatively radio-resistant.

### INFECTIONS OF THE SALIVARY GLANDS

**Acute Epidemic Parotitis (Syn. Mumps).** This is a virus infection occurring commonly in childhood. The parotid gland is more often affected than the other salivary glands. It becomes swollen, and pain is

nævus. In early life, the masses of melanoblasts form in the lower layers of the epidermis and gradually migrate deeply into the dermis, when they reach maturity (Dawson, 1925). Thus, young immature pigmented nævi are flat and are mostly of the junctional type and show active proliferation; later they change to the compound variety and ultimately mature in the adult as a wholly intradermal lesion. This last variety is palpable and remains quiescent. It is generally accepted that pigmented moles are malformations of the skin, and nævus cells are epidermal rather than neuronal in origin. Abnormalities of nerve fibres are sometimes associated with these lesions.

*Treatment.* Treatment is usually unnecessary, for the vast majority of pigmented nævi remain innocent, but those lying exposed to irritation or injury should be removed lest repeated trauma excites malignant change. Thus, nævi on the hands or feet, or on the shaving area of the face should be excised. An electric or chemical cauterisation is dangerous for it may cause malignant change. During excision care should be taken to avoid grasping or touching the lesion with instruments for fear of causing malignancy. The lesion is excised with a margin of at least  $\frac{1}{2}$  inch (12.5 mm.) of skin. Bluish-black moles are particularly sinister and should be widely excised. A large disfiguring melanoma in a child is best removed and the area closed by suturing or skin grafting.

**Melanomas (Malignant).** These are rare compared with the pigmented nævi. They may arise from a pre-existing pigmented lesion or develop *de novo*. Malignancy is suggested by: (a) Increase in size; (b) deepening pigmentation; (c) a palpable thickening which indicates superficial early malignant change (an extension of junctional activity); (d) the presence of moisture or ulceration; (e) bleeding from the surface; or (f) enlargement of regional lymphatic glands. Malignant melanomas arise in adult life, usually between the ages of thirty and seventy years, and affect both sexes equally. They are situated commonly on the lower limbs and especially on the feet; the head, neck, face and trunk are next involved in order of frequency. The nail bed is a well-known site. The anal or genital skin may be affected. Melanomas may also occur in the eye and meninges, and less commonly in the abdominal viscera.

Malignant melanomas disseminate widely and their rate of spread is usually rapid and rather unpredictable. Secondary deposits are conveyed through the lymphatics to regional lymph glands and through the blood stream to all parts of the body—including the skin. The liver is commonly involved. In advanced cases, especially with bulky secondary deposits in the liver, melanin may discolour the urine (melanuria). Transplacental spread occasionally occurs. Thus, a new-born infant of a mother with a malignant melanoma may develop secondary deposits. Occasionally, metastasis occurs several years after excision of an apparently benign melanoma (pigmented nævus). Thus secondary deposits may occur in the liver long after the removal of a melanoma of the eye; hence the

mouth sometimes following a febrile illness accounts for recurrent infection. *Streptococcus viridans* is then usually found in the saliva. The unilateral or bilateral parotitis results in marked "sialectasis." In females of this age group, Sjögren's syndrome is found. It consists of unilateral or bilateral recurrent parotitis, dryness of the eyes (keratoconjunctivitis sicca) and the mouth, atrophic glossitis, pharyngitis, achlorhydria, arthritis of the hands and scleroderma. The parotid secretion in these cases is viscid and infected, and the sialographic appearances are similar to those in other types of recurrent parotitis (*vide infra*). (e) In elderly edentulous people, ill-fitting dentures may cause parotitis by damaging the buccal part of the parotid duct.

#### Clinical Examination and Investigation.

The swollen parotid gland, the regional lymph nodes and the mouth are carefully inspected. Evidence of dental sepsis, ill-fitting dentures or ulceration of the mouth may be found. The opening of the parotid duct, seen adjacent to the upper second molar tooth, may be swollen, inflamed or scarred.

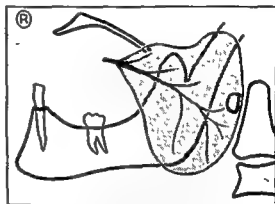


FIG. 438. Diagram showing the distribution of the ducts in the parotid sialogram. (S. S. Rose, *Annals Roy. Coll. Surg. Eng.*)

After expression of the parotid gland by gentle massage or placing a few drops of concentrated lemon juice in the mouth, the salivary flow can easily be seen. This secretion may be viscid, muco-purulent or absent. The saliva should be examined bacteriologically. A plain radiograph may demonstrate a densely calcified stone, but it is very rare in the parotid gland. The state of the parotid duct and its terminal acini may be shown by sialography. The opening of Stensen's duct is first dilated with a lacrimal duct dilator and  $\frac{1}{2}$  to 1 ml. of Neohydriol or diodone is injected into the duct through a blunt-ended cannula attached to a 5-ml. syringe. The cannula is withdrawn and any excess contrast medium is mopped away. Anteroposterior and lateral views are then taken. A normal sialogram (Fig. 438) shows a duct of even calibre with fine tapering terminal branches. If the gland is abnormal the ducts may be irregularly dilated and globules of contrast medium may lie within the gland—the appearances of saccular dilatation of the acini (Fig. 439). Sometimes pooling of the contrast medium remains in the salivary tissue long after injection. It is thought to be due to extravasation through the terminal ducts weakened by surrounding inflammation rather than to excess pressure during sialography (Patey and Thackeray, 1955), and may be mistaken for saccular dilatation of the



produced by tension within the inflamed gland, especially on eating. The inflammation usually subsides without treatment within a few days. The other salivary glands may also be involved. Abscesses and local lymphadenitis sometimes develop. Orchitis and pancreatitis are rare complications which may occur without evident parotitis in people exposed to infection. A second attack of mumps is exceptional.

**Acute and Recurrent Parotitis.** Acute inflammatory (non-epidemic) swelling of the parotid gland may occur at any age. Frequently the condition recurs, leading to permanent changes in the gland. The first attack cannot be easily distinguished from mumps, for in the acute stage the elastic parotid swelling is similarly ill-defined, occupying the sulcus between the mastoid process and the lower jaw and extending over the masseter region, and also, as in mumps, the pinna is displaced forward. Moreover, suppuration of the gland rarely develops. Infection of the parotid gland may be caused by : (1) Oral sepsis. (2) Diminished salivary secretion resulting from dehydration in prolonged fever or after operation. Acute parotitis from this cause used to be common in the elderly, but nowadays it is prevented by the maintenance of adequate fluid requirements and nursing attention to the mouth. (3) Dryness of the mouth (xerostomia) due to atrophy of the salivary secretory epithelium, which is liable to occur in the elderly. (4) Ill-fitting dentures leading to irritation, oedema and scarring of the opening of the parotid duct. (5) Congenital or post-inflammatory stenosis of the salivary duct. *Causal Organisms.*—The *Streptococcus viridans* is most commonly found; staphylococcal, streptococcal and mixed infections due to *E. coli* and diphtheroids may also be found. Occasionally pneumococcal infections occur and lead to abscess formation.

Acute non-epidemic parotitis is roughly divided into the following groups : (a) Suppurative parotitis of the new-born. It is rare and is usually associated with a generalised staphylococcal infection. (b) Recurrent parotitis of childhood which presents in the first decade of life. Acute episodes of discomfort and swelling in the parotid region lasting for three or four days lead to residual enlargement of the salivary gland. Recurrences occur at intervals of every few weeks or months. In most cases the mouth is clean and the opening of Stensen's duct appears normal, but occasionally the orifice is small and sometimes mucopurulent material speckled with white debris can be expressed from the parotid gland into the mouth. Calculi rarely develop. Sialography shows various degrees of pooling of contrast medium in relation to the terminal acini and even dilatation of the main duct. Early treatment can prevent progressive changes in the salivary gland. (c) In early adult life (at the time of eruption of the wisdom teeth), alteration of the bite may cause injury to the opening of Stensen's duct obstructing the salivary flow. Radiography shows dilatation of the duct, but not of the acini (S. Rose, 1953). (d) In middle age, more often in women than men, dryness of the

mandibular gland to form salivary "thrombi" of thick mucus and cellular debris around a focus of organisms—according to Rose calcification of these "thrombi" gives rise to microcalculi; (5) the upward flow of saliva along Wharton's duct compared with the horizontal flow along Stensen's duct favours salivary stagnation in the submandibular gland. Infection plays an important role in calculus formation, and is encouraged by narrowing of the duct opening with diminished salivary flow.



FIG. 440. Radiograph showing calculus formation in the submandibular salivary gland. Fusion of many calculi gives the appearance of a solitary stone.

**Submandibular Calculus.** There is discomfort and recurrent swelling of the gland, aggravated by eating. Two types of stones are found: (1) The single, elongated or club-shaped stone in Wharton's duct. It can usually be felt in the floor of the mouth and, being calcified, is visible in radiographs. Inspissated muco-pus may be expressed from the oedematous duct. (2) Multiple calculi situated mostly in the substance of the gland (Fig. 440). Palpation of Wharton's duct in the floor of the mouth and X-ray examination determine the type of stone present. Sialography (Fig. 441) indicates the state of the ducts and accurately localises the stones. "Sialectasis" is less evident in the submandibular than the parotid gland. **Differential Diagnosis.**—Enlarged submental lymph glands due to regional inflammation or malignant growth are distinguished from swelling of the submandibular salivary gland by lack of fluctuation in

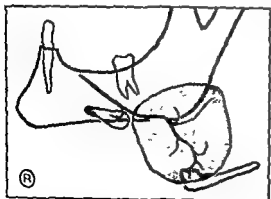


FIG. 441. Diagram showing the distribution of the ducts in a submandibular sialogram. (S. S. Rose, *Annal. Roy. Coll. Surg. Eng.*)

size and the finding of a primary focus and enlarged lymph nodes elsewhere. **Congenital cysts** are smooth and painless and usually lie in the midline. **Treatment.**—A single stone lying in Wharton's duct should be removed by incising the oedematous wall of the duct over the stone in the mouth. Care is taken to prevent the calculus slipping backwards. Multiple calculi are treated by excision of the submandibular gland, carried

acini (*sialectasis*). Sometimes a sialogram shows distortion of the ducts and filling defects may be produced by parotid tumours.

**Treatment of Acute and Recurrent Parotitis.** In the acute stage, palliative treatment with analgesics and local heat (infra-red lamp or short-wave diathermy) is beneficial. Dilatation of the opening of the duct with a lacrimal probe assists drainage if the intra-oral part of the duct is narrowed. Systemic antibiotics are advisable if the gland is acutely inflamed. Alternatively 1 to 2 ml. of penicillin (100,000 units) or 1 gm. of streptomycin may be instilled into the duct daily for six days (S. Rose,



FIG. 439. Sialogram showing irregular filling in upper portion of the parotid gland.

1954). Recurrent attacks are prevented to some extent by gentle massage of the parotid gland towards its buccal opening, and the use of sialogogues, such as acid drops and chewing gum. In addition, any dental sepsis is treated, and ill-fitting dentures are corrected. Conservative treatment is often successful in mild parotitis, but intractable recurrent parotitis with advanced inflammatory changes may call for removal of the gland. The facial nerve should, of course, be preserved although the dissection in these cases is usually difficult owing to scarring of the parotid tissue.

*The submandibular salivary gland*, like the parotid, is liable

to infection, giving rise to discomfort and swelling especially during meals. The inflammation responds to conservative measures for a time, but later, chronic infection and calculi often develop. Tumours occur in the submandibular gland, but as they are less common than in the parotid in the proportion of 1 to 10, a swelling of the submandibular gland is more likely to be due to infection than a tumour.

**Salivary Calculi.** About  $1\frac{1}{2}$  litres of saliva are secreted daily. The secretion of the parotid gland is serous, whereas that from the submandibular gland contains a large proportion of mucus, and is therefore more viscid. The sublingual and buccal glands secrete mucus only. The higher incidence of salivary calculi in the submandibular gland compared with that in the parotid gland is attributed to : (1) The greater mucus content of its secretion ; (2) its more alkaline saliva favouring the formation of insoluble calcium salts ; (3) its higher solid content, namely of inorganic salts and organic material ; (4) the tendency of the sub-

of a pleomorphic adenoma (mixed parotid tumour) is thought to arise from other separate tumour foci in the gland around the original growth. The pleomorphic adenoma is locally malignant, but metastasis rarely occurs. The *cylindroma* is probably a modified type of pleomorphic adenoma. It is a rare, malignant tumour giving rise to early metastasis in the lungs. (c) *Anaplastic carcinoma*, a malignant variant of the pleomorphic adenoma metastasises to regional lymph nodes and to the lungs, liver and adrenal gland. (d) *Adenolymphoma*, rarer than the pleomorphic adenoma; it is believed to be derived from developmental residues of salivary duct tissue in the lymph nodes contiguous with the salivary glands. The histology may suggest a secondary adenocarcinoma to the inexperienced. This slow-growing tumour is usually encapsulated and has a lobular surface. Its cut surface is yellowish and contains numerous cystic spaces. Microscopically, the epithelium shows intracystic papillary formation, and comprises two layers, a surface layer of columnar epithelium and a basal layer of cuboidal cells. The stroma consists of lymphoid tissue with germinal centres.

This should be regarded as a benign tumour, but it may have multiple foci.

A parotid tumour arises most commonly in the part of the gland situated just in front of and below the lobe of the ear (Fig. 442), but any part of the parotid gland may be involved. These swellings occur slightly more frequently in females than males, and appear mostly between the ages of twenty to fifty years. As a rule, these slow-growing tumours do not cause much discomfort. Thus treatment may not be sought until the swelling is very large. The pleomorphic adenoma is locally malignant and gradually involves the skin and deeper structures. The adenolymphoma should be regarded as a benign swelling, but this, too, may have multiple foci of origin. It is impossible to distinguish the above pathological groups on clinical examination. It is rarely possible to decide on clinical examination whether the growth is benign or malignant. Sialography shows the filling defect, but does not help in deciding its nature. Malignant change should be suspected if there is: (a) Rapid increase of size; (b) pain; (c) facial weakness; (d) infiltration of the



FIG. 442. A parotid gland tumour. (Arnold S. Jackson's patient.)

out when the acute infection has subsided and dissection is therefore easier.

**Parotid Calculus.** This is very rare, but cellular debris consisting of thickened secretion and chalky deposits may be seen in association with ascending infection from the mouth. Dilatation of the opening of Stensen's duct with a lacrimal probe and expression of the secretion from the gland relieves the condition.

**Salivary Fistula.** Injuries of the salivary glands or ducts by incisions or penetrating wounds are liable to produce a salivary fistula. In the past, parotid abscesses were incised and temporary fistulae occasionally resulted, but nowadays the abscesses are treated conservatively with antibiotics, and this complication therefore does not occur. Healing of a fistula arising from the glandular tissue usually takes place within a few weeks providing the main duct is intact. A persistent fistula from the parotid gland is best treated by radiotherapy to the gland or avulsion of the auriculotemporal nerve which carries the secretomotor fibres. Either method of treatment stops the secretion. If the fistula arises from damage of the duct, the cut ends are joined together over an indwelling nylon suture, or occasionally the fistula—if it lies anterior to the masseter muscle—can be made to drain the mouth, by inserting an unabsorbable suture into the fistulous tract; this stitch is introduced and tied on the buccal side of the cheek; it acts as a seton, and causes the intervening tissue between the fistula and the buccal mucosa to slough by pressure necrosis.

### PAROTID TUMOURS

Diagnostic problems abound in the parotid region.—R. T. PAYNE, 1950.

Tumours of the parotid gland are relatively uncommon; their behaviour is variable and their histology is complex. They are best classified along the lines suggested by Willis (1948): (a) *Adenoma*, a slow growing, solid encapsulated growth, really a variant of the next group; (b) *Pleomorphic adenoma* ("mixed parotid tumour"), by far the commonest type of salivary tumour; the description "pleomorphic" is better than the time-honoured term "mixed." This tumour is encapsulated and has a nodular surface. It is of firm to hard consistency. On section, this growth which is semi-translucent, shows areas of dense whitish tissue, and is soft and gelatinous in parts. It is now generally accepted that it originates from salivary gland epithelium although the histology is complex. Some of these tumours show gradations of atypical glandular tissue or solid or cystic epithelial parts spreading into a mucinous matrix, probably derived from secretory epithelium, whereas others show squamous metaplasia giving rise to muco-epidermoid cysts resembling sebaceous cysts. Sometimes scanty single detached cells are seen throughout mucinous tissue giving it the appearance of "cartilage." Sometimes the histological picture may resemble that of a carcinoma. Recurrence

moreover, if radiotherapy has been used, the resulting fibrosis of the parotid region will make subsequent excision of the gland difficult, and therefore jeopardises the facial nerve.

In the past, fear of damage to the facial nerve and inadequate knowledge of the pathology of these tumours has led to incomplete removal followed by recurrences. In recent years, total parotidectomy with preservation of the facial nerve has become accepted. A Y-shaped incision is used; the upper limbs of it embrace the lower half of the ear, and the distal limb of the incision is placed posterior to the mandible and extended downwards as required. After exposure of the surface of the parotid gland, the origin of the facial nerve at the stylomastoid foramen is located, and its branches are freed by blunt dissection from the substance of the parotid. An electrical stimulator should be used to help identify the facial nerve and its branches. When the radicles of the seventh nerve are freed, the deep aspect of the parotid gland can be removed from its bed without risk of producing facial paralysis. The wound heals well and is barely visible afterwards. After total parotidectomy with preservation of the facial nerve, there is a temporary facial weakness which, however, soon recovers. When the more radical operation has been carried out, the resulting facial palsy may be treated by means of the fascial sling operation. Care is taken to protect the cornea from ulceration by the use of eyeshades and lubricating drops. If conjunctivitis or corneal ulceration develops, tarsorrhaphy is indicated. Occasionally, hyperæsthesia, flushing and excess sweating of the side of the face occurs after operation (Frey's syndrome). This complication can sometimes be relieved by avulsion of the auriculotemporal nerve. The more recent adoption of radical surgery in the treatment of parotid tumours has so far reduced the rate of recurrence of pleomorphic adenomas (Redon, 1953), but it is too early to say what the long term results will be. *Benign tumours*, such as lipomas, simple cysts, hæmangiomas and lymphangiomas may occur in the parotid region. These should be excised if possible.

*Other salivary glands* are occasionally affected by tumours similar to those occurring in the parotid gland. The diagnosis is, however, rarely made by clinical examination, but is determined by microscopy after operation. These tumours can also occur in ectopic foci of salivary tissue in the oral or nasal cavities.

skin (Fig. 443) ; (e) metastasis in the glands in the neck ; (f) recurrence after removal.

**Differential Diagnosis.** (1) A sebaceous cyst over the parotid region may resemble a superficial cystic salivary neoplasm. (2) Lymphadenopathy in the neck near the angle of the jaw may be difficult to distinguish from a parotid swelling, but discovery of a primary focus or nodes else-



FIG. 443. Carcinoma of the parotid gland which has infiltrated the skin. (Dr. H. Clive Sims' case.)

where is evidence against a parotid tumour. (3) Tumours of the jaw, usually show radiologically. (4) Infection of the parotid gland, if acute, shows signs of inflammation, and recurrent parotitis causes the gland to vary in size and consistency, depending on the stage of the disease.

**Treatment.** As a pleomorphic adenoma of the parotid is usually slowly growing and possibly of multicentric origin, adequate initial treatment is essential. Moreover, since enucleation together with removal of a margin of the salivary tissue around the tumour is frequently followed by recurrence it is now generally accepted that more radical treatment is required. If the history and clinical signs of the swelling indicate a slowly growing

tumour and if at operation it is encapsulated, the neoplasm is to be regarded as of low-grade malignancy. Nevertheless, total removal of the parotid gland is advisable, but care is taken to preserve the facial nerve and its many branches. If the tumour is frankly malignant, total parotidectomy, including the facial nerve, should be carried out together with block dissection of the lymph nodes if involved. It is the border-line tumour that is difficult to assess, but the decision to perform the more radical operation is guided by the rate of growth and findings at operation, including the opinion of frozen sections. Useful information may often be obtained provided a representative part of the tumour has been examined, but it is not always possible for the experienced histologist to be dogmatic. In old patients with a slow-growing tumour, operation is probably better avoided. Parotid tumours are radio-resistant, and,

affects the debilitated and undernourished patient, and may result from iron-deficiency anemia or avitaminosis. It may affect patients who have undergone gastrectomy or intestinal resection. Glossitis and angular stomatitis are seen. Treatment consists of prescribing an adequate and mixed diet with the addition of vitamins and/or iron salts as necessary. Patients who have had prolonged treatment with antibiotics, especially Aureomycin, may develop *moniliasis* ("thrush") of the mucous membrane. This causes painful patchy desquamation of the mouth and is best treated by the local application of gentian violet and immediate withdrawal of the antibiotic. *Cancrum oris*, which is an infective gangrenous process involving the mouth, is nowadays rarely seen. *Behcet's disease* is a chronic non-specific infection affecting the uveal tract, mouth and genitalia and gives rise to recurrent painful ulceration of the mouth and vulva. Progressive blindness may develop. The cause of the disease is unknown and there is no specific treatment.

**Tumours of the Lips and Mouth.** *Benign lesions* such as papillomas, fibromas, lipomas, myomas and hæmangiomas can

occur. These are best excised, and in all cases microscopic examination of the tumour should be carried out. A diffuse lymphangioma of the lips gives rise to a condition known as *macrocheilia*; this vascular malformation may also involve the tongue (*magroglossia*). Both these conditions require plastic surgery if there is no natural diminution in size. *Salivary tumours* arising in the small salivary glands of the mouth are occasionally seen as small, firm, circumscribed swellings. These should be removed. The diagnosis is only made after histological examination.

**Malignant Lesions.** Oral cancer is an uncommon form of malignant disease, forming 3 per cent. of cancer deaths per year (Stanford Cade, 1949). The disease, which predominantly occurs in males, is gradually declining. The predisposing causes are similar to those of superficial glossitis (see Chap. 64), a precancerous condition. Dental sepsis, smoking and drinking spirits are aggravating factors. As in cancer of the tongue, an ulcerative, papilliferous or nodular lesion may occur. The squamous-celled epitheliomatous ulcer is commonest. The patient complains of an ulcer or a nodule in the mouth which may interfere with swallowing.



FIG. 445. A mucous cyst of the lip.



## CHAPTER 63

### DISEASES OF THE MOUTH AND LIPS

**Congenital Abnormalities.** The commonest of these are *cleft palate* and *cleft lip*. These deformities are discussed in Chap. 16. A *congenital short frænum of the upper lip* is a less common condition. The frænum of the upper lip which is shorter than normal, extends between the two central incisor teeth (Fig. 444). If untreated it will leave a gap in the midline. A plastic procedure is necessary. The short frænum is cut across transversely, above and parallel to the alveolar margin and the median extension between the teeth is separated from the alveolus. The incision is sutured in a longitudinal direction, and thus the frænum is lengthened.

**Cystic Swellings of the Mouth.** A *sublingual dermoid cyst* presents as a rounded swelling in the floor of the mouth. This condition usually arises in adult life ; it is rarely seen in children. The cyst is lined by squamous epithelium and contains sebaceous material which can be indented with the finger. It does not transilluminate. If large, the cyst may bulge into the submental region. Excision of the swelling should be carried out, using an incision in the submental region. A *retention cyst* of the buccal mucous glands may arise anywhere on the lining of the mouth (Fig. 445). This cyst is usually small and contains clear mucus. It is best excised

under local anæsthesia. A *ranula* is a type of retention cyst. It lies characteristically on the floor of the mouth off the midline, where it presents as a smooth, thin-walled glistening swelling beneath the mucous membrane. Aspiration should be tried first, but, if unsuccessful, excision should be carried out. If this is not possible, the buccal surface lining of the cyst should be removed, with due regard to Wharton's duct and superficial veins ; the resulting circular raw edge is finally sutured to allow open drainage.

**Inflammation** of the mouth and lips is occasionally seen in surgical practice. *Stomatitis*

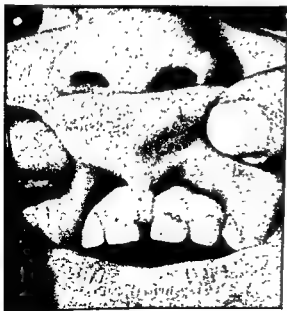


FIG. 444. A congenital short frænum of the upper lip showing the gap between the central incisor teeth.

removed and block dissection of the enlarged lymph glands on the same side of the neck is carried out ; if practical, this should be performed in continuity as a one-stage procedure. Following resection of the jaw, the resulting deformity may, after a long interval, be corrected by a metal splint or bone graft. This plastic procedure is only advisable in relatively young and fit patients. The five-year survival rate for cancer of the floor of the mouth is 41 per cent. (Stanford Cade, 1949). *Cancer of the cheek* also occurs more often in men than women, and again elderly people are affected. The irritation of a carious tooth, the wearing of ill-fitting dentures or a patch of leukoplakia often precedes the disease. There is an increased incidence of this condition where tobacco and betel-nut chewing is practised. Radiotherapy is the best treatment and produces a survival rate of over five years in about 60 per cent. of cases (Cade, 1949). Excision of the glands is preferable to radiotherapy if the nodes are palpable and operable. Local recurrence is difficult to treat, for wide excision of a previously irradiated area produces marked deformity and dysfunction of the mouth ; as these patients are often undernourished, the healing of plastic procedures is poor, and a salivary fistula may develop. *Cancer of the Palate*.—There are points of difference between malignant disease involving the hard and soft palate (Cade, 1949). Carcinoma of the hard palate affects males and females equally, and radiotherapy and surgery combined give the best results (53 per cent. have over a five-year survival), whereas in cancer of the soft palate, males are more affected than females, and cervical glands are twice as frequently involved, often bilaterally. Radiotherapy is the treatment of choice, but only produces a 5 per cent. five-year survival rate.

Pain is uncommon at first, and dysphagia is also a late symptom, for it is due to infiltration of the surrounding muscles. Quite often, the first sign is a swollen lymph gland in the neck. Early diagnosis gives the best chance of cure. The mouth should be carefully examined in a good light, and the mobility of the tongue and palate observed. On palpation, a malignant lesion is found to be indurated. There may be enlargement of the cervical glands which, when hard and fixed, indicate an advanced stage of the disease. In addition to local examination, the general condition of the patient should be assessed. A Wassermann blood test should be carried out and a biopsy of the lesion done. *Cancer of the lip*



FIG. 446. Carcinoma of the lower lip. This was first noticed by the patient three months previously. (Mr. Anthony Green's case.)

more often involves the lower lip than the upper, in the proportion of 9 to 1. Men (90 per cent.) are more commonly affected than women. It develops between the age of fifty and seventy years, usually as a slow-growing lesion (Fig. 446). Enlargement of the lymph glands develops late. Radiotherapy or excision are equally effective. The prognosis is good, especially in cases in which the glands are not involved; 85 per cent. of cases survive over five years. *Cancer of the floor of the mouth* is a localised lesion at first, but ultimately spreads to the alveolus. The submandibular lymph glands are involved in about half of the cases, sometimes bilaterally. If the alveolus has become involved, radiotherapy is preferable to surgery, and produces good results. If the lymphatic glands are involved and operable, block dissection of the neck should be carried out. If the growth has spread well into the mandible, resection of this bone is necessary; the affected part of the mandible is widely

frænum. This plastic procedure is preferable to the time-honoured practice of snipping the base of the tongue with scissors, for healing after the former operation is better, whereas, after the latter, marked scarring may result, leaving the tongue still "tied down." (g) *Congenital ankyloglossia* is a rare condition in which the anterior margin of the under surface of the tongue is tethered to the floor of the mouth. As the mobility of the tongue is diminished, feeding and phonation may be difficult. A plastic procedure is advisable in order to mobilise the fore-part of the tongue.

**Inflammation.** *Acute Glossitis.*—Acute inflammation of the tongue may result from swallowing corrosive or excessively hot fluids. Insect bites also produce acute inflammatory swelling. The tongue may also become very œdematous as a result of allergic reaction to sensitising agents such as some drugs, injections of sera or antibiotics. In severe cases, treatment consists of sucking ice and spraying the tongue with adrenaline solution (1 : 1,000). Antihistaminic drugs are useful in allergy. Occasionally tracheotomy is required if œdema spreads downwards and involves the air passages. *Chronic Superficial Glossitis.*—This is a well-recognised clinical entity. Alcohol, smoking, oral sepsis, syphilis and spicy foods are possible exciting causes. The anterior two-thirds of the tongue are most commonly affected. Its surface shows areas of heaped up and hypertrophied epithelium, giving the appearance of patches of white paint (leukoplakia). Later, fissuring of the surface, probably due to scarring of the deep layers of the dermis, appears, and finally atrophy of the papillæ leaves a raw, red, smooth surface. As these changes occur irregularly the various stages may be seen in patches on the tongue. This condition may cause soreness of the tongue. It persists for many years and alters slowly. About a third of the cases develop malignant change. Similar white areas of hypertrophic epithelium may also be seen in the lining of the mouth. These, too, have malignant tendencies. No special treatment is required for this clinical condition, except in the case of syphilitic infections which are treated accordingly. However, periodic observation is necessary lest carcinoma develops. Any thickening or ulceration is suspicious of malignancy; in cases of doubt, biopsy is necessary. *Atrophic Glossitis.*—A smooth, red surface is also seen in association with vitamin or iron deficiency and should be treated by correcting the avitaminosis or anæmia.

**Black Hairy Tongue.** This curious condition is usually associated with the administration of oral antibiotics. The filiform papillæ are dark and appear as an area covered with black "hairs"; the change is probably due to alteration of the flora of the mouth by drug therapy.

**Acute Ulcers of the Tongue.** A traumatic ulcer due to irritation by a jagged tooth is commonly seen, and is cured by removal of the tooth. *Dyspeptic ulcers* also commonly occur; they are small white painful shallow ulcers which usually clear up in a few days without any special treatment.

## CHAPTER 64

### THE TONGUE

**Congenital Abnormalities.** (a) *Absence of the tongue*—very rare. (b) *Bifid tongue*—very rare. (c) *Hypertrophy* of the tongue is occasionally seen in cretins and mental defectives. (d) *Hemihypertrophy* of the tongue may be associated with defective growth of the limbs. (e) *Excessive mobility*, due to abnormal length of the tongue or of its frænulum, is occasionally seen in infancy; the tongue may fall back and block the oropharynx leading to suffocation. Such infants are best nursed prone and fed in this position in order to prevent this catastrophe. (f) *Short frænulum* of the tongue is a fairly common deformity; it prevents protrusion of the tip of the tongue beyond the lower incisor teeth (Fig. 447). This familial condition does not usually cause any serious handicap, and in the majority of cases does not interfere with eating or phonation. The synonymous term "tongue-tie" is unfortunate, and should be



FIG. 447. Congenital shortening of the frænulum of the tongue.

discarded, for though the tongue is partly tethered, the condition is not as serious as this term suggests. Mothers with babies born with this condition seek medical advice early, and can be reassured that there will be no serious disability. Occasionally, the insistence of the mother, who may have had her tongue "snipped" in childhood, may lead to a similar operation being performed on her infant. Operation should be considered only in the occasional case when the pronunciation of a few consonants is imperfect. Division of the frænum, followed by a plastic repair, should be carried out; the offending fold of mucous membrane is divided horizontally and freed; it is then repaired by suturing the edges of the incision vertically, thereby lengthening the

fibromas, lymphangiomas and adenomas are best excised. Such localised tumours should be examined microscopically to confirm the diagnosis. An *ectopic thyroid tumour* occasionally occurs at the junction of the anterior two-thirds and the posterior third of the tongue. It may be cystic, but more often consists of ectopic thyroid tissue. Its size may cause difficulty in swallowing or breathing. Before contemplating its removal, it is necessary to make sure that there is thyroid tissue elsewhere. The presence of the normal thyroid gland can be determined by the use of a tracer dose of radioactive iodine, and subsequent scanning of the neck with a Geiger-Mueller counter. If other thyroid tissue is present, the swelling in the tongue can be safely removed.

**Carcinoma of the Tongue.** The tongue is a common site for oral cancer and occurs more frequently in men than women, in the proportion of 9 to 1. It arises between the ages of fifty and seventy years. The possible exciting causes are the same as those discussed under *chronic superficial glossitis*. The decreasing incidence of the disease is probably due to better dental hygiene and decline in syphilitic infection in the past fifty years. The edge of the anterior two-thirds of the tongue is most often involved (Fig. 449), though any part of the tongue can develop a carcinoma.



FIG. 449. Carcinoma of the tongue showing an ulcerated indurated plaque on the outer margin. (Mr. Anthony Green's case.)

Carcinoma of the tongue is usually a squamous-celled epithelioma. It presents as an ulcer or hard nodule which later ulcerates, in either case a crater develops with a dirty sloughing base and raised, hard edges. Occasionally a papilliferous growth arises. Sometimes a carcinoma arises in an area of superficial chronic glossitis (*vide supra*). Ulceration and induration in a fissured tongue suggests malignant change. The lymph glands which drain the tongue are numerous, but those situated along the jugular vein are most commonly affected. The upper group (jugulo-digastric) drain mostly the posterior third of the tongue, and the lower group (jugulo-omohyoid) receive lymph vessels from the rest of the

**Chronic Ulcers.** *Tuberculous ulceration of the tongue* is rare nowadays, for it usually arises in patients with advanced pulmonary tuberculosis. The ulcer may be superficial or deep; its edge is undermined and its surface is grey and soft. Pain is usually a feature. Treatment is that of the primary tuberculous lesion. *Syphilis of the tongue* is also rarely seen. A primary chancre can occur in this situation and is associated with cervical adenitis. In secondary syphilis, mucous lesions appear as soft red patches on the dorsum of the tongue, and snail track ulcers occur on the lips, soft palate and fauces. As these lesions are severely infected with spirochaetes they are therefore very contagious. In the tertiary stage, syphilis of the tongue may appear as: (a) Chronic superficial glossitis (*vide supra*); (b) chronic gummatous infiltration of the tongue causing thickening of its substance followed by fibrosis with fissuring and/or ulceration; or (c) a gumma, which is often in the midline of the tongue; this indurated nodule may break down and ulcerate deeply; it is painless and may be mistaken for a carcinoma (*vide infra*). The diagnosis rests on finding other signs of syphilis and a positive Wassermann reaction. In doubtful cases biopsy examination is necessary. Syphilis of the tongue is treated by mouth washes and the usual anti-syphilitic drugs.

#### TUMOURS OF THE TONGUE

**Benign Tumours.** These are uncommon. A *cavernous angioma* presents as a purplish, soft, compressible and irregular swelling. It may be discrete or diffuse. As bleeding and ulceration of the surface are liable to occur, excision should be carried out if possible (Fig. 448); otherwise, electrocautery is advised. Other tumours, such as papillomas, lipomas,



FIG. 448. Ulcerated angioma of the tongue which was subsequently excised.

fibromas, lymphangiomas and adenomas are best excised. Such localised tumours should be examined microscopically to confirm the diagnosis. An ectopic thyroid tumour occasionally occurs at the junction of the anterior two-thirds and the posterior third of the tongue. It may be cystic, but more often consists of ectopic thyroid tissue. Its size may cause difficulty in swallowing or breathing. Before contemplating its removal, it is necessary to make sure that there is thyroid tissue elsewhere. The presence of the normal thyroid gland can be determined by the use of a tracer dose of radioactive iodine, and subsequent scanning of the neck with a Geiger-Mueller counter. If other thyroid tissue is present, the swelling in the tongue can be safely removed.

**Carcinoma of the Tongue.** The tongue is a common site for oral cancer and occurs more frequently in men than women, in the proportion of 9 to 1. It arises between the ages of fifty and seventy years. The possible exciting causes are the same as those discussed under *chronic superficial glossitis*. The decreasing incidence of the disease is probably due to better dental hygiene and decline in syphilitic infection in the past fifty years. The edge of the anterior two-thirds of the tongue is most often involved (Fig. 449), though any part of the tongue can develop a carcinoma.

Carcinoma of the tongue is usually a squamous-celled epithelioma. It presents as an ulcer or hard nodule which later ulcerates, in either case a crater develops with a dirty sloughing base and raised, hard edges. Occasionally a papilliferous growth arises. Sometimes a carcinoma arises in an area of superficial chronic glossitis (*vide supra*). Ulceration and induration in a fissured tongue suggests malignant change. The lymph glands which drain the tongue are numerous, but those situated along the jugular vein are most commonly affected. The upper group (jugulo-digastric) drain mostly the posterior third of the tongue, and the lower group (jugulo-omohyoid) receive lymph vessels from the rest of the



FIG. 449. Carcinoma of the tongue showing an ulcerated indurated plaque on the outer margin. (Mr. Anthony Green's case.)



tongue. The submental and submandibular glands drain the tip of the tongue. Lymphatic spread across the midline to the contralateral glands also occurs, more especially from the anterior and posterior parts of the tongue. Enlargement of the cervical glands may at first be wholly or partly due to inflammatory swelling, and therefore at this stage the glands are soft compared with the typically hard malignant glands found later. In the later stages, the movements of the tongue are restricted, making swallowing difficult; there is also usually marked *factor oris* and loss of weight. Distant metastatic deposits rarely develop, and the patient ultimately dies from bronchopneumonia or cachexia. On examination, the ulcer is easily visible if situated on the anterior two-thirds of the tongue, whereas if situated on the posterior third, a mirror may be required to demonstrate the lesion. The extent of the growth is best assessed by examination with a finger, thereby malignant induration can be easily appreciated. Deviation of the tongue to one side on protrusion and complete fixation are late signs. Enlargement of the regional lymph glands should be sought. These are usually enlarged on the same side as the lesion, but bilateral cervical metastasis may occur. Only about a third of the patients when first seen are clinically free from involvement of cervical glands. Whereas about half the patients have cervical deposits when the lesion is on the anterior two-thirds of the tongue, about three-quarters of the patients have them when the lesion is on the base of the tongue. The more posterior the lesion, the later it is likely to be diagnosed.

*Differential Diagnosis.* A *gumma* which causes fibrotic induration of the tongue may resemble a carcinoma; therefore other signs of syphilis should be sought, and a Wassermann blood test should be carried out in all cases. A positive result, however, does not exclude a carcinoma, it may coexist with syphilis. The diagnosis established is by biopsy. A *traumatic ulcer* should be considered if there is an adjacent jagged tooth or denture. As this type of ulcer is liable to become malignant, biopsy is again necessary if the lesion persists after removal of the cause. A *tuberculous ulcer* is indolent and has soft undermined edges. Pulmonary tuberculosis is usually present. In addition, tubercle bacilli may be found in a smear made from the lesion. *Glossitis mediana rhomboidea* presents as a lozenge-shaped thickening of the centre of the tongue in the region of the tuberculum impar. The cause of the condition is unknown. It is benign and does not require any treatment.

*Treatment.* Most cancers of the tongue can be recognised clinically, but in cases of doubt a biopsy should be done. This confirms the diagnosis and also indicates the activity of the growth. The treatment of this disease depends on the stage at which it is first seen. If the growth is confined to the tongue, radiotherapy is the treatment of choice. Those growths on the anterior two-thirds of the tongue are usually well differentiated and show a slow but effective response to radiotherapy.

whereas those on the posterior aspect are often anaplastic and respond rapidly. If the cervical nodes are involved and these are mobile, block dissection should be carried out *after* radiotherapy has been completed and the tongue lesion has healed, provided the patient's general condition is sufficiently good to withstand the operation. If the cervical glands are fixed, and/or the patient is in a poor state, radiotherapy is advisable; excision is impracticable, especially if the carotid sheath is invaded. Prophylactic block dissection of the neck is not justifiable, but periodic examination is essential so that at the first sign of involvement of the regional nodes, excision may be carried out. If the growth involves the alveolus of the lower jaw, radiotherapy is contraindicated, as bone necrosis might develop. Therefore, wide excision, including the affected side of the mandible, should be carried out and combined with removal of the cervical tissue (as in block dissection), provided the patient's general condition is good. When excision is contemplated any oral sepsis must first be treated by the dental surgeon. Administration of a high calorie diet with the full range of vitamins will assist post-operative healing. At operation, wide excision is necessary to circumvent the growth and to ensure approximation of healthy skin flaps; the mucous membrane should be repaired as accurately as possible to prevent a salivary fistula, for this complication is difficult to cure. After operation, tethering of the remainder of the tongue, narrowing of the oropharynx and loss of part of the jaw may make swallowing difficult. A soft semi-solid diet may therefore be necessary. Occasionally, there is bilateral cervical gland enlargement due to lymphatic spread across the midline of the tongue; bilateral block dissection should then be carried out in two stages, separated by an interval of two to three weeks.

Recurrence after treatment presents a difficult problem, for the area is usually scarred as a result of radiotherapy and/or surgery. However, wide excision and repair is justifiable in a fit patient. Radical methods should only be used if there is a reasonable chance of eradicating the disease and leaving the patient comfortable.

Prognosis is favourably influenced by early diagnosis, marked differentiation of the tumour cells, and adequate radiotherapy and/or surgery.

tongue. The submental and submandibular glands drain the tip of the tongue. Lymphatic spread across the midline to the contralateral glands also occurs, more especially from the anterior and posterior parts of the tongue. Enlargement of the cervical glands may at first be wholly or partly due to inflammatory swelling, and therefore at this stage the glands are soft compared with the typically hard malignant glands found later. In the later stages, the movements of the tongue are restricted, making swallowing difficult ; there is also usually marked fœtor oris and loss of weight. Distant metastatic deposits rarely develop, and the patient ultimately dies from bronchopneumonia or cachexia. On examination, the ulcer is easily visible if situated on the anterior two-thirds of the tongue, whereas if situated on the posterior third, a mirror may be required to demonstrate the lesion. The extent of the growth is best assessed by examination with a finger, thereby malignant induration can be easily appreciated. Deviation of the tongue to one side on protrusion and complete fixation are late signs. Enlargement of the regional lymph glands should be sought. These are usually enlarged on the same side as the lesion, but bilateral cervical metastasis may occur. Only about a third of the patients when first seen are clinically free from involvement of cervical glands. Whereas about half the patients have cervical deposits when the lesion is on the anterior two-thirds of the tongue, about three-quarters of the patients have them when the lesion is on the base of the tongue. The more posterior the lesion, the later it is likely to be diagnosed.

*Differential Diagnosis.* A gumma which causes fibrotic induration of the tongue may resemble a carcinoma ; therefore other signs of syphilis should be sought, and a Wassermann blood test should be carried out in all cases. A positive result, however, does not exclude a carcinoma, it may coexist with syphilis. The diagnosis established is by biopsy. A traumatic ulcer should be considered if there is an adjacent jagged tooth or denture. As this type of ulcer is liable to become malignant, biopsy is again necessary if the lesion persists after removal of the cause. A tuberculous ulcer is indolent and has soft undermined edges. Pulmonary tuberculosis is usually present. In addition, tubercle bacilli may be found in a smear made from the lesion. *Glossitis mediana rhomboidea* presents as a lozenge-shaped thickening of the centre of the tongue in the region of the tuberculum impar. The cause of the condition is unknown. It is benign and does not require any treatment.

*Treatment.* Most cancers of the tongue can be recognised clinically, but in cases of doubt a biopsy should be done. This confirms the diagnosis and also indicates the activity of the growth. The treatment of this disease depends on the stage at which it is first seen. If the growth is confined to the tongue, radiotherapy is the treatment of choice. Those growths on the anterior two-thirds of the tongue are usually well differentiated and show a slow but effective response to radiotherapy,

reaction exceptionally is acute and may form an abscess in which the fungus grows plentifully but not in the form of granules. More commonly the reaction leads to the formation of a mass of hard fibrous tissue, almost avascular and on section looking very like a sarcoma or scirrhus carcinoma; embedded in this may sometimes be seen small abscesses, but in many cases it needs very careful microscopic examination to find any trace of the organism. Sooner or later softening occurs and an abscess is formed in which the granules are present. The inflammation spreads by continuity and often follows the line of least resistance; it has little respect for any tissue, always excepting surface epithelium and dense fascia such as the dura mater.

Thus, actinomycosis should be considered in any subacute or chronic inflammation, or hard swelling in the cervicofacial region, especially if chronic abscesses or sinuses are present. This infection occurs a little more commonly here than in the intestinal tract, but infection in both sites is similar in behaviour and chronicity. Once the diagnosis has been established by finding the actinomyces in the lesion, treatment by antibiotics is given. Penicillin in full doses (a million units daily for six weeks) is effective; other antibiotics are also of benefit. *Tuberculous and syphilitic infection of the jaw rarely occurs.*



FIG. 450. Osteitis of the maxilla in infancy. Note the infra-orbital oedema.

*Necrosis of jaw* may follow irradiation, but this complication is infrequently seen nowadays owing to improvement of radiotherapeutic techniques. Radionecrosis is a painful condition causing diffuse inflammation and swelling of the jaw; trismus may also develop. The condition subsides with drainage of the abscess pockets and removal of sequestra, but the disease is usually chronic.

**Swellings of the Gum.** An *epulis* is an outgrowth from the alveolar margin or periodontal region arising close to the neck of a tooth. There are various types: the fibrous epulis is a smooth, firm, pedunculated swelling attached to the periosteum; the granulomatous epulis consists of a mass of granulation tissue lying in the same situation; microscopically these are composed of fibrous and granulation tissue respectively, and the latter may contain giant cells. Another but rare variety is seen in infancy; clinically it resembles a fibrous epulis, but on histological examination contains a collection of foam cells, the origin of which is

## CHAPTER 65

### DISEASES OF THE JAWS

**Infection.** *Osteitis due to dental infection* is the most common, and arises from an alveolar or periapical abscess due to an infected tooth. It causes acute inflammation, spreading, in the case of the lower jaw, into the submental region, and in the case of the upper jaw to the face and infra-orbital tissue. The tooth causing the abscess is usually carious or dead; there is swelling and marked tenderness in the bucco-gingival sulcus adjacent to the root of the diseased tooth. Pressure on the tooth towards its socket or tapping the crown of the tooth intensifies the pain. Radiological examination shows an area of bony absorption around the root of the tooth. In the acute stage, treatment consists of drainage of the abscess at the alveolar margin of the tooth and administration of antibiotics and sedatives; hot mouth washes are also prescribed. When the acute infection has subsided, the diseased tooth is extracted, but occasionally it can be saved. An open fracture is liable to become infected at the line of the fracture which often corresponds to the axis of a tooth; thus removal of the tooth (or teeth) adjacent to the fracture line is necessary, in addition to reduction and fixation of the fracture. *Osteitis of the maxilla in infancy* is a recognised clinical entity. It is caused by infection around an unerupted tooth, usually the upper first molar. In some cases, it is a localised manifestation of bacteraemia, for occasionally pyogenic organisms grow on blood culture. It presents with a painful swelling of the face, spreading into the infra-orbital region and so may be mistaken for orbital cellulitis (Fig. 450). On examination of the mouth, the alveolar margin is swollen and tender; the condition usually subsides within a few days of the onset with the discharge of pus from the site of infection. Treatment consists of administration of antibiotics and drainage of the area with minimal interference; extraction of unerupted teeth should be avoided as it may result in maldevelopment of future dentition. In some cases, part of the upper jaw is destroyed by the infection, and forms a sequestrum, which is shed later.

*Actinomycosis of the jaw* is caused by *Actinomycosis bovis* (or *Israelii*). This organism is found in the crevices of the teeth or tonsils. Zachary Cope (1949) well describes the development of the disease:

That the organism which is so frequently present in the body does not more often cause disease is due to the fact that it finds difficulty in penetrating normal epithelial surfaces; when by accident, disease or by necessary operation the epithelial surface is broken, the actinomyces escape into the cellular tissues and begin to grow, well away from the surface. The extraction of a tooth or a perforation of the appendix will be sufficient to allow its escape. The tissue

cells are also seen amongst the stroma. Tumours resembling adamantinomas have also been observed in the long bones, and the base of the skull. Total removal of the tumour is curative. It is often possible to retain the hard inferior rim of the mandible, and so avoid the disfigurement of complete division of this bone (F. C. Wilkinson, 1952). Radiotherapy is of little benefit. *Intra-alveolar epidermoid carcinomas* are rare and arise within the jaw, and possibly are derived from remnants of the dental lamina. They spread and ulcerate into the mouth, and lead to glandular involvement.

*Odontomes* arise from malformation or disturbance of the enamel, dentine or cement of the teeth, and appear as a conglomerate mass of these elements. They occur



FIG. 451. Adamantinoma arising in the lower molar/premolar region. (Courtesy of the Eastman Dental Hospital, London.)

mostly in the young in the vicinity of an unerupted tooth, and radiologically dense areas of cement or denticles can be seen. Removal of these tumours is usually necessary owing to infection or maldevelopment of the teeth.

*Epithelial-lined Cysts of the Jaw.* A dental cyst occurs at the root of an imperfect tooth, forming an epithelial-lined cavity within the jaw. The cyst expands the bone and causes thinning of its surface; this can be seen radiologically. An infected cyst, arising at the root of a diseased tooth, is lined by granulation tissue (Fig. 452). Extraction of the affected tooth and drainage of the cavity cures the condition. A *dentigerous cyst* (follicular odontome) arises in connection with an unerupted



FIG. 452. Dental cyst associated with an upper lateral incisor tooth. (Courtesy of the Eastman Dental Hospital, London.)

unknown. A small hæmangioma on the gum may simulate an epulis. Epulides are benign tumours and their removal with an adjacent gum margin usually cures them ; in addition extraction of carious teeth in the vicinity is necessary. *Hypertrophy of the gums* is sometimes seen in association with dental sepsis ; the soft gingival tissue only may be enlarged, but bony thickening of the underlying alveolar ridge also occurs. Submucous removal of the enlarged bone and dental treatment is advisable if the deformity interferes with mastication, or is unsightly.

**Tumours of the Palate.** *An apical abscess* at the root of an infected tooth can give rise to a painful, circumscribed swelling on the hard palate above the margin of the alveolus ; dental extraction is required. *An ectopic salivary tumour* may arise on the palate forming a firm, localised swelling which slowly increases in size, and may ultimately ulcerate ; such a neoplasm is more often a *cylindroma* than a "mixed" salivary tumour, but the distinction can only be recognised on histological examination. The prognosis for a cylindroma is bad, for local recurrence and metastasis in the glands and viscera are liable to occur (Kenneth Harrison, 1956). Thus, removal by wide excision is necessary as radiotherapy is of little benefit. *A carcinoma of the hard palate* may also occur, and appears first as a nodule in the mucosa which later ulcerates. *A gumma* is also known to develop at this site.

**Perforation of the Palate.** *Congenital clefts* are by far the commonest abnormality and should the repair of such a deformity be incomplete, a perforation will remain (see under cleft palate). *An acquired perforation of the palate* may be caused by a penetrating object, such as injury due to a fall with a pipe in the mouth. This type of penetration can usually be repaired easily, and heals well. More rarely, syphilitic infection with the formation of a gumma may leave a hole in the palate. Occasionally the spread of a malignant growth from the antrum may penetrate into the mouth.

**Swellings of the Jaws.** *Dental epithelial tumours* consist of the following (Willis, 1948) : *Adamantinoma* (ameloblastoma) is so-called, as the microscopic appearance of the tumour is similar to that of the enamel organ, but the tumour fails to form enamel. Willis suggests that "carcinoma of the tooth germ residues" is a more suitable description. This neoplasm affects adults of both sexes equally, and appears first as a solid swelling, and later becomes cystic ; it is an uncommon tumour and more often involves the lower jaw than the upper ; it is characteristically slow growing and painless. Radiologically this localised swelling has a honey-combed appearance and later shows as a mass of cysts of different sizes, expanding the jaw (Fig. 451). An *adamantinoma* is usually benign, but rarely malignant changes develop. Histologically it is composed of follicles which are outlined with palisade (columnar) epithelium and which contain a fine stellate reticulum within ; sometimes whorls of squamous

rise to the clinical condition known as *cherubism*, in which both jaws are enlarged. A diet, rich in calcium and vitamin D improves early leontiasis ossea and cherubism (E. D. D. Davis, 1956). Plastic procedures may be necessary in advanced cases if the deformity is marked. General bone diseases also affect the jaw. In *Paget's disease* (osteitis deformans), a diffuse fibrous dysplasia may occur, causing enlargement of the jaw in addition to the other skeletal changes affecting the pelvis, long bones, skull and spine. In *acromegaly*, there is overgrowth of the jaw, in addition to the characteristic enlargement of the face, hands and feet; the pituitary fossa is increased in size.

### CANCER OF THE UPPER JAW

By J. CHALMERS BALLANTYNE

Malignant tumours of the upper jaw usually arise in the maxillary or ethmoid sinuses, often at their junction. The squamous-cell carcinoma is the commonest, but adenocarcinoma is not rare. The basal-cell type of growth is confused by a number of synonyms: Antral adamantinoma, adenocystic endothelioma, or cylindroma; and transitional-cell carcinoma. It behaves like the mixed salivary tumour in growing slowly, distending and eroding bone, metastasising very late, and sometimes ending fatally by local extension. Fibrosarcoma and osteosarcoma are rare. It must not be forgotten that secondary tumours may arise in the upper jaw from primary growths in the breast, prostate or thyroid gland.

Epistaxis and nasal discharge are always suspicious symptoms when they are unilateral. Encroachment on the medial wall of the sinuses will cause unilateral nasal obstruction, as also will the presence of polypi. The malignant polypus is characteristically single and solid, red and fleshy, and bleeds readily when touched; simple polypi, however, may also occur (from secondary infection) and these tend to be multiple. Epiphora is caused by obstruction of the nasolacrimal duct due to spread of the growth in this direction, whereas swelling or ulceration of the hard palate results from extension downwards through the floor of the antrum. The upper alveolus may be expanded, and the related teeth may become loose or even fall out. The tumour mass may extend forwards to cause a swelling of the cheek, or upwards to cause diplopia. Late symptoms include ulceration of the cheek; pain, earache and toothache, from involvement of the fifth cranial nerve; headaches, from involvement of the dura mater; and trismus, again due to involvement of the fifth cranial nerve. Lymphatic glandular metastasis is uncommon, but may occur late in the disease. The outstanding clinical feature of new growths of the upper jaw is that their symptoms and signs are *unilateral*, in contrast with most of the simple inflammatory conditions of dental or sinus origin. They can be confirmed radiographically by the presence of



tooth. This single cyst occurs in early adult life, more often in the lower jaw in relation to the third molar tooth. Dental treatment is required as above.

*Other Tumours of the Jaw.* An osteoma gives rise to a hard swelling which grows slowly and, in the upper jaw, encroaches into the maxillary antrum. If large, it produces disfigurement and interferes with closure of the jaw. The tumour should be removed if possible and the resulting deformities corrected by means of a prosthesis. Callus following a fracture of the jaw may resemble an osteoma. A *chondroma* rarely occurs in the jaw. *Osteoclastoma* (giant-celled tumour of bone) rarely develops in the jaw. Its appearance and behaviour are similar to osteoclastomas of long bones. The jaw gradually expands with the growth of the tumour, causing softening of the bone surface, and sometimes a pathological fracture. This swelling consists of numerous cysts of variable size, filled with mucinous tissue or blood. The osteoclastoma resembles *osteitis fibrosa* associated with hyperparathyroidism, but the osteoclastoma should be regarded as a neoplasm and osteitis fibrosa as a type of fibrous dysplasia; differentiation is difficult in the early stages, and can only be determined by biopsy and the blood chemistry. Total removal of the tumour (osteoclastoma) cures the condition. *Carcinoma of the jaw* most commonly arises from spread of the disease from adjacent buccal mucosa. *Sarcoma* of the jaw may involve the surface or the centre of the bone. It arises in young adults and has similar features to those of sarcoma of the long bones. Pain and swelling of the jaw occur, and there is infiltration of the surrounding structures including the sinuses and skin. Pathological fracture may occur. Its rate of growth varies; the osteoblastic type is slower growing than the osteolytic variety, but in either case the prognosis is bad. Recurrences after wide excision occur and metastasis soon occurs in the lungs. Most patients do not survive two years (K. H. Thoma, 1940). *Metastatic malignant deposits* may occur in the jaw, and may be osteolytic or osteoblastic. *Multiple myelomatosis* may affect the jaw.

*Fibrous dysplasia* of the jaw arises in different forms, consequently the terminology is confused. A localised area of dysplasia in the bone may consist of variable amounts of osteoid, fibrous or calcified tissue, and proliferation and resorption of the bone trabeculae may take place in addition to metaplasia of the abnormal bone; thus such conditions variously called fibro-osteoma, ossifying fibroma and fibroma may develop. These benign malformations arise in early adult life and grow slowly, causing deformity of the jaw. The radiological appearances vary with the amount of calcification, osteoid tissue and cyst formation.

*Leontiasis ossea* is a curious, rare entity and is due to a localised fibrous dysplasia of the maxilla, spreading to the facial bones and skull. It causes prominence of the alveolar ridge, thus giving the patient a leonine appearance. The disease encroaches on the air sinuses and orbit. It is a slow-growing process. A similar dysplasia occurring in infancy, gives

## SECTION VII

### THE NECK

by

D. L. B. FARLEY AND ARNOLD S. JACKSON

#### CHAPTER 66

### SWELLINGS OF THE NECK

**SURGICAL** diseases of the neck include many distinct pathological conditions, in most of which the presence of a swelling leads the patient to seek advice. The swelling may be of acute or slow onset. It may contain fluid or air, or it may be solid. Pain may be present or absent. Sometimes a swelling is associated with an opening in the skin.

#### ACUTE SWELLINGS

Acute swellings are usually inflammatory, e.g. a furuncle, carbuncle, erysipelas, acute lymphadenitis or cellulitis. Hæmorrhage into a cyst, surgical emphysema and angioneurotic œdema are occasional causes.

**A Furuncle, or Boil.** This is a staphylococcal infection of a hair follicle or sebaceous gland. It occurs most commonly at the back of the neck, or, in the male, in the beard area. The skin becomes red, raised and tender. Usually there is some necrosis of tissue surrounded by a firm abscess wall. After a few days, the necrotic tissue is discharged as a slough (the core), after which healing is usually rapid. Infection may, however, spread to other sebaceous glands or hair follicles, so that a "crop" of boils results. Sometimes a boil resolves without discharging ("blind" boil). A simple dressing may be all that is required, but the application of dry heat hastens natural discharge from a boil. To protect the surrounding skin from infection, shaving should first be undertaken and the skin then painted with spirit. If a boil becomes very painful, but does not discharge, it should be incised under general anaesthesia.

**A Carbuncle.** This, also, is a staphylococcal infection that begins in the skin but spreads subcutaneously. It is most common on the back of the neck, and appears as a painful, tender and indurated area in the skin. The skin becomes reddened, and after about a week pus discharges through several sinuses. Later the central skin sloughs, and healing occurs by granulation. Complications are rare to-day, but include septicæmia and osteomyelitis. *Treatment.*—Systemic penicillin limits the spread of

dense and ill-defined shadows, often with surrounding areas of bone erosion. Biopsy is essential with any unilateral polypus or bleeding mass.

Treatment consists of a flexible combination of radiotherapy and diathermy excision in most cases. If the sinuses are infected or filled with growth, surgery should precede irradiation, but surgery alone is indicated for radioresistant growths such as osteosarcoma. In all other cases, radiotherapy is followed by excision. Palatal fenestration with subsequent diathermy coagulation may suffice, but there is a growing tendency to remove the whole of the upper jaw whenever surgery is undertaken. The cavity is packed with gauze and later replaced by a dental prosthesis and, provided that this is performed at an early stage, the resultant deformity is surprisingly slight.

had time to form, and septic thrombophlebitis. On examination, there is an indurated swelling in the neck below the angle of the jaw and severe œdema of the floor of the mouth. Toxæmia is considerable. *Treatment.*—Preventive treatment includes adequate dental hygiene, the removal of septic tonsils, and the treatment of salivary calculi and sialitis. In its early stages, acute deep cellulitis usually responds rapidly to antibiotics, but operation is necessary if the swelling increases. Surgery is primarily required to release tension, and therefore *fluctuation should not be awaited.* Endotracheal anæsthesia is essential. A curved incision is made below the angle of the jaw, and deepened through the fascia until the submandibular salivary gland is exposed. Sufficient decompression is usually effected if the deep part of this gland is then dislocated from under the mylohyoid muscle. Further transverse incisions through the deep fascia should be made if the release of tension is inadequate. Respiratory distress from œdema of the glottis urgently demands tracheal intubation or tracheotomy (Chap. 68).

#### **Acute Retropharyngeal Abscess.**

This is due to adenitis in the retropharyngeal space, and is most common in childhood. It leads to dysphagia and pyrexia. Later, dyspnoea occurs. There is a visible and palpable swelling in the posterior pharyngeal wall. Spontaneous rupture of such an abscess may prove fatal from inhalation of pus. The abscess should be opened into the pharynx. Endotracheal anæsthesia is the best. Otherwise, anæsthesia should be very light so that the cough reflex is not abolished. The patient's head should be lowered over the edge of the table and pus immediately sucked away in order to prevent inhalation pneumonia or lung abscess.

**Chronic Retropharyngeal Abscess.** This may be pyogenic, but is usually due to tuberculous adenitis, or to tuberculous disease of the cervical spine. The swelling is slower in developing than in the acute variety, and the symptoms less dramatic. On radiography, evidence of tuberculosis of the spine may be seen. The abscess is aspirated, or drained, through a



FIG. 453. Ludwig's angina in a boy of thirteen.

the infection ; later, the application of urea crystals assists separation of the slough. Short-wave diathermy also hastens healing. As there is no tangible abscess cavity, incision of a carbuncle does not lead to worthwhile drainage of pus, and is therefore usually unnecessary. It should, however, be performed in patients with severe pain. A cruciate incision is made, and the four corners of skin are excised and the slough removed.

**Erysipelas.** This is a streptococcal infection of the skin. The affected area is raised and has a well-marked limit. Pus does not form, but vesicles may be seen in the skin. Erysipelas is often accompanied by enlargement of the regional lymph nodes and severe toxæmia. Response to antibiotics is rapid.

**Cellulitis of the Neck.** Three main layers can be recognised in the deep fascia which binds the various structures of the neck together. The *investing fascia* lies beneath the platysma muscle. It passes from the ligamentum nuchæ, invests the trapezius and sternomastoid muscles, and is continuous over the midline with the corresponding sheet of fascia from the other side. Superiorly, it is fixed to the mandible, to the zygomatic arch (after investing the parotid gland), the mastoid process, and the occipital bone. It is attached below to the shoulder girdle ; it is also firmly attached to the hyoid bone. The *prevertebral fascia* covers the prevertebral muscles. It is separated from the pharynx by loose areolar tissue containing lymph glands, known as the retropharyngeal space. This space extends downwards into the posterior mediastinum. The *pretracheal fascia* invests the thyroid gland ; below, it is continued into the mediastinum. These fascial layers are of considerable surgical importance, as inflammation beneath them can only reach the surface with difficulty. Pus therefore tends to spread in other directions and thus may reach the mediastinum ; this complication is likely to be fatal. In certain situations, the fascial planes prove so resistant to spread of infection that the rise of tension is considerable and leads to surrounding œdema.

*Superficial cellulitis* of the neck differs in no way from cellulitis in other parts of the body.

*Cellulitis deep to the investing fascia* results from various causes, e.g. a wound, sepsis in the mouth or infection in a lymph gland. *Ludwig's angina* is an acute cellulitis in the region of the submandibular salivary gland, deep to the mylohyoid muscle and superficial to the hyoglossus (Fig. 453). It may follow infection of the salivary gland, but it is more commonly due to sepsis in the mouth (e.g. dental sepsis) or the throat (e.g. acute tonsillitis, or diphtheria), reaching the tissues of the neck through the submandibular lymph glands. The infection is limited by the attachments of the fascia to the mandible above, the parotid gland behind, and the hyoid bone below. Tension rises high, and the infection tends to travel towards the mouth, deep to the mylohyoid. Complications include œdema of the glottis, which is prone to occur even before pus has

cricoid cartilages may be confused with a thyroglossal cyst. The latter, however, is translucent and becomes elevated on protrusion of the tongue (*vide infra*). Lower in the neck a thyroid swelling may be simulated by a dermoid cyst, but the former moves up and down on swallowing. *Treatment* is excision through a transverse incision, but sublingual dermoids are best removed through the mouth.

**Thyroglossal Sinus** (Syn. Median Cervical Sinus). The isthmus and the greater part of the body of the thyroid gland develop from the ventral wall of the pharynx between the base of the tongue and the tuberculum impar. The descent of the developing thyroid from this position results in the formation of a temporary duct which extends from the posterior part of the tongue to the thyroid isthmus (the thyroglossal duct). The developing hyoid bone usually divides this duct into two parts, but may encircle it. At an early stage in embryonic life, the duct becomes solid and disappears, and its former connection with the tongue is marked by the foramen cæcum. Failure of the thyroglossal duct to disappear completely may result in:—(1) A thyroglossal fistula; (2) a thyroglossal sinus; or (3) a thyroglossal cyst (*vide infra*). A thyroglossal fistula from the midline of the neck to the tuberculum impar is very rare. A thyroglossal sinus itself is seldom congenital, but usually results from sepsis in a congenital tract or cyst, or an unsuccessful attempt at excision of either of these. The sinus is lined by stratified or cuboidal epithelium, and in its wall accessory thyroid tissue may be found. The opening of a thyroglossal sinus usually lies in the midline, anywhere between the hyoid bone and the cricoid cartilage. It commonly has a crescentic upper margin due to inflammatory scarring and consequent shortening of the tract; it is drawn upwards on protrusion of the tongue. The sinus (or fistula) intermittently discharges glairy mucinous material. Attacks of inflammation commonly occur and result in a painful swelling and subsequent discharge of pus. *Treatment* is excision. An incision is made in a skin crease and includes the external opening of the sinus. A flap of skin is then turned upwards. A search is first made for any downward extensions of the tract; afterwards the tract is dissected upwards. As it has a variable attachment to the hyoid bone, no attempt should be made to dissect it off the bone in case a part of the tract is left behind. The middle part of the hyoid bone should therefore be resected, but care is taken not to injure the thyrohyoid membrane beneath. The tract is then followed up into the substance of the tongue. As the lingual muscles are adherent to it, the tongue should be cored out round the tract. This part of the dissection is made easier if an assistant's finger is placed in the mouth and pressed on the foramen cæcum. If the tract extends into the pharynx it is transfixed, divided and tied at its junction with the mucosa. During dissection care is taken to recognise and remove any diverticula arising from the tract.

**Thyroglossal Cysts.** These most commonly lie over the thyrohyoid

lateral incision in the neck ; if it is opened into the mouth (cf. acute retropharyngeal abscess) secondary infection is inevitable.

Infection deep to the *pretracheal fascia* sometimes complicates ulcerating growths of the larynx and other diseases of this organ ; it may also follow tracheotomy.

### CHRONIC SWELLINGS

These may be classified according to their contents :—(1) *Cystic Swellings*.—Sebaceous cyst, dermoid cyst, thyroglossal cyst, branchial cyst, lymphatic cyst, cystic hygroma, hydatid cyst, tuberculous abscess, subhyoid bursa, aneurysm, and meningocele. (2) *Solid Swellings*.—Swellings of the thyroid gland (Chap. 69), salivary glands (Chap. 62) or of lymphatic glands ; fibroma, lipoma, hæmangioma, neurofibroma, branchiogenetic carcinoma, cervical rib (Chap. 61), sternomastoid tumour, carotid body tumour. (3) *Air-filled Swellings*.—Pharyngeal pouch, œrocele, and pneumatocoele.

Certain chronic swellings of the neck are associated with an external opening through the skin, or with an internal opening into the mouth or pharynx. Occasionally there is a fistula, i.e. both an external and an internal opening are present. A *branchial sinus* is usually congenital in origin, whereas other abnormal openings are usually acquired from sepsis, new growth, or injudicious incisions. Such sinuses include the *thyroglossal sinus*, those due to inflammatory and neoplastic diseases of the *lymph nodes*, and those in *actinomycosis*.

**Sebaceous Cysts.** These are common in the neck. Other common sites are the scalp and scrotum. They are caused by obstruction of sebaceous glands due to inflammation or plugging with dirt. They are always attached to the skin, and have a tendency to become infected and then to discharge. The contents are cheese-like and have an unpleasant odour. *Treatment*.—If acutely inflamed, surgical treatment of a sebaceous cyst should be confined to drainage of pus ; excision is undertaken when the inflammation has subsided. It is essential to remove the entire capsule of the cyst, otherwise the cyst will recur. Under local anæsthesia, the cyst is either dissected out in one piece, or it is divided right across and the two halves are removed separately. The latter method provides easier access to the deepest part of the cyst which, if its wall is thin, may otherwise be incompletely removed.

**Dermoid Cysts.** These occur at any line of embryological fusion. In the neck, they usually lie in the midline, most commonly above the hyoid bone. If situated beneath the floor of the mouth, a dermoid cyst causes elevation of the tongue. A dermoid cyst is soft and fluctuant, but not translucent. Unless previously infected, it is unattached to skin. The cyst wall consists of fibrous tissue lined by stratified epithelium, together with sweat glands, hair follicles and hairs. The contents are cheesy and large hairs are often present. A dermoid lying over the thyroid or

young children unless there are attacks of inflammation; it is best performed when the patient is six to ten years old. A lubricated ureteric catheter is passed into the sinus as far as it will go and anchored to the skin by sutures (Small). A curved incision convex downward is made below the angle of the jaw and the tract is located (Fig. 454). It is then dissected up to the pharynx where it either terminates as a blind end, or, less commonly, opens into the pharynx (fistula). In the latter case, it is tied and divided flush with the pharyngeal wall. The tract is finally



FIG. 454. Operative excision of a branchial sinus (see text). A ureteric catheter has been passed through the external opening of the sinus. Note the classical situation of the latter. Note also the width of the tract on dissection. (Courtesy of Mr. H. A. D. Small.)

dissected down to the external opening and excised together with a small circle of skin. The catheter remains in place throughout the operation and is removed still within the tract.

**Branchial Cysts.** It is generally believed that branchial cysts arise from persistence of part of the cervical sinus or the second or third branchial clefts. Although a branchial cyst may be seen in childhood it is often not prominent until adult life. It is most often found below and behind the angle of the jaw (Fig. 455), but occasionally lies more posteriorly. On contraction of the sternomastoid muscle against resistance, its relationship to this muscle can be demonstrated. The cyst usually bulges from beneath its anterior border; it never lies superficial or posterior to the muscle. A branchial cyst varies in size, and may become very large. It is not translucent and does not move on deglutition. The lining membrane, which is usually squamous epithelium, lies on a base of fibrous



membrane, but may occur anywhere from within the substance of the tongue to the suprasternal notch. The cyst is usually midline, but one lying superficial to the thyroid cartilage may be found to one side, usually the left. A thyroglossal cyst is rarely seen in infancy, but usually makes its appearance about the age of ten. It may, however, persist into middle age without symptoms. The cyst is smooth, fluctuant and translucent. It is movable from side to side but not up and down. It moves upwards on protrusion of the tongue owing to its attachment to the hyoid bone. If an attempt is made to pull the cyst down away from the hyoid bone, a fibrous cord may be palpated between the two structures. A thyroglossal cyst contains semi-turbid fluid, which may be bloodstained and contain cholesterol crystals. The lining epithelium may be squamous, cuboidal or columnar. Sometimes thyroid tissue is present. A thyroglossal cyst in the neck has to be differentiated from a dermoid cyst, tuberculous abscess, subhyoid bursa and thyroid adenoma. In the tongue it has to be distinguished from an aberrant thyroid gland ("lingual thyroid"). *Treatment.*—Thyroglossal cysts commonly become infected and should therefore be removed. It is never safe to assume that the cyst is the only remnant of the thyroglossal duct. Therefore a tract must also be looked for and, if present, completely removed. If this is not done, recurrence with sinus formation is certain. When the cyst has been dissected out, any tract is dealt with as previously described.

**Branchial Sinus** (Syn. Lateral Cervical Sinus). In the four-week-old embryo, the second branchial arch grows caudally and envelops the third and fourth arches to form the cervical sinus. Later, the second arch fuses with the fifth arch and the sinus disappears. Branchial sinus and branchial fistula are popularly believed to be due to failure of obliteration of the cervical sinus. Some authorities, however, believe them to result from persistence of the thymopharyngeal duct which accompanies the developing thymus gland in its descent from the third branchial pouch. A branchial sinus is more commonly seen than a fistula. The latter consists of a complete tract from the skin to the tonsillar fossa (or just behind it). A congenital branchial sinus discloses its presence at birth or soon afterwards by the discharge of mucus through the external opening which is usually situated over the anterior border of the sternomastoid muscle in the lowest third of the neck. The condition may be bilateral, and is sometimes accompanied by other congenital anomalies, e.g. an accessory auricle. The sinus is lined by squamous or columnar epithelium, and passes upwards deep to the twelfth cranial nerve, and between the internal and external carotid arteries. Attacks of infection may occur and lead to pain and an increase in discharge which may become purulent. Apart from the developmental variety, an acquired branchial sinus may result, though less commonly, from infection of a branchial cyst—itsself congenital (*vide infra*). *Treatment.*—Sometimes the symptoms are so slight that surgery is never required. Operation should be avoided in

young children unless there are attacks of inflammation ; it is best performed when the patient is six to ten years old. A lubricated ureteric catheter is passed into the sinus as far as it will go and anchored to the skin by sutures (Small). A curved incision convex downward is made below the angle of the jaw and the tract is located (Fig. 454). It is then dissected up to the pharynx where it either terminates as a blind end, or, less commonly, opens into the pharynx (fistula). In the latter case, it is tied and divided flush with the pharyngeal wall. The tract is finally



FIG. 454. Operative excision of a branchial sinus (see text). A ureteric catheter has been passed through the external opening of the sinus. Note the classical situation of the latter. Note also the width of the tract on dissection. (Courtesy of Mr. H. A. D. Small.)

dissected down to the external opening and excised together with a small circle of skin. The catheter remains in place throughout the operation and is removed still within the tract.

**Branchial Cysts.** It is generally believed that branchial cysts arise from persistence of part of the cervical sinus or the second or third branchial clefts. Although a branchial cyst may be seen in childhood it is often not prominent until adult life. It is most often found below and behind the angle of the jaw (Fig. 455), but occasionally lies more posteriorly. On contraction of the sternomastoid muscle against resistance, its relationship to this muscle can be demonstrated. The cyst usually bulges from beneath its anterior border ; it never lies superficial or posterior to the muscle. A branchial cyst varies in size, and may become very large. It is not translucent and does not move on deglutition. The lining membrane, which is usually squamous epithelium, lies on a base of fibrous

tissue. The contents of the cyst, which may be clear or pultaceous, consist of epithelial debris and numerous cholesterol crystals. Branchial cysts may be complicated by infection, but this is less common than in thyroglossal cysts. Hæmorrhage into a cyst, and the development of carcinoma, have been reported. Confusion between a branchial cyst and a tuberculous abscess is common. In the latter condition, however, other glands are enlarged. Aspiration is often recommended as a diagnostic test. Although in many cases differentiation is possible, either by finding



FIG. 455. A branchial cyst.

acid-fast bacilli in a tuberculous abscess, or large quantities of cholesterol crystals in a branchial cyst, this test is not entirely reliable, as tuberculous pus sometimes contains cholesterol crystals and acid-fast bacilli are not always to be seen. *Treatment.*—Unless there has been previous infection, a branchial cyst can usually be removed without difficulty, but care must be taken to look for and remove any prolongations between the carotid vessels.

**Lymphatic Cysts.** These may be single or multiple. They usually occur in adults and are most often found in the pos-

terior triangle. Their removal usually presents no difficulty. *Cystic hygroma* is described in Chap. 77.

**Tuberculous Abscesses.** These are most commonly due to tuberculous adenitis (*vide infra*). Other causes include tuberculous disease of the cervical spine and of the laryngeal cartilages.

**Subhyoid Bursa.** This is more common in elderly patients. It lies in the midline immediately below the hyoid bone and may be confused with a thyroglossal cyst, but the shape of a bursa is characteristic for its long axis lies transversely. If disfiguring, or complicated by infection, a subhyoid bursa should be removed.

**Carotid Aneurysm.** This has an expansile impulse. It must be differentiated from the more common S-shaped arteriosclerotic elongation of the artery.

**Pharyngeal Pouch.** (see Chap. 44).

**Aerocele.** This is usually due to herniation of the mucous membrane of the air passages in the region of the larynx. It is often described as a particular hazard of wind instrument players, glass blowers and muezins. Although more common in adults, a laryngocele is occasionally seen in

childhood (Fig. 456). An *ærocele* is most prominent on coughing or on blowing the nose. The swelling is resonant to percussion. As it subsides mucus may accumulate in the throat. The term *ærocele* is also applied to free air in the neck resulting either from perforation of a diseased larynx, or from a tuberculous cavity in the apex of the lung. *Treatment*.—Small *æroceles* may be excised, but large ones are best treated by some form of support.

**Pneumatocoele.** This is an air-filled swelling lying in the lower part of the neck. It is due to herniation of the lung through Sibson's fascia.

**Lipomas.** These are soft, lobulated swellings which in the neck are usually localised, but may be diffuse. They may be subcutaneous or subfascial. Their excision is usually easy.

**Neurofibromas.** These tumours are occasionally seen arising from the peripheral cervical nerves or from cranial nerves entering the neck. They present as smooth firm swellings, and diagnosis is usually only possible after excision. A plexiform type of neurofibroma may occur in von Recklinghausen's disease.

**Branchiogenetic Carcinoma.** This is very rare, and is said to arise in vestigial rests from the branchial arches. It is a very

malignant squamous carcinoma situated under the uppermost third of the sternomastoid muscle. It is partly cystic and often contains some lymphoid tissue. Some authorities question the origin of this tumour and consider it to be secondary malignant disease of a lymph gland from an undetected primary growth. *Treatment*.—Excision is rarely possible, but if successful the patient must be repeatedly examined in case a primary carcinoma in the nose or throat has been overlooked.

**Sternomastoid Tumour.** This swelling is usually first noticed at birth or shortly afterwards. It disappears after some months. The tumour is due to ischæmic necrosis of the sternomastoid muscle, either *in utero* or as a result of birth trauma. The sternomastoid muscle may be short at birth, or contracture may result after a few years. Often there is an associated facial asymmetry, in some cases with asymmetry of the skull



FIG. 456 A laryngocele (type of *ærocele*) in a boy of four. He is straining in order to make it prominent. (Courtesy of Mr. Denis Browne.)

as well. *Treatment.*—Gentle manipulation is begun as soon as the condition is recognised in order to prevent further contracture. The infant's head should be rotated first to one side and then the other. This manoeuvre should be performed twice daily by the parents; one holds the baby and the other turns its head. The use of force is contraindicated, but after the "tumour" has subsided, more energetic passive movements may be carried out by a physiotherapist, and postural training is given as the child grows. Conservative measures sometimes fail to prevent torticollis; surgical correction should then be carried out.

**Carotid Body Tumour.** This is rare. It is more often benign than malignant; if malignant it does not metastasise. It arises from the carotid body lying within the V of the carotid bifurcation, and presents as a firm, round or oval swelling below the angle of the jaw and deep to the sternomastoid muscle. It grows slowly and tends to envelop the carotid vessels. The tumour is mobile transversely, but not up and down. Transmitted pulsation is always present. Symptoms from pressure on neighbouring structures include pain, hoarseness, cough, dyspnoea and Horner's syndrome. Reflex fainting from an exaggerated sinus reflex has been noted. The cut section of the tumour is pale; this, together with its rounded shape, accounts for its sometimes being called "potato" tumour. Microscopically the growth may be alveolar or peritheliomatous, and small eosinophilic polyhedral cells are seen, together with a variety of giant cells. *Treatment.*—The tumour is usually first thought to be an enlarged lymph node, and exploration is undertaken after investigations have proved negative. The tumour should be removed as it is not radio-sensitive, and may become malignant if not so already. The external carotid artery should be divided close to the tumour (i.e. above it). In this way a plane of cleavage can often be found between the under surface of the growth and the carotid vessels. If the vessels cannot be dissected free, resection of the arterial bifurcation must be considered. As there is a serious risk of hemiplegia following this procedure, it should be deferred until an arterial graft can be inserted to restore continuity between the common and the internal carotid arteries; this operation should be performed under hypothermia to reduce the metabolism of the brain until the graft is in place.

**Actinomycosis** is uncommon. The cervicofacial region is most often affected. A rare acute form of the disease may lead to cellulitis of the neck simulating Ludwig's angina.

**The Thyroid and Parathyroid Glands** (see under *The Surgery of the Endocrine Glands*).

## CHAPTER 67

### THE CERVICAL LYMPH GLANDS

**Tuberculous Cervical Lymphadenitis.** This disease is still common in Great Britain, although its incidence is decreasing. In the United States of America it is comparatively rare. The infection may be human or bovine. The latter used to be the more common, but owing to measures taken to avoid tuberculous contamination of milk this is not so to-day. Tuberculosis of the cervical glands is especially common in children, but no age is exempt and even elderly people may be affected.

*Natural History of the Disease.* The infection arises either through the lymph channels or via the blood stream. Lymphatic spread is the more common, and in the neck the jugulodigastric glands are usually the first to become enlarged. In this case the infection follows a primary tuberculous lesion in the mouth, nasopharynx or tonsil. Such a primary focus usually escapes clinical recognition, presumably because it is very small or has subsided before enlargement of the glands is apparent. As there are many communications between lymph nodes, once the infection gains a hold, it tends to jump from gland to gland, so that outlying nodes become infected. When tuberculous adenitis begins in the supra-clavicular region, however, spread from the chest is to be suspected. Infection through the blood stream results in a more generalised enlargement of glands from the start; the patients may be underweight, pyrexial or have evidence of active pulmonary, abdominal or genito-urinary tuberculosis.

In tuberculous adenitis, the glands are at first enlarged, firm and free from attachment. On section, they are pink and fleshy. In its early stages the disease cannot therefore be differentiated from chronic pyogenic (septic) lymphadenitis. In the majority of cases of tuberculous adenitis caseation and peri-adenitis later occur. There is then fixation of the glands to one another and to surrounding tissues. Softening of the glands from caseation leads to abscess formation within or around the glands. Later, calcification occurs. A tuberculous abscess arising from a gland beneath the investing layer of fascia may penetrate it and lead to a subcutaneous collection of pus ("collar-stud" abscess—Fig. 457). The defect in the fascia may be small, and the superficial part of the abscess may lie over the affected gland or at some distance from it. Although absorption of tuberculous pus may take place, breakdown of the skin overlying an abscess commonly occurs, with the formation of a sinus. This is preceded by thinning of the skin, which becomes dusky red or blue. A tuberculous sinus (Fig. 458) discharges serous fluid, tuberculous

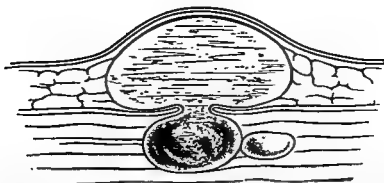


FIG. 457. A "collar-stud" abscess. Caseous tuberculous glands are shown lying beneath the investing fascia of the neck. The abscess from one of them is subcutaneous, having penetrated the fascia.

pus and caseous debris; it only heals when the whole of the necrotic gland has been discharged, during which time considerable scarring of the skin results. Secondary infection is common in softened glands, either from sepsis in the mouth or throat, or by direct invasion along a sinus track. Further glandular enlargement then occurs. This is partly the result of the secondary infection itself, and partly due to its causing a temporary lowering of the patient's resistance to the tuberculous condition.



FIG. 458. Tuberculous glands of the neck. One sinus is present and another is about to form. Note the tuberculous abscess below the angle of the mandible on the patient's left. (Courtesy of Mr. D. J. Waterston.)

*A primary tuberculous complex involving the mouth or throat and one or more cervical lymph glands may be symptomless. The affected glands become fibrosed and calcified, so that one or more hard nodes may persist. In a few patients, however, the primary reaction is severe, with fever, malaise and marked glandular enlargement. In these circumstances glands soften early. In the majority of patients, tuberculous lymphadenitis runs a long course, with recurring attacks of glandular enlargement, abscess formation and the development of sinuses. Relapses are prone to follow attacks of intercurrent infection, particularly in the throat. The E.S.R. may be raised and*

the *Mantoux reaction* is usually positive. Healing of the glands is accompanied by fibrosis and calcification. The latter is easily seen on X-ray examination (Fig. 459).

*Non-caseous tuberculous adenitis* of the neck is less common than the caseous variety. In the *lymphadenoid* type (Fig. 460) the glands show hyperplasia. They are only moderately enlarged and are discrete. They are found throughout the neck and axillæ. Since they are not fixed by



FIG. 459. Calcified tuberculous glands on the right side of the neck. (Courtesy of Dr. J. M. Stewart.)

peri-adenitis, the glands resemble those of Hodgkin's disease. Diagnosis can often only be made by biopsy of one of the nodes. In the *fibrous* type (more commonly seen in elderly patients), there is no hyperplasia and the glands become increasingly fibrotic. *Prognosis*.—Although troublesome to the patient, tuberculous adenitis is a relatively benign condition in the neck, and general dissemination is almost unknown.

*Medical Treatment*. Co-operation between physician and surgeon is essential. Preventive treatment includes care of the milk supply, and the segregation of adults with open pulmonary tuberculosis. B.C.G. inoculation is also becoming popular. Before treatment is begun, the patient's state of health must be determined, and a careful examination undertaken for evidence of tuberculosis elsewhere. In certain cases of chronic ill health or generalised disease, sanatorium treatment is advisable. In tuberculous adenitis restricted to the cervical glands, the aims of treat-



ment are:—(1) To prevent ill health and chronicity of the disease ; and (2) to produce a good cosmetic result. Open air, a high calorie diet and avoidance of fatigue are needed. Splinting the neck by means of plaster of Paris or a surgical appliance is recommended by some surgeons to restrict movement in the neck. Courses of calciferol may assist tuberculous glands to harden.

*Surgical Treatment.* Softening of tuberculous cervical glands is frequently followed by sinuses which heal slowly and result in

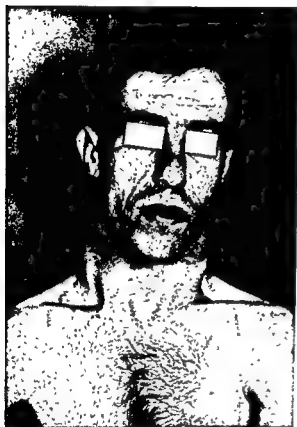


FIG. 460. Lymphadenoid tuberculosis of the neck.

secondary infection. Removal of pus and caseous debris by operation is therefore indicated in cases of abscess formation, particularly when the skin appears to be breaking down. It must be stressed that surgery is concerned primarily with removing pus and dead tissue than with excising all possible contaminated lymph nodes. Such treatment may be combined with chemotherapy (*vide infra*). Surgical methods include aspiration, curettage, expression and excision of tuberculous glands. *Aspiration* of a cold abscess under local anaesthesia usually fails to prevent the development of a sinus, even if streptomycin is injected locally. Although of value in diagnosis, aspiration is therefore of little therapeutic value. *Excision* of

tuberculous glands is undertaken when the infection is confined to a single group of glands which remain persistently enlarged. The presence of a cold abscess or a sinus is not a contraindication, but surgery should not be attempted when enlarged glands lie just above the clavicle and are possibly adherent to the apex of the lung. The operation is performed through a collar incision which includes any sinuses that are present, and dissection should begin in an area of normal anatomy. All obviously infected glands and diseased tissues are dissected out and removed ; care is taken to avoid damage to important structures, such as the internal jugular vein and the accessory nerve. The wound is closed without drainage to reduce the risk of secondary infection. Complete haemostasis is the aim, but any post-operative haematoma should be aspirated. When

tuberculous lymphadenitis is unsuitable for excision, i.e. it is recent, or not confined to a single group of glands, the *incision and curettage* of softened glands may be performed, or curettage alone may be carried out through an existing sinus. These procedures hasten resolution and also allow confirmation of the diagnosis by microscopy. *Expression* (Denis Browne) is helpful in soft glands which are not too deeply placed, and is of particular value in children. A small incision is made over the gland and firm pressure is applied by the fingers and thumb for a few minutes so that all necrotic tissue is squeezed out. Afterwards the skin is sutured. *Tonsillectomy and adenoidectomy*.—Most surgeons agree that the tonsils and adenoids should be removed, even if they are clinically normal, in children with certain or suspected tuberculous adenitis. These procedures should be undertaken before tuberculous glands are excised. Small and apparently innocent tonsils may on serial section show evidence of tuberculosis; furthermore, removal of the tonsils may lessen the risk of secondary infection, from a septic throat, developing later in tuberculous glands. In adults, tonsillectomy is advisable if the tonsils are chronically inflamed. Some surgeons believe that their removal is of benefit in all cases of tuberculous adenitis.

*The Anti-tuberculous Drugs.* At the present day, streptomycin, P.A.S. and Isoniazid take second place to general and surgical measures, and they should not be given routinely owing to their inherent dangers. In order to discourage insensitivity, they should always be given in combination. Although courses of three weeks' duration lead to reduction in the size of glands that have not softened, abscess formation commonly occurs when the drugs are withdrawn. A short course is, however, of value in patients with an acute reaction to a primary complex. Long courses (e.g. six months to a year), combined with such surgical treatment as may be necessary, may be given to patients with persistent or relapsing infection. It must be admitted, however, that the efficacy of medical treatment is difficult to assess, for the disease rarely runs a steady course and there is a strong tendency to natural cure.

*Primary Neoplasms of the Cervical Glands.* A swelling in the neck is the presenting symptom in many cases of Hodgkin's disease and lymphosarcoma. In Hodgkin's disease, the enlarged glands appear in groups, and, in the neck, those first involved are usually in the posterior triangle. Although the glands become very large they remain discrete; this usually serves to differentiate them from tuberculous glands. Subsequently, glandular enlargement may appear in the axilla and groin, and on X-ray mediastinal involvement may be seen. In lymphosarcoma, the disease tends to invade the capsule of the lymph glands and extend to surrounding tissues. Later, the growth may invade the skin so that it becomes reddened and even appears to fluctuate. Primary lymphatic neoplasms are further considered in Chap. 77.

*Secondary Malignant Disease of the Cervical Glands.* The cervical

ment are:—(1) To prevent ill health and chronicity of the disease; and (2) to produce a good cosmetic result. Open air, a high calorie diet and avoidance of fatigue are needed. Splinting the neck by means of plaster of Paris or a surgical appliance is recommended by some surgeons to restrict movement in the neck. Courses of calciferol may assist tuberculous glands to harden.

*Surgical Treatment.* Softening of tuberculous cervical glands is fre-

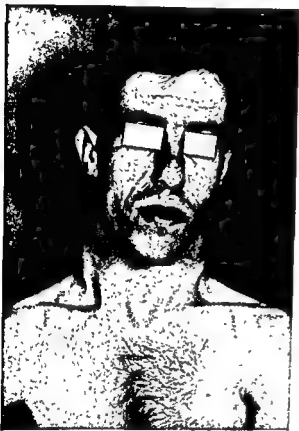


FIG. 460. Lymphadenoid tuberculosis of the neck.

quently followed by sinuses which heal slowly and result in secondary infection. Removal of pus and caseous debris by operation is therefore indicated in cases of abscess formation, particularly when the skin appears to be breaking down. It must be stressed that surgery is concerned primarily with removing pus and dead tissue than with excising all possible contaminated lymph nodes. Such treatment may be combined with chemotherapy (*vide infra*). Surgical methods include aspiration, curettage, expression and excision of tuberculous glands. *Aspiration* of a cold abscess under local anaesthesia usually fails to prevent the development of a sinus, even if streptomycin is injected locally. Although of value in diagnosis, aspiration is therefore of little therapeutic value. *Excision* of

tuberculous glands is undertaken when the infection is confined to a single group of glands which remain persistently enlarged. The presence of a cold abscess or a sinus is not a contraindication, but surgery should not be attempted when enlarged glands lie just above the clavicle and are possibly adherent to the apex of the lung. The operation is performed through a collar incision which includes any sinuses that are present, and dissection should begin in an area of normal anatomy. All obviously infected glands and diseased tissues are dissected out and removed; care is taken to avoid damage to important structures, such as the internal jugular vein and the accessory nerve. The wound is closed without drainage to reduce the risk of secondary infection. Complete haemostasis is the aim, but any post-operative haematoma should be aspirated. When

neck should be operated upon at a time. At the second procedure (approximately three weeks later) the internal jugular vein should be preserved in order to prevent rise of intracranial pressure ; if, however, a period of several months has elapsed since the first operation, it is safe to tie the vein on the second side. Block dissection of malignant glands of the neck carries a mortality of about 5 per cent., but it is of great value in selected cases, and results in little deformity. It is not considered to be worth while in cancer cases unless the glands are palpably enlarged. Malignant cervical glands which are *fixed* should be treated by radiotherapy if the primary growth is under control. Such treatment is of considerable palliative value.

glands may be secondarily involved in lymphosarcoma and in the leukæmias. *Carcinomatous* lymph glands in the neck are, as elsewhere, at first mobile and later hard and fixed. Eventually the skin becomes involved, with sinus development. Enlargement of one or more lymph glands in the neck may be the first evidence of carcinoma. In the majority of cases the primary growth is situated above the clavicle, most commonly in the mouth or pharynx (from the nasopharynx to the pyriform fossa). The primary growth may, however, lie below the clavicle, e.g. in the lung, œsophagus, stomach, kidney or breast. Occasionally, growths of the pelvic organs, and even the testicle, first manifest themselves by metastasis in the supraclavicular glands. Accordingly, the finding of a hard gland in the neck demands a thorough medical examination, an X-ray of the chest and a full blood count. Particular care must be taken to examine the rest of the neck, the skin of the scalp and face, the ear, the mouth and the throat. X-rays of the throat and of the air sinuses, anterior and posterior rhinoscopy, indirect and direct laryngoscopy and œsophagoscopy must also be undertaken if a primary growth within the territory of the ear, nose or throat is not to be overlooked. If these investigations are negative, a barium swallow, barium meal and intravenous pyelogram must be carried out. Biopsy excision of an enlarged gland is of great value in diagnosis, but removal of a part of a mass of fixed growth may be followed by fungation, and is therefore to be avoided.

*Treatment.* The primary growth is first treated, either by radiotherapy or surgery, as necessary. If the primary growth is "curable" and the glands in the neck are *freely mobile* they should be removed *en bloc*. In carcinoma of the lip, the anterior part of the floor of the mouth, or the tip of the tongue, the dissection may be limited to both sides of the neck above the hyoid bone, unless any glands are enlarged outside this area. In carcinoma at other sites in the head and neck a complete dissection of the neck is necessary; sometimes this must be done on both sides. *Block Dissection of the Neck (Crile's Operation).*—The operation consists of wide removal of lymphatic glands of the neck, together with the investing fascia, sternomastoid muscle and internal jugular vein. The skin incision runs along the length of the sternomastoid muscle, and an extension is made from this incision towards the chin. The skin flaps are dissected up over the edge of the mandible, the clavicle, across the midline, and posteriorly as far as the anterior border of the trapezius muscle. The investing fascia is divided at the limits of the wound. The sternomastoid muscle is divided at its origin, and the internal jugular vein tied at this level. A block of tissue containing these structures, the carotid sheath and all the lymph glands, is dissected upwards. During this procedure the omohyoid muscle, the submandibular salivary gland and the lower pole of the parotid gland are excised. The internal jugular vein is finally tied as high as possible and excised. The skin flaps are lightly sutured and a drain inserted. When bilateral dissection is necessary, one side of the

sternum. Bleeding may occur from the anastomosis between the anterior jugular veins of the two sides, but can be readily controlled with artery forceps. (2) *Midline Separation of the "Ribbon" Muscles.*—Strictly in the midline, the "ribbon" muscles—the sternohyoid and sternothyroid—are separated and retracted. (3) *Midline Division of the Thyroid Isthmus.*—The fascia which slings the thyroid isthmus from the cricoid cartilage is divided horizontally. The isthmus, thus mobilised, is clamped with artery forceps and divided vertically in the midline. (4) *Opening of the Trachea.*—The trachea should be opened at the level of its third and fourth rings—those which are exposed by division of the thyroid isthmus (Fig. 461). Parts of these rings are then excised, to allow easy introduction and withdrawal of the tracheotomy tube. (5) *Introduction of Tracheotomy Tube.*—A tube of appropriate size is introduced and secured by tapes tied round the neck.

The best one for most purposes is Durham's tube (Fig. 462). This consists of a rigid outer tube with a right-angled curve, and an inner "lobster-tail" tube which should project for at least  $\frac{1}{2}$  inch beyond the outer tube, to collect secretions or blood clot. An introducer is provided and there should be two inner tubes for each outer tube.

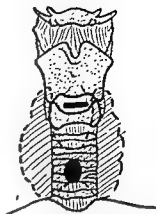


FIG. 461. The opening made through the trachea at the level of the third and fourth rings for tracheotomy, and the transverse incision through the crico-thyroid membrane for laryngotomy.

*After-care.* Both inner and outer tubes may be coughed out of position, and a tracheal dilator (Fig. 463) and introducer must therefore be immediately to hand at all times for at least the first week. The most important single requirement in the after-care of these patients is an efficient sucker, which should be used as often as necessary to remove the

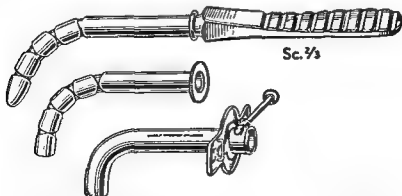


FIG. 462. Durham's tracheotomy outfit. From above downwards: The introducer, the inner "lobster tail" tube, and the rigid outer tube. (Courtesy of Chas. F. Thackray Ltd.)

## THE LARYNX AND PHARYNX

by

J. CHALMERS BALLANTYNE

## TRACHEOTOMY AND LARYNGOTOMY

THERE is no greater emergency in medical or surgical practice than the relief of impending asphyxia by tracheotomy or laryngotomy. Laryngotomy can be performed more easily and more rapidly than tracheotomy, but it has serious disadvantages, and is therefore permissible only in cases of the greatest urgency. In all other cases, when time and opportunity permit, tracheotomy is greatly preferred.

**Indications.** These procedures are most commonly required for the relief of stenosis in the larynx or upper trachea, but tracheotomy is being increasingly used for the suction of secretions from the tracheobronchial tree in unconsciousness or paralysed patients, as in head injuries, tetanus, severe pulmonary infections and the bulbar type of poliomyelitis. It also provides a means of applying positive pressure for artificial respiration. The one clinical feature common to all the causes of laryngeal and upper tracheal obstruction (too numerous to mention here) is *inspiratory stridor*—a noisy dyspnoea occurring during the inspiratory phase of respiration and usually accompanied by indrawing of the supraclavicular fossæ and the suprasternal space. It is not always easy to decide upon the exact point in time at which these operations should be undertaken, but a useful working rule (Suggitt) is that "if a patient has stridor going upstairs he *may* need a tracheotomy, if he has stridor walking about he *will* need one, and if he has stridor lying down he *does* need one."

**Tracheotomy.** Ideally a sandbag or small pillow should be placed under the shoulders so that the head and neck are extended. This brings the larynx and trachea nearer to the surface. The larynx is steadied in the midline between the thumb and middle finger of the left hand.

**Technique.** Tracheotomy is usually performed under local analgesia in adults. No anæsthetic is needed in infants, but general anæsthesia is normally safe for older children. The introduction of a bronchoscope allows of an unhurried operation, but should be avoided in cases of laryngeal cancer. (1) *Incision.*—Although some surgeons use a horizontal incision at the level of the thyroid isthmus, a vertical *midline* incision is preferred by the majority, especially in cases of emergency. This should extend from the thyroid notch (Adam's apple) to the upper border of the

sternum. Bleeding may occur from the anastomosis between the anterior jugular veins of the two sides, but can be readily controlled with artery forceps. (2) *Midline Separation of the "Ribbon" Muscles.*—Strictly in the midline, the "ribbon" muscles—the sternohyoid and sternothyroid—are separated and retracted. (3) *Midline Division of the Thyroid Isthmus.*—The fascia which slings the thyroid isthmus from the cricoid cartilage is divided horizontally. The isthmus, thus mobilised, is clamped with artery forceps and divided vertically in the midline. (4) *Opening of the Trachea.*—The trachea should be opened at the level of its third and fourth rings—those which are exposed by division of the thyroid isthmus (Fig. 461). Parts of these rings are then excised, to allow easy introduction and withdrawal of the tracheotomy tube. (5) *Introduction of Tracheotomy Tube.*—A tube of appropriate size is introduced and secured by tapes tied round the neck.

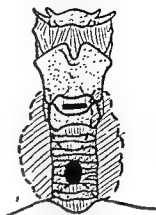


FIG. 461. The opening made through the trachea at the level of the third and fourth rings for tracheotomy, and the transverse incision through the crico-thyroid membrane for laryngotomy.

The best one for most purposes is Durham's tube (Fig. 462). This consists of a rigid outer tube with a right-angled curve, and an inner "lobster-tail" tube which should project for at least  $\frac{1}{2}$  inch beyond the outer tube, to collect secretions or blood clot. An introducer is provided and there should be two inner tubes for each outer tube.

*After-care.* Both inner and outer tubes may be coughed out of position, and a tracheal dilator (Fig. 463) and introducer must therefore be immediately at hand at all times for at least the first week. The most important single requirement in the after-care of these patients is an efficient sucker, which should be used as often as necessary to remove the

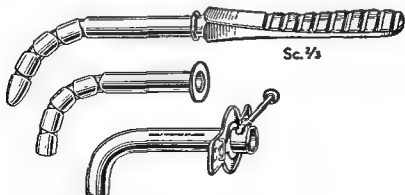


FIG. 462. Durham's tracheotomy outfit. From above downwards: The introducer, the inner "lobster tail" tube, and the rigid outer tube. (Courtesy of Chas. F. Thackray Ltd.)



## CHAPTER 68

# THE LARYNX AND PHARYNX

by

J. CHALMERS BALLANTYNE

### TRACHEOTOMY AND LARYNGOTOMY

THERE is no greater emergency in medical or surgical practice than the relief of impending asphyxia by tracheotomy or laryngotomy. Laryngotomy can be performed more easily and more rapidly than tracheotomy, but it has serious disadvantages, and is therefore permissible only in cases of the greatest urgency. In all other cases, when time and opportunity permit, tracheotomy is greatly preferred.

**Indications.** These procedures are most commonly required for the relief of stenosis in the larynx or upper trachea, but tracheotomy is being increasingly used for the suction of secretions from the tracheobronchial tree in unconsciousness or paralysed patients, as in head injuries, tetanus, severe pulmonary infections and the bulbar type of poliomyelitis. It also provides a means of applying positive pressure for artificial respiration. The one clinical feature common to all the causes of laryngeal and upper tracheal obstruction (*too numerous to mention here*) is *inspiratory stridor*—a noisy dyspnoea occurring during the inspiratory phase of respiration and usually accompanied by indrawing of the supraclavicular fossæ and the suprasternal space. It is not always easy to decide upon the exact point in time at which these operations should be undertaken, but a useful working rule (Suggitt) is that “if a patient has stridor going upstairs he *may* need a tracheotomy, if he has stridor walking about he *will* need one, and if he has stridor lying down he *does* need one.”

**Tracheotomy.** Ideally a sandbag or small pillow should be placed under the shoulders so that the head and neck are extended. This brings the larynx and trachea nearer to the surface. The larynx is steadied in the midline between the thumb and middle finger of the left hand.

**Technique.** Tracheotomy is usually performed under local analgesia in adults. No anæsthetic is needed in infants, but general anæsthesia is normally safe for older children. The introduction of a bronchoscope allows of an unhurried operation, but should be avoided in cases of laryngeal cancer. (1) *Incision.*—Although some surgeons use a horizontal incision at the level of the thyroid isthmus, a vertical *midline* incision is preferred by the majority, especially in cases of emergency. This should extend from the thyroid notch (Adam's apple) to the upper border of the

followed by permanent damage to the larynx, with subsequent stenosis, if the tube is left in position for more than a few hours. It should therefore be replaced by a tracheotomy as soon as circumstances allow.

Tracheotomy and laryngotomy are not always easy operations, but they are life-saving procedures and every medical practitioner should be prepared to undertake them when urgency demands. It therefore behoves every doctor to make himself familiar with their techniques.

### LARYNGEAL INJURIES

The lax structures of the laryngeal inlet may be involved in severe oedematous reactions through a variety of traumatic influences—all of them rather rare. Burns or scalds may result from the inhalation of irritant fumes or gases, the swallowing of corrosive fluids, or the inhalation of steam; local reactions may follow the use of radiotherapy in the treatment of malignant laryngeal neoplasms, and foreign bodies may become impacted at or above the level of the glottis. Urgent dyspnoea requiring tracheotomy or laryngotomy may be caused by any of these conditions, but the general surgeon is perhaps more often concerned with direct injuries of the larynx. These may be due to compression or to penetrating wounds.

**Compression Injuries.** These result from blows or strangulation. External bruising of the skin may be slight, but its absence does not preclude serious intralaryngeal damage. Surgical emphysema involves the tissues of the neck in the first instance, but may later spread to the face, chest, abdomen and upper limbs. Submucosal hæmorrhages may appear on the vocal cords or on any other part of the interior of the larynx, and adhesions may develop between the opposed surfaces of an abraded or lacerated laryngeal mucosa. Stenosis follows in some cases and perichondritis of the cartilages sometimes results if infection supervenes. Fractures of the laryngeal cartilages may accompany these changes; the thyroid cartilage is most commonly involved because of its exposed situation. Fractures of the cricoid cartilage are, however, more serious, for they lead to early infiltration of blood below the level of the vocal cords, and nearly all recorded cases have been fatal.

Dyspnoea may be marked and of sudden onset, and hoarseness is common. Hæmoptysis is usually slight, but local pain and tenderness may be present. Radiography may confirm the presence of a fracture in an ossified cartilage. The chief danger of these injuries is interference with respiration, and tracheotomy may be urgently required. Penicillin should be given prophylactically in every case and must be continued if perichondritis threatens or supervenes. Subsequent stenosis, or fractures of the laryngeal cartilages, may require open operation at a later date.

**Penetrating Wounds.** These are caused by war injuries, stabs or cuts. They are usually fatal because the laryngeal injuries are generally accompanied by damage to vital structures. Oblique wounds usually

secretions which inevitably collect in the early days after tracheotomy. The inner tube should be removed whenever there is any sign of blockage, and replaced immediately by the second inner tube while the first is being cleaned and sterilised. The outer tube should be changed at the end of the first week. If a tracheotomy is to be permanent, a valved inner tube can be substituted at a later date, to facilitate speech.

**Complications.** The most serious complication of tracheotomy and laryngotomy is immediate respiratory failure. When laryngeal obstruction has been present for a long time there is a rise in the carbon dioxide in the alveoli and blood. The free ventilation afforded by the operation

washes the carbon dioxide out of the alveoli, and the carbon dioxide in the blood falls. Hence the normal stimulus to the respiratory centre is removed and failure may ensue. Carbon dioxide should therefore be readily available whenever possible and admin-

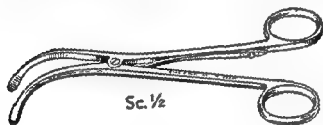


FIG. 463. A tracheal dilator. (Courtesy of Chas. F. Thackray Ltd.)

istered without delay if there is any sign of respiratory failure. Reactionary hæmorrhage may be serious and accidental damage to the right pleura may cause pneumothorax and mediastinal emphysema. Surgical emphysema in the neck may result from too tight a closure of the skin wound. Stenosis of the upper trachea or subglottic larynx will almost certainly follow, as a late sequel, if the opening into the trachea is made too high.

**Laryngotomy.** The same position is adopted as for tracheotomy, with the head and neck extended. Since the operation should be performed only in cases of dire emergency, an anæsthetic is rarely required. Running the index finger of the left hand downwards from the thyroid notch and strictly in the midline, the operator can easily feel a depression below the lower border of the thyroid cartilage. This overlies the cricothyroid membrane, through which entry is gained to the larynx (Fig. 461). After making a short *horizontal* skin incision at this level, the knife (still held horizontally) is thrust through the membrane which lies much more superficially than the trachea. Scissors may be used if a knife is not at hand. If available, an oval laryngotomy tube, flattened from above downwards, is introduced immediately and secured in position by tapes; otherwise the opening must be kept patent by turning the blade of the knife or by any other suitable object available, such as a safety pin. Hæmorrhage is usually slight, but damage to the cricothyroid artery may produce troublesome bleeding which must be controlled without delay after widening the incision and deepening the exposure. Despite the usual ease and apparent simplicity of this procedure, it is almost inevitably

radiotherapy, but there is so far no uniformity of opinion either about the classification or the treatment of *growths of the laryngopharynx*.

Most of the growths previously described as extrinsic laryngeal growths should be included with the pharyngeal group, for they belong correctly to the pharynx in a strictly anatomical sense; they behave like growths in other parts of the pharynx, in producing late symptoms and early metastasis, and their prognosis is correspondingly poor. Such growths include carcinomas of the pyriform fossa and the post-cricoid region (the very lowest part of the pharynx, behind the cricoid cartilage). Slight pain or discomfort in the throat are common in both of these types, and hoarseness may be caused by direct involvement of the larynx or paralysis of the recurrent laryngeal nerve on one or both sides, but the post-cricoid tumour is distinguished by three characteristic features: unlike all other growths in the pharynx, it is much commoner in women than in men (9:1); it tends to occur at an earlier age; and dysphagia, occurring relatively early in the disease, is often preceded by a long history, sometimes of many years, of a "small swallow." This is due to a condition of chronic pharyngitis in the laryngopharynx, commonly known as the Paterson-Brown Kelly or Plummer-Vinson syndrome. This consists of a "small swallow" with microcytic hypochromic anæmia, superficial glossitis, cracks at the angle of the mouth and koilonychia (spoon-shaped nails). It is now firmly established as a precancerous condition.

Most of the very few cures of cancers of the laryngopharynx have been achieved by surgery, but only at the expense of a mutilating operation (pharyngolaryngectomy). The prognosis is desperately poor with any form of treatment, but radiotherapy probably gives a longer average period of survival and is an invaluable palliative. It should therefore be the treatment of first choice in most cases, with the possible exception of younger patients, especially in early growths of the post-cricoid region and especially in the absence of clinical involvement of the cervical glands.

**Cancer of the Larynx.** Cancers of the larynx are, correctly speaking, those tumours which arise within the skeletal framework of the laryngeal cartilages. The vocal cord itself is the commonest site, but growths may also arise above or below this level, when symptoms tend to appear late. In true glottic carcinoma, however, hoarseness occurs very early and, thanks to the paucity of lymphatics in the edge of the cord, metastasis develops in rather less than 5 per cent. of early cases. Removal of the affected cord by *laryngofissure* (splitting of the larynx) gives about 80 per cent. of three-year cures, but leaves the patient with a permanently rough or weakened voice. It has been almost entirely superseded by external irradiation, which gives a slightly higher rate of cure and a perfect voice.

Total laryngectomy is usually indicated for subglottic or late cord

involve the large vessels of the neck and anteroposterior wounds tend to involve the cervical spine, but transverse wounds may involve the larynx alone. Survival is rare, however, even in this type of penetrating wound. Respiratory obstruction and hæmorrhage are the outstanding clinical features, and the only hope of survival is immediate institution of effective treatment. It is permissible to ligate one internal jugular vein; the common and internal carotid arteries should be repaired by direct suture if possible, or otherwise by grafting; other vessels are ligated. Asphyxia may be prevented by intubation through the open wound, but tracheotomy should be performed as soon as facilities permit, and penicillin should be given to prevent infection. No food should be given by mouth for at least twenty-four hours.

### CANCER OF THE PHARYNX AND LARYNX

The vast majority of malignant new growths of the throat are squamous-cell carcinomas. So greatly do cancers of the pharynx differ from those of the larynx in their behaviour and prognosis that they are best described separately.

**Cancer of the Pharynx.** The anatomist divides the pharynx into three parts, the nasopharynx, the oropharynx and the laryngopharynx. *Carcinoma of the nasopharynx* rarely presents with nasal symptoms though obstruction may be evident in proliferative growths, and epistaxis may be severe when ulceration occurs. Much more commonly the growth invades the base of the skull to produce neurological symptoms (which are discussed elsewhere); presses on the Eustachian tube, to cause a conductive deafness on the affected side; infiltrates the soft palate, with subsequent reduction in mobility; or spreads to the upper deep cervical glands, often bilaterally.

*Malignant growths of the oropharynx* occur not uncommonly in the tonsils; ulceration and secondary infection may occur early with a carcinoma, whereas they occur later with a sarcoma which may present clinically simply as a unilateral enlargement of one tonsil. The tonsillo-lingual sulcus is a common site of origin of carcinomas which may spread to the tonsil itself, to the posterior one-third of the tongue, to the palate, or to the upper or lower alveolus. Cervical glandular metastasis occurs early, and may be the first symptom noticed by some patients with oropharyngeal growths. Soreness of the throat has, however, usually been present for some time, and the true nature of the condition is often suspected only after the unsuccessful exhibition of antibiotics for what was thought to be a simple tonsillitis, a quinsy (peritonsillar abscess), or ulcerative tonsillitis due to Vincent's organisms. Referred pain in the ear is common.

It is almost universally agreed that most cancers of the nasopharynx and oropharynx, including their glandular deposits, are best treated by

## SECTION VIII

### THE SURGERY OF THE ENDOCRINE GLANDS

(THE THYROID, THYMUS, PARATHYROIDS AND ADRENALS)

by

J. E. PIERCY

#### CHAPTER 60

#### THE THYROID GLAND

THE thyroid is the largest ductless gland in the body ; its lateral lobes are connected by an isthmus of thin thyroid tissue extending across the second, third and fourth rings of the trachea. The lateral lobes are cone shaped and are moulded to the larynx and trachea. Each upper pole has a posterior wedge of tissue bound down to the pharynx by fascia. At operation, this wedge is stripped of its fascia, thus allowing dislocation of the upper pole to simplify ligation of the superior thyroid vessels. The thyroid gland is enclosed in an envelope of fascia derived from the deep cervical fascia and known as the surgical capsule. It has in addition an internal or true capsule intimately connected with and ensheathing the gland. Surgical dissection of the gland takes place between these two layers. The *pyramidal lobe*, when present, is a finger-like projection of thyroid tissue extending from the upper border of the isthmus, often as high as the hyoid bone. The thyroid gland is intimately connected with the larynx, and therefore moves upward on swallowing. This movement is important when diagnosing swellings of the thyroid. The *arterial supply* of the thyroid gland is richer than that of any other organ in the body. It is derived mainly from two sources : the *superior thyroid artery* which arises from the external carotid and runs downward to reach the upper pole, and the *inferior thyroid artery* which arises from the thyrocervical trunk and runs behind the carotid sheath to reach the posterolateral aspect of the lobe. If both these arteries were ligated, a collateral circulation would develop from the internal and external laryngeal, cricothyroid, lingual, tracheal and œsophageal arteries. A *thyroidea ima artery* may occasionally be present arising from the innominate or subclavian arteries ; it ascends to the lower border of the isthmus.

The *recurrent laryngeal nerves* run upwards, lying in or nearby the tracheo-œsophageal groove. Each nerve usually runs behind the inferior

growths, but preoperative radiotherapy may improve the results, especially in rapidly-growing tumours in younger subjects. A "pharyngeal" voice can nearly always be acquired with persistent effort on the part of the patient and the speech therapist.

To summarise, cancers of the pharynx are characterised by late onset of symptoms, early metastasis, and appalling prognosis; in sharp contrast to this depressing picture is the early glottic cancer of the larynx in which symptoms appear very early, metastasis is late, and prognosis equals or excels that of any other site. It cannot be too strongly emphasised that no patient should be allowed to escape examination with the laryngeal mirror who has suffered from hoarseness, dysphagia or discomfort in the throat for more than three weeks.

## SECTION VIII

### THE SURGERY OF THE ENDOCRINE GLANDS

(THE THYROID, THYMUS, PARATHYROIDS AND ADRENALS)

by

J. E. PIERCY

#### CHAPTER 69

#### THE THYROID GLAND

THE thyroid is the largest ductless gland in the body ; its lateral lobes are connected by an isthmus of thin thyroid tissue extending across the second, third and fourth rings of the trachea. The lateral lobes are cone shaped and are moulded to the larynx and trachea. Each upper pole has a posterior wedge of tissue bound down to the pharynx by fascia. At operation, this wedge is stripped of its fascia, thus allowing dislocation of the upper pole to simplify ligation of the superior thyroid vessels. The thyroid gland is enclosed in an envelope of fascia derived from the deep cervical fascia and known as the surgical capsule. It has in addition an internal or true capsule intimately connected with and ensheathing the gland. Surgical dissection of the gland takes place between these two layers. The *pyramidal lobe*, when present, is a finger-like projection of thyroid tissue extending from the upper border of the isthmus, often as high as the hyoid bone. The thyroid gland is intimately connected with the larynx, and therefore moves upward on swallowing. This movement is important when diagnosing swellings of the thyroid. The *arterial supply* of the thyroid gland is richer than that of any other organ in the body. It is derived mainly from two sources : the *superior thyroid artery* which arises from the external carotid and runs downward to reach the upper pole, and the *inferior thyroid artery* which arises from the thyrocervical trunk and runs behind the carotid sheath to reach the posterolateral aspect of the lobe. If both these arteries were ligated, a collateral circulation would develop from the internal and external laryngeal, cricothyroid, lingual, tracheal and œsophageal arteries. A *thyroidea ima artery* may occasionally be present arising from the innominate or subclavian arteries ; it ascends to the lower border of the isthmus.

The *recurrent laryngeal nerves* run upwards, lying in or nearby the tracheo-œsophageal groove. Each nerve usually runs behind the inferior



thyroid artery, and very often divides into several branches before entering the larynx. Injury of these nerves is discussed under *The Cranial Nerves*, Chap. 52.

### METHOD OF EXAMINATION

The patient should be sitting during examination of the neck, as the thyroid gland is difficult to palpate with the patient recumbent. It is essential to flex the head on the neck to enable the exploring fingers to pass under the relaxed sternomastoid muscles to gain a clear impression of the size, consistency and outline of the goitre. A normal thyroid gland is rarely palpable, except in a very thin neck. By use of the above method, the presence of deeply placed thyroid swellings can be demonstrated; and the upper margin of a hidden retrosternal mass may be palpated as it rises when the patient swallows. It is sometimes necessary to give the patient a sip of water to be held in the mouth and then swallowed when the examining fingers are in position. If displacement of the trachea has been demonstrated, the patient is asked to breathe deeply through the partly open mouth. Stridor may then be elicited suggesting either pressure on the trachea or laryngeal palsy. A brassy or tracheal cough denotes the same thing. It is necessary to decide by inspection and palpation whether the goitre is diffuse, nodular or presenting as a discrete nodule (adenoma). Its consistency is noted. Any tenderness, displacement of the trachea or the presence of enlarged lymph nodes is recorded. A bruit may be heard on auscultation. A careful history is taken and a general examination made to decide whether there is hyperthyroidism or hypothyroidism.

**Investigations.** *Radiography.*—This reveals any displacement or compression of the trachea, and any calcification or retrosternal extension of the goitre. *Estimation of the Basal Metabolic Rate (B.M.R.).*—If expertly carried out, this is of value in diagnosis and as an indication of the effect of treatment. The rate of oxygen uptake is expressed as a percentage of the normal, allowing for age, sex, weight and height. The percentage is increased in hyperthyroidism. *The Blood Cholesterol.*—The normal varies from 150 to 300 mg. per 100 ml. The level is raised in hypothyroidism and lowered in hyperthyroidism. *Radioactive Iodine Uptake.*—The avidity of the thyroid gland for iodine, and the rate of production of thyroid hormone are increased in thyrotoxicosis and decreased in hypothyroidism. After oral administration of a tracer dose of 50 microcuries (in adults) of radioactive iodine  $I^{131}$ , the activity of the gland can be studied in a number of ways. Thus by scanning the thyroid area with a Geiger-Mueller counter four hours after administration of the radioactive isotope,\* its uptake by the gland may be expressed as a percentage of the total tracer dose. The four-hour uptake is usually over 40 per cent. in

\* Isotope = any one of a series of chemical substances of identical chemical properties, but differing in atomic weight.

hyperthyroidism and below 10 per cent. in hypothyroidism. The amount of  $I^{131}$  excreted in the urine within forty-eight hours of the tracer dose is inversely proportional to the activity of the thyroid gland. *Serum Protein-bound Iodine.*—As the thyroid hormone is carried in the blood in combination with protein, estimation of the serum protein-bound iodine gives a measure of the amount of thyroid hormone in the circulation and therefore of the activity of the thyroid gland. The normal range is 4 to 8 mg. per 100 ml. of serum. The amount is increased in hyperthyroidism and decreased in hypothyroidism.

### NON-TOXIC GOITRES

Non-toxic goitres may be either endemic or sporadic. They result in the main from an actual or relative iodine deficiency—there being an actual lack of iodine in the water or soil (causing endemic goitre) or an inability of the thyroid gland to utilise and synthesise it into the thyroid hormone (causing sporadic goitre). Nodular changes, commonly seen in this class of goitre, are brought about by its response to the demands of the body for thyroid hormone at puberty, pregnancy and other periods of stress.

**Non-toxic Diffuse Goitre.** The goitre is soft and diffusely enlarged, and may first appear at puberty (puberal goitre). It occasionally, because of its size, gives rise to pressure symptoms. It may appear during pregnancy or after childbirth, and later in life nodules may develop. It is unusual for it to become frankly toxic or give rise to symptoms. In endemic goitre regions, the prophylactic use of approximately 1 part of potassium iodide to 200,000 parts of table salt has reduced the incidence of this type of goitre. Treatment of diffuse non-toxic goitre with thyroid extract (30 to 120 mg. daily), particularly when there is evidence of hypothyroidism, is also often satisfactory, but must be continued long after the goitre has regressed. The purpose of this treatment is to rest the thyroid gland by inhibiting the thyrotrophic hormone. Subtotal thyroidectomy is indicated if the goitre gives rise to pressure effects or becomes nodular. Sometimes the operation is done for cosmetic reasons.

**Non-toxic Nodular Goitre (Adenomatous Goitre).** Nodular enlargement of the thyroid gland is usually easily recognised by inspection and palpation. The adenomatous or nodular changes are more common after the age of forty years, but may also occur in children and young adults. The nodules vary in consistency according to whether they consist of cystic spaces filled with colloid or solid masses of hyperplastic epithelial tissue; occasionally the goitre becomes calcareous. It is possible for only one lobe to be involved. Iodine deficiency and long-continued demands upon the thyroid gland result in the formation of these areas of hyperplasia and involution.

The symptoms arising from non-toxic nodular goitres are chiefly due to pressure. They range from a feeling of tension in the throat, increased by

fatigue or emotion, to cough, dyspnoea, hoarseness, stridor and dysphagia. The nodules, as they increase in size, may become retroclavicular or retrosternal; it is in these positions that they produce pressure manifestations which are due to impaction and obstruction at the thoracic inlet. Large veins may be seen coursing over the chest wall and in the neck (Fig. 464).

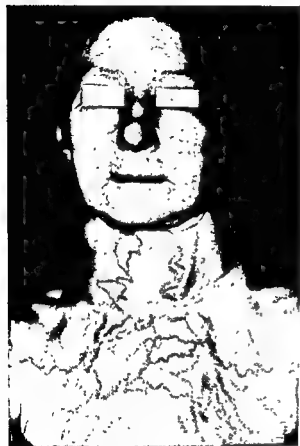


FIG. 464. Engorged veins due to impaction of a goitre at the thoracic inlet.

These veins become more distended if the arms are raised above the head. *Hæmorrhage* may take place within a nodule causing it suddenly to enlarge and become painful. The discomfort subsides after a few days and the nodule slowly regains its original size. This occasionally results in spontaneous cure. If pressure is already present, bleeding makes it worse. Multinodular goitres may be present for twenty or more years without giving rise to symptoms, but it is likely that the majority of them ultimately become toxic. Symptoms are few and may only consist of undue fatigue, dyspnoea on exertion, palpitations, irritability, and loss of weight. There may also be tremor of the fingers and dampness of the palms. This long-continued mild thyrotoxicosis affects the cardiovascular system and may

give rise to paroxysmal auricular fibrillation and lead to congestive heart failure. Occasionally the apparently non-toxic nodular goitre becomes frankly toxic. Nodular goitres in children and young adults are to be looked upon with grave suspicion, for they may prove to be malignant or at any rate are liable to recur after operation.

**Treatment.** Non-toxic nodular goitres should be treated surgically by selective partial thyroidectomy (i.e. removal of all nodules, retaining only the apparently normal tissue). Operation is recommended in the symptomless non-toxic nodular goitre as an insurance against:—(1) Pressure from its gradual increase in size; (2) hæmorrhage and consequent sudden pressure; (3) thyrotoxicosis; and (4) carcinoma.

**Retrosternal Goitre.** The common type of retrosternal goitre is the retrosternal prolongation of a nodular goitre, or a discrete adenoma arising

from the lower pole of a thyroid lobe (cf. true intrathoracic goitre). The retrosternal mass may ultimately reach enormous size, but may be present for many years without giving rise to signs of pressure. Furthermore, the goitre may not be discernible in the neck, but may cause auricular fibrillation. Such a goitre may not be discovered until the chest is X-rayed. The upper limit of the tumour, lying deeply behind the sternum, may be palpable only when the patient swallows. *Early symptoms of pressure* consist of a sensation of fullness in the neck, slight



FIG. 465. X-ray showing a retrosternal goitre and tracheal displacement.

stridor on exertion and huskiness of the voice. The trachea is usually displaced laterally. *Severe symptoms of pressure* gradually develop as the goitre increases in size. Stridulant breathing becomes apparent even at rest; the cough becomes harsh and brassy, and the voice harsh and indistinct. Cyanosis of the face is noticeable, particularly on exertion. *Sudden pressure* may occur as the result of hæmorrhage into a known or hitherto unsuspected retrosternal goitre. Pain is a constant feature accompanying this complication. The *X-ray picture* of a retrosternal goitre may give an erroneous idea of its size and depth (Fig. 465) and give the impression that removal would be a formidable undertaking, but the reverse is often the case, for it is rarely necessary to split the sternum to deliver even very large retrosternal goitres.

The *true intrathoracic* or aberrant goitre, which originates in the mediastinum, is rare. It can only be removed after first splitting the sternum, for it is often both deeply placed and adherent, and its blood supply is derived from vessels arising in the mediastinum. If there is any doubt that an X-ray shadow in the mediastinum is of thyroid origin, a valuable investigation is to give a tracer dose of radioactive iodine and scan the chest with a Geiger-Mueller counter.

### TOXIC GOITRES

Thyrotoxicosis is the result of over-activity of the thyroid gland producing an excess of its hormone. Almost all the clinical signs and symptoms of thyrotoxicosis, whether the associated goitre is primary diffuse or secondary nodular, are due to this over-production of the normal thyroid hormone. The thyroid is stimulated to increased activity by the thyrotropic hormone from the pituitary gland, and becomes enlarged and hyperplastic. The *primary diffuse toxic goitre* (primary thyrotoxicosis) arises in a previously normal thyroid gland, whereas the *secondary thyrotoxic nodular goitre* (secondary thyrotoxicosis) arises in a gland previously the site of a simple nodular goitre which

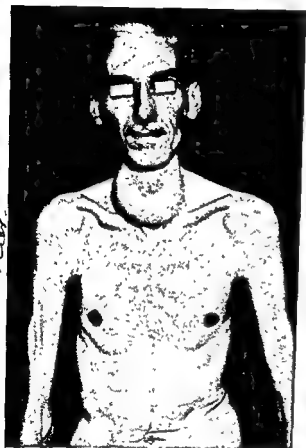


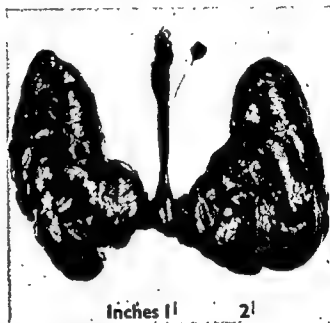
FIG. 466. A patient with a diffuse thyrotoxic goitre showing wasting of muscles.

may have been present for many years before the onset of toxic manifestations.

**Primary Toxic Diffuse Goitre.** This is far more common in women than in men, and may begin to show itself at any age between puberty and the menopause, although it is not unknown in childhood and old age. There may be a constitutional background or predisposition toward primary thyrotoxicosis. It can be precipitated by emotional disturbance, although it is unlikely that a single upset, fright or accident can initiate thyrotoxicosis. The *goitre* (Figs. 466 and 467) is diffuse or lobulated, firm and often pulsating. Its presence is usually noticed by the patient soon after symptoms begin. The increase in circulating thyroid hormone commonly affects the metabolism, the nervous system and the cardiovascular

system, resulting in the classical signs and symptoms of thyrotoxicosis. A voracious appetite, loss of weight, hot skin, moist palms and dyspnoea are an expression of the metabolic disturbance. Nervousness, depression, anxiety and fine tremor of the fingers are due to effects on the nervous system. Palpitations, tachycardia, auricular fibrillation and, in severe cases, congestive heart failure are caused by toxic effects on the myocardium. Thyrotoxicosis may be graded into mild, moderate and severe; the first category may be difficult to diagnose, often being confused with anxiety states. The goitre may be prominent or small; very occasionally it is impalpable. Eye signs are commonly present in thyrotoxicosis.

FIG. 467. A diffuse toxic goitre showing a pyramidal lobe and the attached "delphian" lymph node.



Those produced by thyroid hypersecretion are:—(a) Widening of the palpebral fissure or stare, caused by retraction of the upper lid. This is often confused with exophthalmos (protrusion of the eyeballs). (b) von Graefe's sign, the lagging of the upper lids as the eyeballs rotate downwards. These signs disappear when the thyrotoxicosis has been controlled or eliminated. Exophthalmos is *not* caused by thyroid hypersecretion although it is present in about half the cases of primary thyrotoxicosis (Fig. 468). The protrusion of the eyes is most likely produced or initiated by some fraction of the thyrotropic hormone of the anterior pituitary. There is an excess of retro-orbital fat and lymphocytic infiltration of this and the œdematous ocular muscles. The more rapid the increase of retro-orbital pressure, the greater is the danger to the eyes from œdema of the conjunctivæ and corneal ulceration. The rapidly progressive and severe type is called *malignant exophthalmos*. When severe and progressive, it is treated by retro-orbital deep X-ray therapy (with or without

tarsorrhaphy), or occasionally by orbital decompression (see Naffziger's operation, Chap. 52). It is advisable to allow these patients to remain slightly toxic and so to lessen the thyrotropic activity of the pituitary gland. Partial thyroidectomy is contraindicated during the acute phase when the exophthalmos is severe or progressive. It must be withheld until the patient's toxicity has been partly controlled for several months by other means without exophthalmos having become worse.

**Secondary Toxic Nodular Goitre.** Unlike the primary toxic diffuse goitre, symptoms appear in patients who have had a pre-existing goitre for some years, and, although the metabolism and nervous systems are involved to some extent, the main stress is on the cardiovascular system. The toxic process is slow, and usually not severe. Paroxysmal auricular fibrillation is often present in the later stages, and may ultimately develop into established fibrillation, resulting in congestive failure. Cardiac decompensation most commonly appears after the age of fifty. These goitres are firm with an irregular nodularity. The nodules vary in size and consistency, but are usually firmer than the surrounding thyroid tissue. If calcified, the nodules are stony hard. Occasionally only one lobe is involved, and its continued growth may displace the trachea. *Hæmorrhage* may take place into a nodule giving rise to sudden discomfort, increase in size, and often tracheal compression. The *secondary toxic nodular goitre* (Fig. 469) is a common cause of auricular fibrillation. The thyrotoxic origin of the cardiac irregularity is often not appreciated unless a nodular goitre is seen or palpated in the neck. It is most important that all patients with auricular fibrillation should undergo a thorough examination of the relaxed neck in an attempt to palpate the upper margin of a deeply placed or a hidden substernal goitre, for a large number of patients with nodular goitres and auricular fibrillation regain normal cardiac rhythm after subtotal thyroidectomy. Eye changes may be associated with the toxic nodular goitre, but to a lesser extent than in the primary toxic diffuse type.



FIG. 468. Exophthalmos, lid retraction, widening of palpebral fissures and ophthalmoplegia. (Dr. Raymond Greene's patient).

**Thyroid Adenoma.** There are two types of adenoma:—(a) *True benign epithelial neoplasm*, completely encapsulated from the surrounding

goitre, symptoms appear in patients who have had a pre-existing goitre for some years, and, although the metabolism and nervous systems are involved to some extent, the main stress is on the cardiovascular system. The toxic process is slow, and usually not severe. Paroxysmal auricular fibrillation is often present in the later stages, and may ultimately develop into established fibrillation, resulting in congestive failure. Cardiac decompensation most commonly appears after the age of fifty. These goitres are firm with an irregular nodularity. The nodules vary in size and consistency, but are usually firmer than the surrounding thyroid tissue. If calcified, the nodules are stony hard. Occasionally only one lobe is involved, and its continued growth may displace the trachea. *Hæmorrhage* may take place into a nodule giving rise to sudden discomfort, increase in size, and often tracheal compression. The *secondary toxic nodular goitre* (Fig. 469) is a common cause of auricular fibrillation. The thyrotoxic origin of the cardiac irregularity is often not appreciated unless a nodular goitre is seen or palpated in the neck. It is most important that all patients with auricular fibrillation should undergo a thorough examination of the relaxed neck in an attempt to palpate the upper margin of a deeply placed or a hidden substernal goitre, for a large number of patients with nodular goitres and auricular fibrillation regain normal cardiac rhythm after subtotal thyroidectomy. Eye changes may be associated with the toxic nodular goitre, but to a lesser extent than in the primary toxic diffuse type.

thyroid tissue. (b) *Involucional nodule*, the end result of alternating hyperplasia and involution. The adenoma may be found in patients of any age, but rarely before puberty. It may be *toxic* or *non-toxic*. In the former case, the patient usually presents with symptoms of minimal thyrotoxicosis, whereas in the latter the complaint is only of a lump in the neck. On examination, a smooth, rounded swelling rising on deglutition is observed or palpated in the neck. If an adenoma is large, the trachea is usually displaced to the opposite side. It has been generally recognised that all discrete adenomas, toxic or non-toxic, should be removed for the following reasons:—(1) *Malignancy* occurs in over 10 per cent. of non-toxic adenomas. (2) *Toxicity* develops in the majority, and may give rise ultimately to auricular fibrillation. (3) The *continued growth* of an adenoma eventually causes pressure symptoms. (4) *Hæmorrhage* may occur into the substance of an adenoma, causing sudden enlargement, and consequent pain and pressure effects.

*Radioactive iodine uptake* has recently shown us that the functionally active or "hot" nodule can be distinguished from the functionally non-active or "cold" nodule, and as the "cold" nodule has a high inci-

dence of carcinoma it must be completely removed. The "hot" nodule, on the other hand, in a euthyroid or subthyroid person may be a response of the thyroid to maintain an adequate amount of thyroid hormone in the body. This being so, according to Raymond Greene, the nodule will on occasion soften and decrease in size if the patient is given a course of thyroid by mouth.



FIG. 469. A patient with a toxic nodular goitre. The goitre had been present for over twenty years when auricular fibrillation developed.

### THYROIDITIS

**Acute Thyroiditis.** Acute inflammation of the thyroid is very rare. It may follow the specific fevers, and a goitrous gland is more susceptible than the normal thyroid. The onset is sudden and gives rise to severe discomfort in the neck, pain on swallowing, hoarseness and pyrexia.



There is a generalised tender swelling of the thyroid gland. Despite chemotherapy, an abscess may occur in either lobe. Fluctuation can usually be elicited. The abscess should be drained.

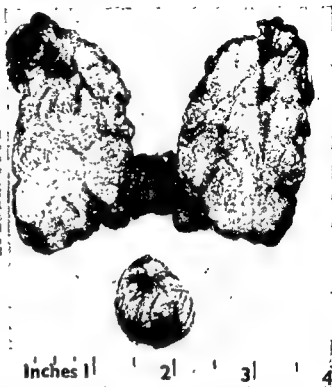
**Subacute Non-suppurative Thyroiditis.** This disease may present with pain, tenderness and pyrexia; the thyroid may be enlarged to three or four times its normal size. The thyroid is firm and a little tender on palpation. Alternatively, the condition may present itself in a more chronic form; the thyroid is then tender, but there is no associated constitutional reaction. There is commonly a history of recent sore throat, stiffness of the neck, and pain on swallowing. Occasionally signs of inflammation follow hæmorrhage into a nodular goitre or an adenoma. The tender swelling is then confined to one lobe and may give rise to pressure symptoms. The inflammation and swelling usually resolve spontaneously. In many clinics needle biopsy (with a Silverman's needle) is carried out when the diagnosis is in doubt. Further confirmatory evidence is obtained by the radioactive iodine test which reveals diminished activity of the inflamed thyroid gland. *Treatment* includes administration of antibiotics, cortisone and thyroid extract. After resolution has taken place the function of the thyroid usually returns to normal.

**Chronic Thyroiditis.** Chronic thyroiditis is represented by *lymphadenoid goitre* (Hashimoto's thyroiditis) and *Riedel's thyroiditis*. It is unlikely that, as has been suggested, these two types of goitre are due to the same cause or that one is a later stage of development of the other. Many causes have been suggested, but none of them has been proven. *Lymphadenoid goitre* is most often seen in women. It is commonest after forty, but may occur at any age after puberty. In its early stages, lymphadenoid goitre may closely simulate nodular toxic or non-toxic goitre. The lymphadenoid condition must be considered when on palpation the nodules or lobules are found to be unusually smooth and firm. As the condition progresses, the entire gland becomes involved in the lymphadenoid infiltration; the lobes become hard, smooth, discrete and ivory in colour (Fig. 470). *Hypothyroidism* develops. Mild pressure symptoms may occur in the later stages, and consist of a sensation of constriction in the neck, dyspnoea and hoarseness. As the lymphadenoid infiltration gradually replaces all, or nearly all, the secreting thyroid tissue, thyroid extract must be administered indefinitely to control the consequent hypothyroidism. A conservative partial thyroidectomy is necessary when manifestations of pressure develop.

**Riedel's Thyroiditis.** Fibrosis is the main characteristic of this disease. It not only involves the gland, but spreads beyond its capsule to the surrounding fascia and muscles. Occasionally the fibrosis is confined to one lobe. The gland becomes very hard and fixed, though only moderately enlarged. *Fixation and hardness* are the main diagnostic features. In the late stages of the disease hypothyroidism develops. Compression of the trachea, causing hoarseness, dyspnoea, stridor and occasionally

dysphagia, commonly occurs. Riedel's thyroiditis, although closely simulating carcinoma of the thyroid in regard to consistency and fixation, is unlike carcinoma in that the gland retains its anatomical outline allowing recognition of the complete outline of the thyroid. Furthermore,

FIG. 470. Lymphadenoid goitre (Hashimoto). The lymphadenoid process has replaced nearly all of the secreting thyroid tissue. The enlarged lymph gland shown below the thyroid specimen was found just above the suprasternal notch. Surrounding lymph glands tend to be enlarged in this disease.



thyroiditis usually has a longer history than carcinoma. Operation is indicated when symptoms of pressure arise. The isthmus of the thyroid is then removed to relieve the constriction of the trachea.

### CARCINOMA OF THE THYROID

The incidence is about 5 per cent. in non-toxic nodular goitres, whereas it is as much as 10 per cent. in non-toxic discrete adenomas. It occasionally develops in toxic nodular goitres, but is very rare indeed in the primary diffuse toxic type. Pathologists find some types of carcinoma of the thyroid difficult to diagnose under the microscope and many of them hold divergent views regarding the criteria of malignancy. Some tumours resemble innocent adenomas so closely that they are known as benign metastasising adenomas; they invade blood vessels giving rise to blood-borne functioning secondary deposits.

The more papillary tissue found in the section the slower growing and less malignant is the tumour—the true papillary type having the best

prognosis. Also, the more differentiated the cells of the tumour the less malignant it is—the undifferentiated types being very malignant.

Neoplastic changes may develop in the thyroid gland at any age, although the more malignant and clinically evident tumours are commoner in the later decades.

**Classification.** (1) *Adenocarcinoma* (alveolar adenocarcinoma). The majority of malignant tumours of the thyroid belong to this group. The malignant changes arise in a nodule of a multinodular goitre or more often in a non-toxic discrete adenoma. They vary from the well-differentiated carcinoma of low-grade malignancy to that of the partially undifferentiated type of moderate-grade malignancy. An adenocarcinoma may sometimes arise *de novo* in otherwise normal thyroid tissue, and is then not encapsulated. The spread of this type of growth tends to be more rapid, but depends mainly on the degree of differentiation. Metastasis from adenocarcinomas is mainly blood borne. (2) *Papillary carcinoma* and *papillary adenocarcinoma* are described as such because of the preponderance of papillary tissue. The true papillary carcinoma is the least malignant of all thyroid carcinomas. These slowly growing neoplasms give rise to chains of large, soft secondary nodes (lateral aberrant thyroid nodes) often as large as a thyroid lobe although the primary growth may be too small to be detected by the naked eye. The *papillary cystadenoma* (papillary adenocarcinoma) is not very malignant, but the more undifferentiated the tissue, the more malignant it is. (3) *Undifferentiated carcinoma* includes all other carcinomas. They are further classified as: simplex; scirrhous small, large and spheroidal celled; pleomorphic giant-celled (Fig. 471); and carcinomas displaying sarcomatous tendencies. The common factor in all these rapidly growing malignant tumours is that they are undifferentiated (anaplastic). It is often difficult to recognise microscopically that they originate from the thyroid gland. They are found in the upper age groups. They spread rapidly by direct invasion by the blood stream, and by the lymphatics. The prognosis is poor. Occasionally the spread may be so rapid that a sarcoma is suspected and indeed it may be difficult to distinguish microscopically an anaplastic carcinoma from a sarcoma. There are, therefore, some tumours, so undifferentiated, that their exact nature cannot be determined. *Reticulo-lymphosarcoma* is included in the undifferentiated group of malignant tumours. It grows very rapidly and metastasises both by the blood stream and by the lymphatics. It responds, but only temporarily, to deep X-ray therapy.

**Clinical Groups.** (1) *Clinically Obvious Carcinoma*.—The growth is advanced having broken through the true capsule of the thyroid and invaded surrounding structures. The mass is hard, fixed, nodular and tender. Firm lymph nodes are often palpable in the neck and radiating pain may be present. Hoarseness due to tracheal pressure or infiltration or involvement of a recurrent laryngeal nerve often occurs. The cancer

is in an advanced stage when as obvious as this, and is likely to be undifferentiated and to be found in the upper age group. The prognosis is very bad and the patient usually dies within a year, in spite of deep X-ray therapy or thyroidectomy. (2) *Clinically Suspected Carcinoma.*—Malignancy is suggested when a nodular goitre or a discrete adenoma



FIG. 471. Patient with pleomorphic giant-celled carcinoma of the thyroid. The goitre had been present for over forty years with four months' history of rapid increase in size and pressure symptoms (fatal termination).

which, having been stationary for many years, suddenly begins to increase in size. The patient complains of discomfort in the goitre often with a sense of constriction in the neck. On examination, the nodular mass is found to be tender and firm. The prognosis is very much better in the suspected case, for it is likely that the carcinoma is still contained within the capsule of the adenoma and can therefore be satisfactorily removed. Such a growth is most often differentiated in type, and is commonly seen in the middle age group. It must not be confused with the sudden tenderness and increase in size caused by hæmorrhage into an adenoma.

(3) *Clinically Hidden Carcinoma.*—This type of growth is first diagnosed either during partial thyroidectomy for a nodular goitre or on routine post-operative microscopy. The prognosis is excellent if the growth has not already invaded the blood vessels and given rise to secondary deposits. It is important, therefore, that all discrete non-toxic adenomas and all nodular goitres should be removed, particularly if they have recently become tender and have increased in size.

**Treatment of Carcinoma of the Thyroid.** *Thyroidectomy* is the treatment of choice, particularly in the early stages of the disease. The extent of the operation depends on the stage and extent of the disease and the

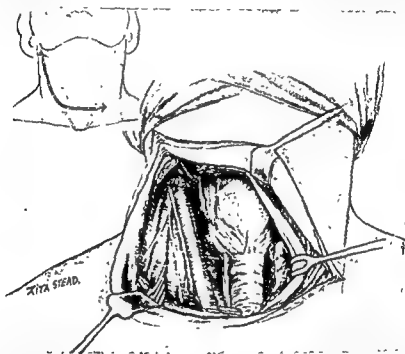


FIG. 472. Unilateral block dissection for advanced carcinoma of one lobe. The left lobe remains *in situ*. In practice, this lobe should also be removed to allow for post-operative radioactive iodine therapy if found necessary. (*Techniques in British Surgery.*)

type of tumour:—(a) *Carcinoma Discovered at Operation.*—If a carcinoma is recognised macroscopically at operation performed for an “adenoma” or a “nodular goitre,” a complete removal of the involved lobe (lobectomy) should be undertaken whether the growth is still confined within the capsule of the nodule or has spread beyond it. The other lobe is explored and most of it is removed. (b) *Unsuspected Malignancy.*—When a carcinoma is diagnosed initially by post-operative microscopy, observation of the patient is usually sufficient unless the growth has spread beyond the capsule of the adenoma when either deep X-rays or a further more extensive operation is indicated. (c) *Clinically Obvious Carcinoma.*—(1) The growth has spread beyond its capsule; (2) has invaded lymph glands in the neck; or (3) has invaded surrounding fascia and muscles.

A complete lobectomy on the involved side is undertaken together with the prethyroid muscles and any involved glands. The opposite lobe is also removed. A *unilateral block dissection* of the neck is undertaken, if considered necessary, and includes removal of the internal jugular vein, the sternonastoid muscle and all fascia and regional lymph nodes (Fig. 472). Deep X-ray therapy or radioactive iodine therapy is given if it is considered that further malignant tissue remains in the neck. The choice of the post-operative treatment depends on whether the cancer is of a type sensitive to deep X-ray or whether it can take up radioactive iodine. To allow radioactive iodine to concentrate in local or distant metastases, total thyroidectomy is performed. Concentration of the iodine could not occur in the secondary deposits if thyroid tissue still remained in the neck. *Deep X-ray therapy* is indicated :—(1) After thyroidectomy, for the treatment of any residual carcinoma in those types of tumour sensitive to deep X-rays. (2) Post-operatively to lessen the possibility of a local recurrence. (3) For late and inoperable carcinoma or sarcoma. Sometimes the rapid and progressive spread of a neoplasm may temporarily respond to deep X-ray therapy, and occasionally the tumour then becomes amenable to surgery.

### RADIOACTIVE IODINE

Radioactive iodine has proved an invaluable aid in the study, diagnosis and treatment of diseases of the thyroid gland. Therefore every thyroid clinic should have a well-equipped isotope laboratory. *Precautions.*—Patients who have had therapeutic doses of radioactive iodine must be isolated and nursed in separate cubicles or rooms for several days until the radioactivity of their excreta has diminished to a safe level. The education of the nursing staff in the safe handling of radioactive patients and their excreta is one of the fundamental necessities of the thyroid clinic. The nurses wear rubber gloves when handling such patients and their excreta which are transported to the ageing room of the laboratory and there stored until the activity is sufficiently low for disposal. The thyroid is the only gland in the body which stores and utilises iodine. It follows, therefore, that if iodine is given by mouth, it is carried by the blood and stored in the thyroid gland. The action of the radioactive isotope in therapeutic amounts is similar to that of deep X-ray therapy or radium in that it destroys or diminishes the activity of the tumour cells it comes into contact with.

**Use in Diagnosis.** Radioactive iodine is used in very small doses (10 to 100 microcuries) as an aid to diagnosis. In thyrotoxicosis, the more avid the thyroid gland is for iodine, the more thyrotoxic is the patient. The lower the uptake of iodine below normal, the more subthyroid is the patient. From the known amount of the radioactive isotope, and the measured amount collected from the urine in forty-eight hours, the uptake in the thyroid can be estimated and its level of activity assessed. The

quantitative estimation of protein bound radioactive iodine in the blood is also used as a diagnostic measure. In thyrotoxicosis, all circulating radioactive iodine is protein bound within forty-eight hours, whereas it is only very slowly bound in subthyroid conditions.

**Use in Treatment.** A. *Thyrotoxicosis*.—The primary toxic diffuse goitre is more satisfactorily treated than is the secondary toxic nodular goitre, for the uptake of the radioactive isotope is more uniform and predictable in the former. The patient receives the amount of radioactive iodine which gives an emanation of 5 to 15 millicuries. Although not usually recommended for thyrotoxic patients under forty-five years, as it has not been proved that the radioactive isotope will not in time cause cancer of the thyroid, it is nevertheless given for the following conditions irrespective of the age of the patient :—(1) A recurrent thyrotoxic goitre which is small, adherent and difficult to remove ; (2) recurrent thyrotoxicosis associated with recurrent laryngeal nerve palsy ; (3) thyrotoxicosis resulting in severe myocardial damage and congestive heart failure with a low cardiac reserve ; (4) thyrotoxicosis associated with other serious organic or mental diseases ; (5) thyrotoxicosis with severe and progressive exophthalmos ; (6) severe thyrotoxicosis which cannot be controlled or prepared for operation by means of the antithyroid drugs thiouracil or Neomercazole (carbimazole). Treatment by adequate radioactive iodine gives excellent results. The maximum benefit from the treatment is usually apparent within three months, although it may not be observed for five or six months. As the thyrotoxicosis disappears, the goitre usually decreases in size. Inadequate treatment leaves the patient still toxic whereas overdosage gives rise to myxœdema. B. *Carcinoma of the Thyroid*.—It is not possible satisfactorily to treat primary carcinoma of the thyroid or its secondary deposits by radioactive iodine, for the isotope is bound to concentrate first in the normal thyroid tissue. It is necessary, therefore, first to remove this target by means of total or near total thyroidectomy. This allows a sufficient uptake of the isotope into any remaining malignant tissue. Under such circumstances radioactive iodine is of value in the treatment of a local tumour or its secondary deposits if they concentrate, or can be induced to concentrate, the isotope. About half the malignant thyroid tumours have some uptake, but only 15 to 20 per cent. have sufficient uptake to allow satisfactory post-operative treatment of secondary deposits or malignant remnants. The majority of papillary and undifferentiated carcinomas do not have an uptake of the isotope, but it may be induced in their secondary deposits after removal of the thyroid and the production of myxœdema. Before treatment by radioactive iodine, a small dose of the isotope is given and traced by means of the Geiger-Mueller counter to determine the extent, if any, of radioactive iodine uptake, and a further small dose is given some months after treatment to ascertain whether or not activity is still present and further radioactive therapy is needed.

## PREPARATION FOR SUBTOTAL THYROIDECTOMY

All patients with *severe* thyrotoxicosis, whether the associated goitre is primary diffuse or secondary nodular, are prepared for subtotal thyroidectomy by administration of *potassium iodide* and the *antithyroid drugs*, *methyl thiouracil* (0.2 gm. twice daily) or *Neomercazole* (10 mg. three times daily), whereas patients with the *moderate* degree of thyrotoxicosis commonly accompanying both the secondary nodular goitre and the single adenoma are prepared with iodine alone (0.3 gm. of potassium iodide daily). The antithyroid drugs act directly on the thyroid gland inhibiting the synthesis of the thyroid hormone. This results in lowering of the basal metabolic rate and in alleviation of the thyrotoxicosis. The typical hyperplasia and vascularity of the thyrotoxic gland, however, remains as before and may even increase. Overdosage of the antithyroid drugs gives rise to myxœdema; operation must not be undertaken until the basal metabolic rate regains its normal level, for in myxœdema there is intolerance to morphine, and thickening of the vocal cords may result in laryngeal obstruction. *Iodine* administered to a thyrotoxic patient produces a rapid improvement, and the gland becomes firmer and less vascular; the benefit from administration of iodine is, however, only temporary; it reaches its maximum at or about the third week when operation should therefore be undertaken. Subtotal thyroidectomy must never be performed on a patient prepared by one of the antithyroid drugs alone. These drugs, by increasing the hyperplasia, vascularity and friability, particularly of the diffuse goitre, greatly increase the technical difficulties of the operation. The drug is replaced by potassium iodide for the final two or three weeks' preparation; by this method, the goitre is brought to a stage of involution and the difficulties resulting from administration of the antithyroid drugs are largely overcome. All severely thyrotoxic patients, therefore, are brought to a comparatively non-toxic state by the use of the antithyroid drugs, and thus the many dangers previously associated with operation have been eliminated. Improvement after medical treatment is shown by gain in weight, lowering of the pulse rate and lessening of sweating and nervous symptoms.

**Toxic Manifestations of the Antithyroid Drugs.** The antithyroid drugs can give rise to several toxic manifestations. These include pyrexia, headache, rashes, vomiting and diarrhoea, joint pains, swollen lymph nodes, jaundice, leucopenia, and agranulocytosis. If agranulocytosis is suspected, the drug is stopped immediately; and if proven by a blood count the patient is admitted to hospital and given massive doses of penicillin and blood transfusions. *Neomercazole* may be less toxic than the thiouracils and gives rise to fewer side effects. Furthermore, the goitre is rendered less vascular than with the thiouracils.

**In-patient Preoperative Care.** Those thyrotoxic patients who have been prepared as out-patients with methyl thiouracil or Neomercazole and



potassium iodide, and admitted to the hospital in a sufficiently non-toxic state, undergo operation within forty-eight hours of admission. Those thyrotoxic patients who are admitted with auricular fibrillation and signs of congestive heart failure are kept at complete rest in bed. If the heart rate, as counted at the apex, exceeds 160 per minute, sodium iodide, 0.5 gm. in 1 litre of isotonic saline, is given intravenously. Salyrgan (or a similar drug) is also sometimes necessary. *Quinidine* is never given before operation. Subtotal thyroidectomy is withheld until the physician is satisfied that the congestive failure has been reduced to a minimum. X-rays of the chest are necessary in patients with nodular goitres to demonstrate any possible retrosternal prolongation. A *laryngoscopic examination* of the vocal cords is usually done to detect any involvement of the recurrent laryngeal nerves, and always before operating on a regrowth of a goitre.

On the day previous to operation, a fluid diet is given, and a small enema or a glycerine suppository is ordered if necessary. The site of operation is prepared. Sodium barbitone (medinal) 0.3 to 0.6 gm. is given at night. On the morning of operation, a further skin preparation is carried out, and Omnopon 20 mg. ( $\frac{1}{2}$  gr.) and scopolamine 0.4 mg. ( $\frac{1}{40}$  gr.) are injected one hour before operation. A further injection of half the above doses of Omnopon and scopolamine is given half an hour before operation. Elderly patients, with evidence of cardiovascular degeneration, are given only one injection of Omnopon and scopolamine.

*Anæsthesia* is induced by a slow intravenous injection of 0.4 to 0.6 gm. of pentothal and is continued by means of nitrous oxide with the high percentage of oxygen necessary for all thyrotoxic patients. *Endotracheal anæsthesia* is reserved specially for those patients with hard and fixed goitres (including carcinoma of the thyroid, lymphadenoid and Riedel's thyroiditis, retrosternal goitre, and those goitres with substernal or retroclavicular prolongations), and also for those patients in whom there is tracheal compression.

### THE OPERATION

The adjustable bridge of the operating table is raised under the patient's shoulders until the neck is extended and prominent. The surgeon, using a continuous action syringe with a 3-inch needle attached, injects the subcutaneous tissue of the neck for an area extending from the thyroid notch of the larynx above, to the suprasternal notch below, and laterally as far as the external jugular veins. The solution used is 1/200,000 adrenaline in normal saline. This technique lessens capillary oozing and also facilitates raising the flaps by clearly defining the fascial planes.

The incision is transverse and slightly convex downwards. Its level is approximately  $1\frac{1}{2}$  inches above the suprasternal notch and usually extends from one external jugular vein to the other. It is continued through the superficial fascia and the platysma muscle. Care is taken not to go beyond

this plane as the anterior jugular veins may be injured. Reflection of the upper flap is then begun by nicking the areolar tissue deep to the platysma whilst the upper flap is being rolled upwards. The upper flap should be raised in the average case to the level of the thyroid notch of the larynx. In patients with very large goitres or high upward prolongations, the flap is raised to the level of the hyoid bone. The lower flap is then similarly raised.

A midline sagittal incision is made through the fascia between the

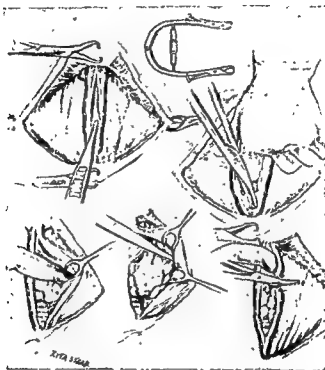


FIG. 473. Separation, retraction and section of the "strap" muscles. (Note Joll's self-retaining skin-flap retractors. (*Techniques in British Surgery.*)

infrahyoid muscles (Fig. 473) and enlarged with scissors upwards to the thyroid notch, and downwards to the suprasternal notch. The thyroid isthmus is now visible, covered by its deep fascia (surgical capsule). This capsule is opened with blunt dissecting forceps and the forefinger of the left hand is then insinuated between it and the isthmus, down to the front of the trachea. With the finger in position, the fascia is cut with scissors down the midline, in order to expose the trachea and the inferior thyroid veins. These veins, draining into the left innominate vein, are doubly ligated and sectioned. The fascial space between the thyroid isthmus and the trachea is then opened by means of forceps. Transverse section of the infrahyoid muscles (Fig. 473) is undertaken if the lobe is found to be unduly adherent, friable, or vascular, or if retraction gives a poor exposure.

potassium iodide, and admitted to the hospital in a sufficiently non-toxic state, undergo operation within forty-eight hours of admission. Those thyrotoxic patients who are admitted with auricular fibrillation and signs of congestive heart failure are kept at complete rest in bed. If the heart rate, as counted at the apex, exceeds 160 per minute, sodium iodide, 0.5 gm. in 1 litre of isotonic saline, is given intravenously. Salyrgan (or a similar drug) is also sometimes necessary. Quinidine is never given before operation. Subtotal thyroidectomy is withheld until the physician is satisfied that the congestive failure has been reduced to a minimum. X-rays of the chest are necessary in patients with nodular goitres to demonstrate any possible retrosternal prolongation. A laryngoscopic examination of the vocal cords is usually done to detect any involvement of the recurrent laryngeal nerves, and always before operating on a regrowth of a goitre.

On the day previous to operation, a fluid diet is given, and a small enema or a glycerine suppository is ordered if necessary. The site of operation is prepared. Sodium barbitone (medinal) 0.3 to 0.6 gm. is given at night. On the morning of operation, a further skin preparation is carried out, and Omnopon 20 mg. ( $\frac{1}{2}$  gr.) and scopolamine 0.4 mg. ( $\frac{1}{4}$  ss gr.) are injected one hour before operation. A further injection of half the above doses of Omnopon and scopolamine is given half an hour before operation. Elderly patients, with evidence of cardiovascular degeneration, are given only one injection of Omnopon and scopolamine.

Anæsthesia is induced by a slow intravenous injection of 0.4 to 0.6 gm. of pentothal and is continued by means of nitrous oxide with the high percentage of oxygen necessary for all thyrotoxic patients. Endotracheal anæsthesia is reserved specially for those patients with hard and fixed goitres (including carcinoma of the thyroid, lymphadenoid and Riedel's thyroiditis, retrosternal goitre, and those goitres with substernal or retroclavicular prolongations), and also for those patients in whom there is tracheal compression.

### THE OPERATION

The adjustable bridge of the operating table is raised under the patient's shoulders until the neck is extended and prominent. The surgeon, using a continuous action syringe with a 3-inch needle attached, injects the subcutaneous tissue of the neck for an area extending from the thyroid notch of the larynx above, to the suprasternal notch below, and laterally as far as the external jugular veins. The solution used is 1/200,000 adrenaline in normal saline. This technique lessens capillary oozing and also facilitates raising the flaps by clearly defining the fascial planes.

The incision is transverse and slightly convex downwards. Its level is approximately  $1\frac{1}{2}$  inches above the suprasternal notch and usually extends from one external jugular vein to the other. It is continued through the superficial fascia and the platysma muscle. Care is taken not to go beyond

as the anterior jugular veins may be injured. Reflection of the skin is then begun by nicking the areolar tissue deep to the platysma muscle. The upper flap is being rolled upwards. The upper flap should be reflected to the level of the thyroid notch of the larynx. In cases with very large goitres or high upward prolongations, the flap is reflected to the level of the hyoid bone. The lower flap is then similarly

A line sagittal incision is made through the fascia between the



473. Separation, retraction and section of the "strap" muscles. Note Collie's self-retaining skin-flap retractors. (*Techniques in British Surgery.*)

and muscles (Fig. 473) and enlarged with scissors upwards to the thyroid notch, and downwards to the suprasternal notch. The thyroid gland is now visible, covered by its deep fascia (surgical capsule). This is opened with blunt dissecting forceps and the forefinger of the left hand is then insinuated between it and the isthmus, down to the front of the trachea. With the finger in position, the fascia is cut with scissors in the midline, in order to expose the trachea and the inferior thyroid veins. These veins, draining into the left innominate vein, are doubly ligated and sectioned. The fascial space between the thyroid isthmus and the trachea is then opened by means of forceps. Transverse section of the thyroid muscles (Fig. 473) is undertaken if the lobe is found to be adherent, friable, or vascular, or if retraction gives a poor exposure.

potassium iodide, and admitted to the hospital in a sufficiently non-toxic state, undergo operation within forty-eight hours of admission. Those thyrotoxic patients who are admitted with auricular fibrillation and signs of congestive heart failure are kept at complete rest in bed. If the heart rate, as counted at the apex, exceeds 160 per minute, sodium iodide, 0.5 gm. in 1 litre of isotonic saline, is given intravenously. Salyrgan (or a similar drug) is also sometimes necessary. *Quinidine* is never given before operation. Subtotal thyroidectomy is withheld until the physician is satisfied that the congestive failure has been reduced to a minimum. *X-rays* of the chest are necessary in patients with nodular goitres to demonstrate any possible retrosternal prolongation. A *laryngoscopic examination* of the vocal cords is usually done to detect any involvement of the recurrent laryngeal nerves, and always before operating on a regrowth of a goitre.

On the day previous to operation, a fluid diet is given, and a small enema or a glycerine suppository is ordered if necessary. The site of operation is prepared. Sodium barbitone (medinal) 0.3 to 0.6 gm. is given at night. On the morning of operation, a further skin preparation is carried out, and Omnopon 20 mg. ( $\frac{1}{2}$  gr.) and scopolamin 0.4 mg. ( $\frac{1}{400}$  gr.) are injected one hour before operation. A further injection of half the above doses of Omnopon and scopolamine is given half an hour before operation. Elderly patients, with evidence of cardiovascular degeneration, are given only one injection of Omnopon and scopolamine.

*Anæsthesia* is induced by a slow intravenous injection of 0.4 to 0.6 gm. of pentothal and is continued by means of nitrous oxide with the high percentage of oxygen necessary for all thyrotoxic patients. *Endotracheal anæsthesia* is reserved specially for those patients with hard and fixed goitres (including carcinoma of the thyroid, lymphadenoid and Riedel's thyroiditis, retrosternal goitre, and those goitres with substernal or retroclavicular prolongations), and also for those patients in whom there is tracheal compression.

### THE OPERATION

The adjustable bridge of the operating table is raised under the patient's shoulders until the neck is extended and prominent. The surgeon, using a continuous action syringe with a 3-inch needle attached, injects the subcutaneous tissue of the neck for an area extending from the thyroid notch of the larynx above, to the suprasternal notch below, and laterally as far as the external jugular veins. The solution used is 1/200,000 adrenaline in normal saline. This technique lessens capillary oozing and also facilitates raising the flaps by clearly defining the fascial planes.

The incision is transverse and slightly convex downwards. Its level is approximately  $1\frac{1}{2}$  inches above the suprasternal notch and usually extends from one external jugular vein to the other. It is continued through the superficial fascia and the platysma muscle. Care is taken not to go beyond

Ligation of the *inferior thyroid artery* is an essential step in the operation. It considerably lessens the tendency to regrowth of the gland, allows transection of the gland with a minimal loss of blood, diminishes the danger of post-operative hæmorrhage and it enables more gland tissue to be left behind, thus ensuring the safety of the parathyroid bodies and of the recurrent laryngeal nerves. The artery, a branch of the thyro-cervical trunk, is not constant in position, but it usually passes medially and transversely from behind the carotid sheath to enter the postero-inferior surface of the lobe near the junction of its middle and lower thirds. The safety of the recurrent laryngeal nerve is ensured by isolating and ligating the inferior thyroid artery just as it emerges from behind the common carotid artery.

The upper and lower poles of the dislocated lobe are now clamped and cut. The intervening bridge of tissue lying between the forceps is carefully inspected before being sectioned; the *parathyroid glands* may be recognised lying closely apposed to the posterior surface of the lobe, outside its true capsule. They are terra cotta in colour, but inconstant in size and position. Great care must be taken to preserve these glandules, as tetany may follow their removal or injury. Parathyroid tissue may be recognised by its tendency to become dark blue when only slightly contused. If a parathyroid body is found to be detached it is implanted into the sternomastoid muscle. The *recurrent laryngeal nerve* must be carefully safeguarded by not encroaching on its normal anatomical position with forceps or ligatures, and by careful search elsewhere in case the nerve has been displaced from its normal position by the goitre. After careful scrutiny, the intervening bridge of tissue is sectioned obliquely down to the trachea and the left freed lobe is then rotated across the midline whilst the thyroid isthmus is being cleared by scissors from the trachea (Fig. 475). The cut surface of the thyroid remnant is approximated and anchored to the side wall of the trachea by means of interrupted catgut sutures. The *pyramidal lobe*, present in 75 per cent. of goitres, is mobilised and detached at this stage.

The procedure followed for resection of the left lobe is now repeated on the right side. It is advisable to drain the residual cavity in the majority of cases. A small strip of corrugated rubber is passed bilaterally through an opening made just lateral to the "strap" muscles and emerges at the lateral extremity of the skin incision (Fig. 476). The infrahyoid muscles if divided are sutured, and the deep fascia is closed in the midline by a continuous catgut suture. The platysma is then sutured and the skin edges approximated by clips (Fig. 476).

**Amount of Thyroid Tissue to be Conserved.** This depends upon :—  
(1) The pathology of the goitre; (2) its size; (3) the degree of toxicity; (4) whether the latter has been controlled by Neomercazole; (5) whether the inferior thyroid vessels have been ligated.

**Primary Toxic Goitre.** (a) Average toxicity and size: one-eighth of

Cutting the muscles leads neither to impairment of the cosmetic result nor to any limitation of movements if followed by careful suture. It provides a better exposure in a difficult case and facilitates the recognition and ligation of the middle thyroid veins and also the dislocation of the lobe. The *middle thyroid veins* vary in size, number, and position, and are occasionally absent. When present, they are isolated from the surrounding fascia by blunt dissection, doubly ligated and sectioned. The left lobe, with the exception of the upper pole, is now dislocated

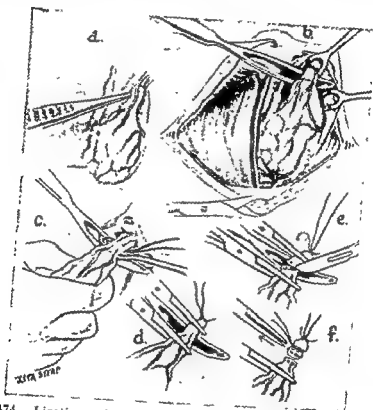


FIG. 474. Ligation and section of the superior thyroid leash of vessels.  
(*Techniques in British Surgery.*)

freely from its bed with the fingers. The upper pole is wedge-shaped with the thin edge of the wedge bound down to the pharynx posteriorly by fascia. When this fascia is stripped free, the pole can be levered forwards and downwards and dislocated fully into the wound. The freeing of this posterior wedge must precede the complete dislocation of the upper pole. Dissecting forceps are now used to open up the potential space between the inner aspect of the upper pole and the cricothyroid muscle. A Kocher's blunt director is slipped into this space and is passed under the pole to emerge on its freed outer aspect (Fig. 474). The upper pole and its vessels (terminal branches of the *superior thyroid artery*) now lie superficial to this instrument, and the vessels can easily be controlled and sectioned.

the gland is conserved; (b) severe toxicity: one-tenth of the gland is retained.

*Toxic Nodular Goitre.* Rather more tissue is conserved than the one-eighth retained in the average primary toxic goitre and the subtotal thyroidectomy is, in fact, *selective*: i.e. the line of resection is planned to conserve that tissue which approximates as near as possible to normal. The upper poles are often found to be least involved and this fact is made use of when assessing the amount and position of the tissue to be retained. However, when adenomas are palpable in the posterior margin of the lobe (that portion to be conserved), the lobe is sectioned at a more superficial level to retain a sufficient amount of thyroid tissue after the adenomas have been shelled out from the cut surface. The compressed capsule is then reconstituted into a lobe.

Elderly patients with secondary toxic goitre, and particularly those with auricular fibrillation and congestive heart failure, undergo an almost complete thyroidectomy. A small strip of tissue is left behind on either side; surprisingly, such patients do not develop myxœdema.

The part of the thyroid lobe remaining consists of its posterior border and that portion of tissue adjacent to the posterolateral aspect of the trachea to ensure the safety of the recurrent laryngeal nerve and the parathyroid bodies.

#### POST-OPERATIVE CARE

The patient, on returning to the ward, is placed in a modified Fowler's position. An injection of morphine 10 mg. ( $\frac{1}{4}$  gr.) is given. Discomfort behind the neck, and pain in the region of the gums and ears is frequently complained of; these symptoms respond to aspirin and phenacetin. A post-operative reaction, indicated by a rise of temperature and pulse rate, usually subsides within forty-eight hours; a more severe reaction may take several days to settle. All toxic patients are given potassium iodide 0.3 gm. daily post-operatively for six or seven days. Barbitone 30 mg. ( $\frac{1}{2}$  gr.) is given three times a day; it is valuable for those patients who are nervous and apprehensive. Vomiting is uncommon, but if it occurs the patient is encouraged to drink as much as possible. *Tracheitis* is rarely encountered; it is relieved by steam inhalations containing tincture of benzoin. *Drainage strips* are removed twenty-four hours, and skin clips forty-eight hours after operation. Dressings are dispensed with on the third day. The wound is dusted with sulphathiazole powder and is covered by a gauze or lint collar which is attached at the back of the neck by tapes. *Blood-stained serum* may collect and give rise to a small fluctuating area. These effusions usually localise at the lateral margins of the wound. They are probed and freed with the end of a blunt needle. Penicillin is ordered if there is evidence of inflammation. The patient is allowed up on the second day after operation and usually discharged from hospital on the seventh or eighth day.



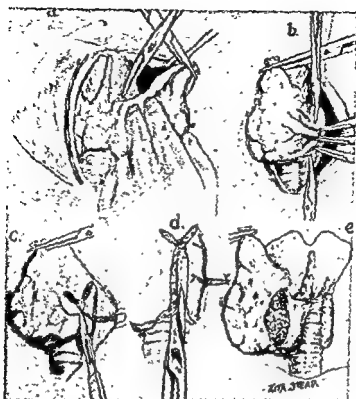


FIG. 475. Section of a thyroid lobe. (*Techniques in British Surgery.*)

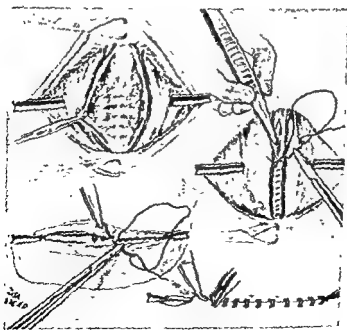


FIG. 476. Method of drainage and closure of the muscles, platysma and skin. (*Techniques in British Surgery.*)

consciousness. If, however, the paralysis is due to post-operative œdema of the nerve, or to its compression by fibrous tissue, days or weeks elapse before the signs become apparent. Injury to a recurrent laryngeal nerve during subtotal thyroidectomy is in most cases an avoidable accident. Injury to both nerves during operation must be a very rare tragedy. Paralysis of both vocal cords is usually the result of an injury to one nerve when operating on a patient who already has an unrecognised paralysis of the other cord. It follows, therefore, that preoperative laryngoscopy is especially necessary in patients who have noticed a change in the voice and in those who are to undergo operation for recurrent goitre. Should this examination demonstrate a unilateral cord paralysis, meticulous care must be taken to avoid injury to the nerve on the normal side. An operation devised by King, to retract and tether the arytenoid cartilages, greatly relieves patients with bilateral vocal cord paralysis.

**Embolism.** Arterial embolism may result from thyrotoxic auricular fibrillation.

**Post-operative Myxœdema.** Myxœdema is rare after subtotal removal of a toxic diffuse goitre. It is much more liable to follow removal of a goitre with lymphadenoid changes. Elderly patients need very little thyroid tissue to maintain their metabolic balance. *Treatment* consists in the administration of thyroid extract. The amount given is gradually decreased as improvement occurs. Many patients ultimately regain a normal metabolism, due to regeneration of thyroid remnants.

**Parathyroid Tetany.** This is usually the result of interference with the blood supply of the parathyroid bodies. Symptoms may appear at any time up to forty-eight hours following operation, and may continue for several hours or days. *Treatment.*—Mild transient tetany, as revealed by tingling and stiffness of the fingers, responds quickly to intensive calcium therapy. A heaped teaspoonful of calcium lactate is given at least four times daily and large quantities of milk are ordered. In more severe tetany, 10 ml. of a 10 per cent. solution of calcium gluconate is also given intravenously. Parathyroid extract is reserved for the very rare generalised and acute tetany. Treatment is only necessary for a short time, as the function of the parathyroid glands is soon restored to normal, although tetany resulting from removal of the parathyroid glands requires therapy indefinitely.

**Unsightly Scars.** These are usually the result of local subcutaneous infection. The infection is treated with antibiotics or the sulphonamides. A fluctuating area is probed to allow drainage of any pus. A disfiguring scar, or adhesion, as shown by an upward tugging of the scar on swallowing, may ensue. Several months must be allowed to elapse before the scar is excised and the adhesions separated. *Keloid* formation in a scar is unpredictable; the presence of keloid scars elsewhere on the patient, however, points to this possibility. Two or three short treatments by X-ray therapy improve the condition if given early.

*Auricular Fibrillation.* The administration of digitalis is continued after operation to those patients admitted with auricular fibrillation. It is discontinued when the ventricular rhythm has become regular; if this has not occurred by the sixth or seventh day, the drug is replaced by quinidine sulphate. A tolerance dose of 0.2 gm. (3 gr.) of quinidine is given initially. If this does not cause untoward symptoms, 0.4 gm. (6 gr.) of the drug is given every four hours until the ventricular rhythm becomes regular. The return to normal rhythm, when it occurs, usually does so within the first three days of treatment; at such time, the dose is reduced to 0.2 gm. every four hours for a further forty-eight hours. In the event of the auricular fibrillation remaining uncontrolled, the drug is discontinued after six days. *Transient* auricular fibrillation frequently develops after operation in those patients who have had nodular goitres for many years. This temporary irregularity may continue from twelve to twenty-four hours. Quinidine is not indicated in these cases.

### POST-OPERATIVE COMPLICATIONS

**Hæmorrhage** due to slipping of a ligature from an artery or from a large vein quickly produces cyanosis and stridor by pressure on the trachea. Swelling of the neck need not be obvious. The wound must be reopened immediately in the ward and the deep clot removed. The acute obstruction having been relieved, the patient is transported to the operating theatre where the bleeding vessel is located and ligated under general anaesthesia. Hæmorrhage from small vessels or general oozing gives rise to slower and more insidious obstruction. Slight cyanosis and huskiness of the voice may be present, and is associated with diffuse swelling of the neck. The patient is transferred to the operating theatre where the wound is reopened; the clot is removed and the vessel, if located, is ligated. In either case, if obstruction remains after removal of the clot, tracheal collapse is present and tracheotomy must therefore be performed.

**Thyrototoxic Crisis.** This should no longer be encountered, as toxicity should be controlled before operation by antithyroid drugs. It presents with a rapid pulse, restlessness, a high temperature, sweating, vomiting and diarrhoea, and often culminates in delirium. The patient is given morphine, oxygen by means of nasal catheters or oxygen tent, intravenous sodium iodide (1 gm. twice daily given in intravenous saline), and fluids intravenously and by mouth.

**Pulmonary Complications.** Pulmonary complications are unusual. They include pneumonia, acute pulmonary oedema, and pulmonary embolism.

**Recurrent Laryngeal Nerve Injury** (see also under *The Cranial Nerves*, Chap. 52). Evidence of paralysis of one or both vocal cords is apparent, either at the time of the injury or soon after the patient has regained

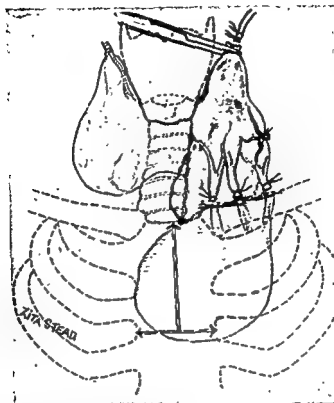


FIG. 477. The line of section through the sternum to allow for delivery of an impacted retrosternal goitre. The transverse section is not always necessary. (*Techniques in British Surgery.*)

### CARDIAC FAILURE

Total or near total thyroidectomy may occasionally be considered for patients suffering from cardiac failure whether the failure is associated with thyrotoxicosis or not. The metabolism having been reduced by the operation, it follows that the tissues need less oxygen and the damaged heart is able to cope more adequately. Operation is not, however, undertaken in the thyrocardiac patient with congestive heart failure until the failure is controlled by means of rest, diuretics and digitalis. In the event of the cardiac reserve being dangerously low and response to medical treatment poor, the final treatment of choice would be radioactive iodine therapy.

### REMOVAL OF A DISCRETE ADENOMA

The same steps and technique are followed as in the exposure of a toxic goitre. The incision must be sufficiently long to allow for exploration of both lobes. In all operations for adenoma, the lobe of the opposite side must be explored. If an adenoma is discovered there it is enucleated; if the lobe is hyperplastic or enlarged its anterior part is resected.

### RECURRENT GOITRE

Regrowth of either a diffuse or a nodular goitre may occur after subtotal thyroidectomy. A recurrence is usually due to the fact that the inferior thyroid arteries have not been tied, too much hyperplastic tissue has been retained or that the retained tissue is nodular. The recurrence rate with unligated arteries is from 2 to 6 per cent., whereas it is less than 1 per cent. when the arteries have been tied and all nodular tissue removed. Incomplete removal of the pyramidal lobe is often followed by hypertrophy of the remaining portion. This will ultimately produce an unsightly swelling in the midline of the neck which is often accompanied by a recurrence of toxic symptoms. The apparent recurrence following the removal of a discrete adenoma is not a regrowth. It is the result of the continued growth of another adenoma which was small and unrecognised at the initial operation. Removal of a recurrent goitre may provide one of the most difficult operations in thyroid surgery. The adhesions resulting from the first operation may make it difficult or impossible to find a line of cleavage between the gland and the deep fascia. The internal jugular vein is often adherent to the gland, and the recurrent laryngeal nerves and parathyroid bodies are difficult to locate, being either hidden by fibrous tissue or densely adherent to the thyroid.

### DELIVERY OF A RETROSTERNAL GOITRE

After the lobe has been freed in the neck, upward traction of the lobe is made with the left hand, whilst the forefinger of the right hand, keeping in close contact with the true capsule of the adenomatous mass, is inserted into the mediastinum. Maintaining the closest possible contact with the capsule, the finger is swept around, separating the pleura and cellular tissues from it. As the traction and separation progress, the adenomatous mass is gradually drawn upwards until the fingers can be insinuated below its lower margin. The tumour is then levered upwards from the mediastinum and delivered into the neck. *Splitting the sternum* (Fig. 477) may be necessary to enable delivery of an impacted retrosternal goitre, when it is of solid consistency or when its capsule is adherent to mediastinal tissues. This extra exposure is essential when the retrosternal tumour is found to be malignant and when dealing with a true or aberrant intrathoracic goitre.

as being either persistent or enlarged. *Microscopically*, however, the thymus of myasthenia gravis is unlike the normal thymus; there are germinal-centre or lymphoid-follicle formations and increased cellularity of cortex and medulla.

A thymoma or thymic tumour (Fig. 479) is found in 12 to 14 per cent. of patients suffering from myasthenia gravis who undergo operation. This type of neoplasm occurs only with myasthenia gravis and is termed a *thymo-lympho-epithelioma*, being a mixture of epithelial and lymphocytic cells in varying proportions. The tumour is encapsulated and slowly growing, usually having the characteristics of a benign neoplasm although it must be considered potentially malignant as some of them invade the pleura, lung and myocardium and occasionally give rise to secondary deposits within the thoracic cavity.

Myasthenia gravis is more common in women than in men in the ratio of 2 : 1, and the majority of patients are between the ages of fifteen and forty years. When associated with thymic neoplasm, however, myasthenia gravis is relatively more common in the male. Tumours, in either sex, usually occur between the ages of thirty and fifty-five

years. The early symptoms of myasthenia gravis are variable, and are often dependent on the occupation of the patient. The shorthand-typist may notice "fatiguability" of the wrists and fingers, whereas the cyclist may notice weakness of the thigh muscles. The clinical picture is never the same in any two patients, as any muscle or group of muscles may be involved first. Other muscle groups are then slowly affected. Patients may first complain of drooping eyelids (Fig. 480) and double vision, later followed by difficulty in smiling and swallowing. Speech may become slurred and nasal, and is often associated with difficulty in swallowing and nasal regurgitation of fluids. The limbs and trunk may next be involved. The spread of muscle "fatiguability" may advance quickly into a complete and generalised picture of the disease, or more slowly, perhaps leaving the lower limbs uninvolved for many months. The process may be continuous or intermittent. The *bulbar type* of myasthenia gravis is that in which only the muscles supplied by the bulbar nerves are involved. The picture of severe generalised myasthenia gravis is a pathetic one. The patient lies helpless in bed unable to speak, to swallow, or to raise



FIG. 479. A thymoma in the left lobe of the thymus.

## CHAPTER 70

### THE THYMUS

(IN RELATION TO MYASTHENIA GRAVIS)

THE association of myasthenia gravis with epithelial tumours of the thymus had long been known, but it was not until 1936 that Blalock of Baltimore removed such a tumour, and thus relieved the associated myasthenia gravis. Similar results were obtained by removing apparently normal thymus glands from myasthenic patients, and much work and research has since been undertaken to place the operation of thymectomy on a rational basis.

Myasthenia gravis is characterised by an abnormally rapid exhaustion of the voluntary muscles, and it has recently been shown that the thymus gland releases or is intimately concerned with the release of a substance which affects neuromuscular transmission. The "fatiguability" is caused by this curare-like substance from the thymus interfering with the action of acetyl choline. This thymic secretion extracted from thymus glands removed from patients suffering from myasthenia gravis, has a specific action on muscle-nerve preparations. It reduces muscle contrac-

tions and also gives rise to temporary paralysis when injected into small animals. The formation and discharge of this substance by the thymus explains the fact that thymectomy can alleviate or cure myasthenia gravis. If the disease has been present for many years, however, irreversible changes may have occurred in the voluntary muscles, and these changes result in a poor response to neostigmine (prostigmine) and to an unsatisfactory result from thymectomy. The thymus gland enlarges from birth until puberty, and then slowly decreases in size to become atrophic in the aged, although it does not entirely disappear. It weighs anything from 5 to 40 gm., varying considerably both in normal and myasthenic individuals of the same age. The thymus of myasthenia gravis is not necessarily larger than normal, nor does its size bear any relation to the severity of the disease (Fig. 478). Its appearance, therefore, is not unlike that of a normal thymus and it should not be described



FIG. 478. The thymus gland removed from a patient, aged thirty years, suffering from myasthenia gravis.

side effects. The early and most common ones are gastro-intestinal disturbances (nausea, colic and vomiting), sweating, pallor and fasciculation of voluntary muscles. The drugs should be taken just before or with meals, to lessen or prevent these side effects. Myasthenia gravis is often progressive, requiring the administration of ever-increasing doses of the cholinergic drugs at shorter and shorter intervals. In long-standing cases the dosage required is often very large and the response limited, for the condition is likely to have become partially irreversible. In such patients,



FIG. 481. (a) A patient with myasthenia gravis showing ptosis and drooping lips. (b) The same patient with a temporary increase of muscular power twenty minutes after neostigmine by mouth.

side effects, as shown by intense stimulation of the gastro-intestinal tract, make it difficult to gain a satisfactory therapeutic response unless atropine is also given at regular intervals.

Operation is recommended for all patients suffering from generalised myasthenia gravis in whom the weakness is reversible, except for elderly patients or those suffering from other diseases. The need for an ever increasing dosage of neostigmine and lessening response to the drug is an added indication for operation, as this suggests the presence of a thymoma. If a tumour is verified by radiography, operation is undertaken after the completion of X-ray therapy. Patients with mild localised and easily controlled myasthenia in whom ocular symptoms predominate, may be kept under observation until evidence of increasing severity or spread of the myasthenia manifests itself.



the limbs from the bed. An attempt at drinking causes weak coughing, dyspnoea and regurgitation of fluids through the nose. The mouth sags open and the patient is unable to cough up bronchial secretions.

**Diagnostic Tests.** (1) Ensure that the weakness is a "fatiguability" of muscles, i.e. made worse by repetitive exercise and improved by rest. (2) The response to neostigmine, 1 to 2 mg. by intramuscular injection (equivalent in action to 30 to 60 mg. by mouth). Some response always takes place in from ten to thirty minutes. (3) The intravenous injection of Tensilon rapidly gives rise to a brief increase of muscular strength in myasthenia. (4) The presence of resistance to the depolarising activity of decamethonium (C 10) may be used as a diagnostic test.



FIG. 480. Bilateral ptosis and drooping of the lower jaw in myasthenia gravis.

### TREATMENT

The anticholinesterase (cholinergic) drugs do not directly influence the disease process, and are not in any way concerned with the development of remissions. They are used not in the hope of a cure, but to increase the muscular power temporarily. Administration of the drugs is repeated at intervals as the muscular power diminishes (Fig. 481). Every patient is studied individually to determine the requirements of neostigmine.

The aim is to gain as much activity as possible without side effects. If the myasthenia is mild, one tablet (15 mg.) of neostigmine three or four times a day may suffice. Any degree of severity of the disease may be encountered, requiring up to 750 mg. neostigmine daily, or even more. In the severe cases, tablets may need to be reinforced by 1 or 2 mg. of the drug by intramuscular injection. An injection is necessary first thing in the morning if the patient is too weak to swallow tablets. Nobody is better able to judge the requirements of neostigmine than the patient. *Mestinon* (pyridostigmine bromide), a more recent drug, is an analogue of neostigmine; a tablet of 60 mg. is equal in strength to 15 mg. of neostigmine. In the majority of cases of myasthenia gravis it acts slightly longer than neostigmine and produces fewer side effects. *Overdoses* of cholinergic drugs give rise to parasympathetic stimulation, leading to uncomfortable

endotracheal tubes. The anæsthetic is given through an efficient airway and well-fitting mask, held in position by a rubber harness. The anæsthesia is kept light, so that the patient can quickly and rapidly resume a normal, adequate respiratory excursion.

### THYMECTOMY

A small horizontal incision is made just above the suprasternal notch extending down to the deep fascia (Fig. 482). A further incision begins at the middle of the horizontal incision and extends vertically down the midline of the sternum for approximately three-quarters of its length, or to the level of the fifth or sixth rib. The deep cervical fascia is opened by

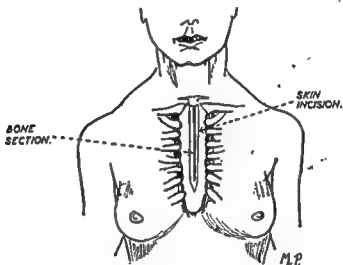


FIG. 482. The skin incision and line of sternum section in thymectomy. (*Textbook of British Surgery*, Vol. II.)

a vertical midline incision to expose the lower border of the thyroid gland and frequently in this situation the cornua of the thymus gland. In this plane, the forefinger is forced to its full length directly under the sternum, into the anterior mediastinum. The lower blunt blade of a Sauerbruch's sternum splitter is inserted through the cervical opening into the mediastinum and a clean sharp bite into the midline of the sternum is made. As the assistant forcibly retracts the cut edges of the sternum, several further bites are taken in a similar manner. The sternum is thus gradually divided to the required length (Fig. 483). With the divided sternum adequately retracted, the next procedure is to expose fully the anterior surface and lateral margins of the thymus, and then to dissect it free from the surrounding structures (pleura, aorta and pericardium). Blood vessels are ligated and divided as they appear. When the surgeon is uncertain as to the whereabouts of the pleura, the anæsthetist is asked to

## PREOPERATIVE CARE

The myasthenic patient, although co-operative and eager for surgical help, is often anxious, fearful and inclined to panic, and therefore should be admitted to hospital at least a week before operation. During this period of observation, confidence is gained, the diagnosis is verified and the optimum dosage of neostigmine or Mestinon is established.

**Assessment of Severity of the Myasthenia.** Soon after admission, the patient, with due warning, is taken off the anticholinesterase drug for approximately eighteen hours, although the period would be less if the patient became seriously distressed or helpless. A full examination and test of muscle power is then made and recorded. A *cholinergic* drug is then injected, either neostigmine 1 to 2 mg. intramuscularly or the rapidly acting Tensilon, 10 mg. intravenously. A syringe containing 0.65 mg. (100 gr.) of atropine should be in readiness in case of gastro-intestinal side effects. The patient is re-examined ten to thirty minutes after the injection of neostigmine and the increased muscular activity and any residual weakness recorded. The more complete the response to the injection, the higher is the scale of activity and the greater the confidence in regard to control of the myasthenic state during the post-operative period. *Residual muscular weakness* after an adequate injection of neostigmine is evidence of some irreversibility of the myasthenia. The patient should regain sufficient power to produce a sharp explosive cough, otherwise inability to expel plugs of mucus from the bronchi could lead to post-operative collapse of the lungs. The patient is maintained before the operation on the maximum dose of neostigmine that can be tolerated without side effects. An X-ray of the chest is taken in search of a thymic tumour. If such a tumour is detected, operation is postponed until after the patient has had a course of deep X-ray therapy which improves the results of operation when a thymic tumour is the cause of the myasthenia gravis and does not increase the technical difficulties of the operation. The normal thymus gland or that of myasthenia gravis not due to a tumour cannot be demonstrated on an X-ray film. The majority of thymic tumours associated with myasthenia gravis, however, are relatively radio-opaque and can be best seen in a lateral view of the anterior mediastinum, lying on the aorta or on the pericardium. Postero-anterior views are less helpful, but tomography is of value.

An *enema* must *never* be given to a patient suffering from myasthenia gravis, for it may cause severe collapse or a syncopal attack in which the patient becomes unconscious, cold, pallid and clammy. This temporary collapse may be due to stimulation of a parasympathetic system already sensitive and overactivated by cholinergic drugs.

**The Anaesthetic.** Avoid agents with *curare* properties. Anaesthesia is induced with intravenous pentothal and continued with oxygen and cyclopropane. Irritation is reduced to a minimum by avoiding ether and

endotracheal tubes. The anæsthetic is given through an efficient airway and well-fitting mask, held in position by a rubber harness. The anæsthesia is kept light, so that the patient can quickly and rapidly resume a normal, adequate respiratory excursion.

### THYMECTOMY

A small horizontal incision is made just above the suprasternal notch extending down to the deep fascia (Fig. 482). A further incision begins at the middle of the horizontal incision and extends vertically down the midline of the sternum for approximately three-quarters of its length, or to the level of the fifth or sixth rib. The deep cervical fascia is opened by

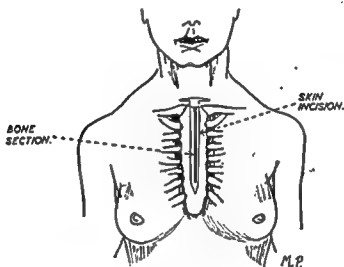


FIG. 482. The skin incision and line of sternum section in thymectomy. (*Textbook of British Surgery, Vol. II.*)

a vertical midline incision to expose the lower border of the thyroid gland and frequently in this situation the cornua of the thymus gland. In this plane, the forefinger is forced to its full length directly under the sternum, into the anterior mediastinum. The lower blunt blade of a Sauerbruch's sternum splitter is inserted through the cervical opening into the mediastinum and a clean sharp bite into the midline of the sternum is made. As the assistant forcibly retracts the cut edges of the sternum, several further bites are taken in a similar manner. The sternum is thus gradually divided to the required length (Fig. 483). With the divided sternum adequately retracted, the next procedure is to expose fully the anterior surface and lateral margins of the thymus, and then to dissect it free from the surrounding structures (pleura, aorta and pericardium). Blood vessels are ligated and divided as they appear. When the surgeon is uncertain as to the whereabouts of the pleura, the anæsthetist is asked to

inflate the lungs; after this, the pleura and the mottled lung beneath bulge forward and are easily identified.

The upper poles (cornua) of the gland are identified and exposed, extending up into the neck, often to the level of the lower border of the thyroid isthmus. The cornua are freed and as they are turned down the under surface of each lobe is separated from the left innominate vein. It is at this stage that care is taken to identify, ligate and section the main thymic vein ("great vein of Keynes") which drains into the left innominate vein. The cornua, the lateral

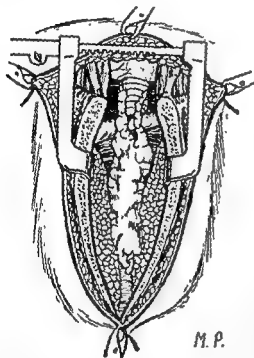


FIG. 483. The connective tissue has been incised and the thymus gland exposed. (The lungs and pleura lie to either side; the left innominate vein, aorta and pericardium behind.) (*Textbook of British Surgery, Vol. II.*)

borders and the posterior surface of the gland having been freed, all that now remains is to strip the lowest portion of the gland from the pericardium. If by mischance the pleura is opened during the dissection, a sucking noise is heard on inspiration, and the anaesthetist is immediately warned to expand the lungs. The hole is then sutured with a continuous stitch of fine catgut. When the thymus gland has been removed, the remaining cavity, bounded by the aorta, pericardium, lungs and innominate vein is inspected for oozing before the retractor is removed and the cut edges of the sternum allowed to fall together. Each cut edge of the sternum is bored obliquely down to its inner table and a piece of No. 2 chromic catgut is then threaded through the perforated end of an awl and withdrawn through the bone with the instrument. This is repeated

in line on the opposite side and the ends are ready for tying. When this procedure has been repeated at three or four sites, the chromic catgut sutures are firmly tied one by one, locking the cut edges of the bone closely together. The divided periosteum is united with interrupted sutures of catgut. The skin incision is then closed.

The above description of thymectomy pertains also to removal of a thymic tumour. With the divided sternum adequately retracted, the thymic tumour is easily identified as a hard irregular nodule quite unlike the soft glandular consistency of the rest of the thymus. It varies in size from a walnut to a tangerine orange. The thymus is exposed and freed as described above; that portion containing the tumour is left until last. An attempt is then made to peel the thickened pleura from the surface of

the tumour, but if this is impossible the lung is fully expanded by the anaesthetist and the adherent pleura excised with the tumour. Removal of a thymic tumour from the aorta or from the pericardium is usually a straightforward procedure, although on occasions a portion of the pericardium may have to be excised with neoplasm. It is rare for a tumour to spread towards the root of the lung or to invade the lung.

### POST-OPERATIVE CARE AND COMPLICATIONS

Myasthenia gravis gives rise to a problem in surgery which is met with in no other condition. It is the danger of post-operative respiratory paralysis, which may follow either a myasthenic or a cholinergic crisis.

**Myasthenic Crisis.** The weakness of myasthenia gravis is made worse by any infection (e.g. dental abscess or influenza), and is also temporarily worsened by the pain and trauma of an operation. The patient with a limited ability to cough because of myasthenia, made still more limited by mediastinotomy, may be unable to expel a plug of mucus from a bronchus and thus develop collapse of the lung. If by mischance the pleura was opened during the operation, partial collapse of the lung could also occur from this cause. A desire to cough is then created. A vicious circle is thus set up, leading to an increase in the myasthenic state and possibly culminating in myasthenic respiratory paralysis. To offset this danger, meticulous care must be taken to safeguard the pleura during the operation, and the optimum dose of neostigmine is given post-operatively to allow for an explosive cough with sufficient power to expel mucus. Overdosage must, however, be avoided, for it could lead to cholinergic paralysis.

**Cholinergic Crisis.** Cholinergic drugs, when given in doses beyond their maximum effectiveness, give rise to paralysis of voluntary muscles. The greatest care must therefore be taken to recognise the early stages of overdosage and to distinguish the weakness produced from that of myasthenia. Gastro-intestinal disturbances such as borborygmi, nausea and colic are its earliest signs; it is important not to mask them with atropine. Other signs are excessive salivation, cold sweats, fasciculation of the voluntary muscles, panic and respiratory weakness. The weakness of myasthenia gravis, on the other hand, is not accompanied by any of the above manifestations excepting, perhaps, anxiety and panic. The differential diagnosis, therefore, is not difficult if atropine has not been given to cloud the issue. If it has been given and the incorrect diagnosis of a myasthenic crisis is made, a further injection of neostigmine could result in respiratory paralysis and death. A myasthenic crisis is treated by injection of the maximum dose of a cholinergic drug and atropine is given if gastro-intestinal side effects appear. A cholinergic crisis is treated by stopping cholinergic drugs and giving intravenous atropine sulphate 1 to 3 mg.

**Treatment Common to Both Types of Crisis.** (1) Oxygen. (2) If the

inflate the lungs ; after this, the pleura and the mottled lung beneath bulge forward and are easily identified.

The upper poles (cornua) of the gland are identified and exposed, extending up into the neck, often to the level of the lower border of the thyroid isthmus. The cornua are freed and as they are turned down the under surface of each lobe is separated from the left innominate vein. It is at this stage that care is taken to identify, ligate and section the main thymic vein (" great vein of Keynes ") which drains into the left innominate vein. The cornua, the lateral

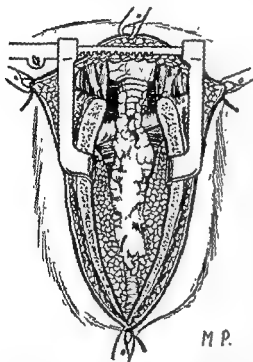


FIG. 483. The connective tissue has been incised and the thymus gland exposed. (The lungs and pleura lie to either side ; the left innominate vein, aorta and pericardium behind.) (*Textbook of British Surgery, Vol. II.*)

borders and the posterior surface of the gland having been freed, all that now remains is to strip the lowest portion of the gland from the pericardium. If by mischance the pleura is opened during the dissection, a sucking noise is heard on inspiration, and the anæsthetist is immediately warned to expand the lungs. The hole is then sutured with a continuous stitch of fine catgut. When the thymus gland has been removed, the remaining cavity, bounded by the aorta, pericardium, lungs and innominate vein is inspected for oozing before the retractor is removed and the cut edges of the sternum allowed to fall together. Each cut edge of the sternum is bored obliquely down to its inner table and a piece of No. 2 chromic catgut is then threaded through the perforated end of an awl and withdrawn through the bone with the instrument. This is repeated

in line on the opposite side and the ends are ready for tying. When this procedure has been repeated at three or four sites, the chromic catgut sutures are firmly tied one by one, locking the cut edges of the bone closely together. The divided periosteum is united with interrupted sutures of catgut. The skin incision is then closed.

The above description of thymectomy pertains also to removal of a thymic tumour. With the divided sternum adequately retracted, the thymic tumour is easily identified as a hard irregular nodule quite unlike the soft glandular consistency of the rest of the thymus. It varies in size from a walnut to a tangerine orange. The thymus is exposed and freed as described above ; that portion containing the tumour is left until last. An attempt is then made to peel the thickened pleura from the surface of

improvement ; there is the likelihood, however, that in these the progress of the disease will have been stopped. The latter category would comprise those patients of over forty-five years of age with a long history of the disease. Many patients with a poor or fair early result find their strength and activity steadily improving as time goes by.

The results of *thymectomy* for *thymic tumours* are far less good, although



FIG. 484. (a) A patient with *myasthenia gravis*. (b) The same patient ten days after *thymectomy*.

they have improved since the practice of giving preoperative X-ray therapy to the mediastinum. The operation is strongly indicated, however, in view of the potential malignancy of the tumour.

Much about the disease is still obscure and the field open to fruitful research. The diagnosis is often difficult and the prognosis uncertain, but with the aid of recent biochemical and other tests such as the use of decamethonium and electromyography, these should be made with greater certainty in the future.



weakness has become irreversible and does not respond to drugs and the cough remains ineffectual in expelling bronchial secretions, bronchoscopy and suction should be undertaken. (3) If respiratory paralysis has set in, immediate tracheotomy must be performed and suction instituted to help maintain a clear airway. Manual artificial respiration is then begun or may be provided by a respiratory apparatus. The Beaver respirator has proved satisfactory and can be attached to the tracheotomy tube. It provides controlled rhythmic respirations with the correct percentage of oxygen for the unconscious or paralysed patient. Continuous oxygen must be given, for any prolonged cerebral anoxia may result in coma. When the paralysis comes under the control of drugs, and consciousness and strength are to some extent regained, the respirator is detached and respiration allowed to proceed normally. The tracheotomy opening is retained for a few days to allow bronchial secretions to be sucked out.

The operative mortality in thymectomy is approximately 4 per cent.; the usual causes of death are pulmonary infections, respiratory paralysis or a myasthenic crisis. The patient suffering from myasthenia gravis is particularly liable to pulmonary infections; these increase the myasthenia. Every precaution must be taken to keep the patient free from infection. Thus those having contact with the patient must wear masks for several days after the operation. The maximum strength is maintained with the optimum dosage of neostigmine (careful watch being kept to recognise and treat impending myasthenic or cholinergic crises—*vide supra*). An enema must not be given for fear of inducing a cholinergic crisis; nor must any form of intravenous infusion be given as a routine, for fear of inducing œdema and collapse of the lungs. Quick recovery from the anæsthetic is essential, to allow for early resumption of adequate respiratory exchange and an early return to oral administration of cholinergic drugs. The number of tablets is gradually reduced as the myasthenia diminishes. A routine X-ray of the chest is taken with a portable apparatus twenty-four hours after operation so that any collapse of the lung can be promptly recognised. Penicillin is given at the slightest indication of infection. Pneumothorax or hæmothorax may occur if there is an unsutured tear of the pleura. Aspiration of the chest should be carried out when necessary.

Hæmorrhagic serum may collect in the mediastinum and seep through the sectioned sternum to give rise to a fluctuating pulsating swelling under the skin. This should be aspirated after the fifth or sixth day.

**Results of Thymectomy.** The results of operation are variable; the best are obtained when the patients are young and when the duration of the disease is short. A complete or near complete remission rate is gained in 70 per cent. of female patients under thirty-five years of age (Fig. 484). The majority of the remainder are improved, but still require a varying amount of neostigmine or Mestinon to increase their scale of activity to enable them to work. In 10 per cent. of cases there is no apparent

slight contusion (Fig. 485); other tissue does not react in the same manner. The function of the parathyroids is the production of a hormone which regulates the calcium content of the blood; the normal level of serum calcium is 9 to 11 mg. per 100 ml. *Hyperparathyroidism* or over-secretion of the hormone is characterised by a high level of serum calcium and a low plasma phosphorus, often with decalcification of bones. *Hypoparathyroidism* or undersecretion is characterised by a low level of serum calcium and tetany.

### HYPERPARATHYROIDISM

(Generalised Osteitis Fibrosa Cystica of von Recklinghausen \*)

In *hyperparathyroidism* the bones often decalcify and undergo fibrocystic changes. It must be recognised, however, that the condition can commonly occur without evidence of bone changes, particularly in the early stages of the disease. Nephrolithiasis and nephrocalcinosis (calcification of the kidneys) occur only occasionally without bone changes, but are quite common when such changes are present. The excessive production of parathormone may result from: (a) Parathyroid tumour; (b) simple hypertrophy of parathyroid bodies (c) hyperplasia of normal sized parathyroid bodies.

The *parathyroid tumour* (Fig. 485) is most commonly a benign adenoma; in 5 or 6 per cent. of cases there is more than one adenoma. Hyperparathyroidism is rarely caused by malignant parathyroid tumours as they are not sufficiently differentiated to secrete the hormone. *Histologically* the adenoma resembles normal parathyroid tissue. The tumours vary considerably in size, being usually between 2 and 4 cm. in diameter. They are greyish-yellow and oval or pyriform. They are smooth, soft and encapsulated. In *hyperparathyroidism*, the excessive production of parathormone results in hypercalcaemia with:

- (1) Constitutional symptoms.
- (2) Skeletal changes < diffuse "osteitis" fibrosa cystica.  
osteoporosis or decalcification of bones.
- (3) Renal changes—nephrolithiasis or kidney calcification (nephrocalcinosis).

**Constitutional Symptoms.** The onset and early stages of the condition are insidious, and a prolonged period of invalidism may have elapsed before decalcification of bones, renal symptoms and hypercalcaemia point to the diagnosis. Early complaints are of general asthenia and debility. Aching and tenderness of the bones and joints occur, and are often accompanied by nausea, anorexia and loss of weight. Some of these symptoms and signs result from the high level of serum calcium which lowers muscle tone, and leads to muscle atrophy and weakness (Fig. 486). These vague symptoms may continue for eighteen months or so until muscular weakness and loss of weight are severe and the increasing bone

\* Neurofibromatosis (Chap. 61) is also known as von Recklinghausen's disease.

## CHAPTER 71

### THE PARATHYROID GLANDS

THERE are normally four parathyroid glands situated in close but variable relationship with the posterior borders of the thyroid lobes. Sometimes there are more than four, but seldom less than four. They are oval or pear-shaped bodies about half the size of an average little finger nail. The glandules are brownish or terracotta in colour and are soft and floppy in consistency—quite unlike the texture or structure of the thyroid or its capsule to which they are attached. There are usually two on either side; the upper one is associated with the posterior branch of the superior thyroid artery; the lower one is associated with the inferior thyroid artery. Their blood supply is derived from branches of these arteries. There is, however, a rich anastomotic blood supply, for tetany rarely follows ligation of all four thyroid arteries. Apart from these usual positions, the parathyroids may be found anywhere on the posterolateral surface of the thyroid lobes or in the fat or fascia surrounding the inferior thyroid veins. The parathyroid bodies are never within the true capsule of the thyroid gland, although occasionally they may appear to be, when lodged in a furrow or lying behind a thyroid nodule. The parathyroids develop from the third and fourth branchial clefts—the third in association with the thyroid lobes, the fourth with the thymus. This

embryological association with the thymus explains why parathyroids may be found in the mediastinum and are occasionally embedded in the thymus gland. Normal parathyroid glands are often difficult to recognise during operations on the thyroid, for they may be camouflaged by a fascial or fatty envelope. Therefore the *identity test* is valuable during a partial thyroidectomy, an every effort must be made to recognise and preserve the parathyroids. The test consists in pinching slightly the doubtful tissue with blunt dissecting forceps. If it is parathyroid tissue, it quickly becomes dark blue or black following this

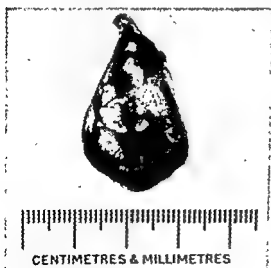


FIG. 485. A parathyroid tumour, a portion of which has become black, the result of a slight contusion. The same phenomenon occurs in the case of a normal parathyroid giving us a means of identity.

and joint symptoms lead to investigations. *Coma* may occur in hyperparathyroidism when the serum calcium exceeds 20 mg. per 100 ml. The skeletal changes result from mobilisation and drainage of calcium and phosphorus from the bones to the blood. *Generalised osteoporosis or decalcification* without cyst formation of bones may be the early and possibly the only change in the skeleton (Fig. 487). In diffuse osteitis fibrosa cystica, there is rarefaction of the cortex of the bones and the marrow is replaced by *spongy fibrous tissue* in which cysts develop. Both changes may be present and either may predominate. *Tumours* resembling osteoclastomas may arise in a phalanx, the jaw or in other bones. They may slowly increase in size and become tender and painful. A pathological fracture is liable to occur through a tumour or at any site of extreme rarefaction. *Deformities* occur due to bending of the softened bones from weight bearing or muscle pull. These occur in the spine, pelvis, thorax and long bones, giving rise to genu valgum, coxa vara, kyphosis and many other deformities. A *pathological fracture* resulting from a trivial injury may be the first evidence pointing to the presence of hyperparathyroidism (Fig. 488).

**Renal Changes.** The hypercalcaemia in hyperparathyroidism results in an increased urinary excretion of calcium and phosphate. These salts may be laid down in the pelvis and calices of the kidney and lead to the formation of calculi. Calcareous deposits in the kidneys may occasionally be present without evidence of skeletal changes. This being so it follows that patients who present with renal symptoms and who prove to have renal calculi should be investigated for hyperparathyroidism. The renal calculi consist of a mixture of calcium oxalate and phosphate. Crystals of the salts may also be deposited in the renal parenchyma with impairment of kidney function. Nephrolithiasis is present in about 25 per cent. of cases, often resulting in renal colic and pyelonephritis which may eventually culminate in renal failure. Men with hyperparathyroidism have renal changes twice as often as women who, however, predominate in the type with the classical skeletal changes. Renal calculi should not be removed until the high serum calcium associated with hyperparathyroidism has been brought to normal level.

**Diagnosis.** Hyperparathyroidism should be considered if a patient presents with evidence of renal calculus, osteoporosis or pathological fracture. Examination of the neck offers little help, for parathyroid tumours are rarely, if ever, palpable. The diagnosis is based on radiological and biochemical investigations. This is important, for the disease may present with both skeletal and renal changes, with one or the other, or even with neither. Radiography gives valuable evidence in a little over 25 per cent. of cases. Positive findings include : (a) Osteoporosis or generalised decalcification of the skeleton ; (b) rarefaction of bones with cyst formation ; (c) mottling of the skull (Fig. 487) ; (d) resorption of terminal phalanges ; (e) absence of dental lamina dura. *Biochemical*



FIG. 486.

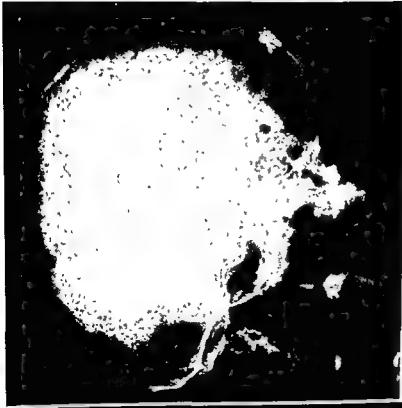


FIG. 487.



FIG. 488.

FIG. 486. Skeletal deformity, asthenia and atrophy of the muscles in hyperparathyroidism.

FIG. 487. Osteoporosis of the skull in generalised osteoporosis. (The bone cysts of this disease are rarely found in the skull.)

FIG. 488. A spontaneous fracture which occurred directly after removal of a parathyroid tumour.

and the posterior mediastinum behind the common carotid arteries are explored from the neck. If there is doubt about any material removed, the decision regarding mediastinotomy (splitting the sternum) must await histological examination. If the surgeon is convinced that there is no enlarged or abnormal parathyroid tissue in the neck, mediastinotomy and further exploration are undertaken. If neither a parathyroid tumour nor abnormal parathyroid tissue are found in the neck or mediastinum, but the patient is definitely suffering from hyperparathyroidism, it is justifiable to remove all but two of the normal parathyroid glands.

**The Operation.** The incision for the exploration of the neck and the further steps to be followed are similar to those of the early stages of partial thyroidectomy as previously described. Search is first directed to the region of the inferior thyroid veins before the under surface of the thyroid lobes is explored. The tumour, a yellowish-grey body, soft, and oval or pyriform, varies in size between 2 and 4 cm. in diameter. It is easily freed from surrounding fascia by blunt dissection. Like all parathyroid tissue, a tumour becomes dark blue or black (contused) at the slightest touch (Fig. 485). The inferior thyroid veins, the upper aspect of the anterior mediastinum behind the clavicles and that part of the posterior mediastinum parallel with the common carotid arteries having been explored, the left lobe of the thyroid is fully exposed. Attention is directed to its posterior border, in particular near the inferior thyroid artery and then along the superior leash of vessels (superior thyroid artery) and posterior wedge of the upper pole. The right lobe is similarly explored. All tumour tissue, or, in its absence, all but two enlarged or hyperplastic parathyroids are removed. If, however, exploration of the neck fails to reveal abnormal parathyroid tissue, the anterior mediastinum must be further explored.

**Mediastinotomy.** Section of the sternum has been described under partial thyroidectomy. With the two halves of the sternum adequately retracted, search is made in the fascia overlying the thymus and pericardium. If a tumour is not identified, the thymus is exposed by blunt dissection and palpated for suggestive lobulated thickening as caused by a parathyroid tumour within its substance. If such a condition of the thymus is found, it is incised and the tumour removed.

**Post-operative biochemical studies** are undertaken daily from the day of operation. It is usual for the high preoperative serum calcium level to fall well below normal after operation, perhaps to 7 to 9 mg. per 100 ml. This low level gradually returns to normal within a few days or weeks, particularly when a high calcium diet is given. This initial fall in the level of serum calcium almost certainly proves the success of the operation, but the final proof must await the histological report on the tumour or tissue removed.

**Results of Operation.** Hyperparathyroidism without treatment is a progressive disease, leading to pain, deformities and ultimately death

*investigations* give the final and important confirmatory evidence. The *normal serum calcium* (9 to 11 mg. per 100 ml.) may be raised as high as 18 to 20 mg. per 100 ml., although 16 mg. is confirmatory evidence. The *normal plasma phosphorus* (3 to 5 mg. per 100 ml.) is reduced to 2 mg. per 100 ml. or under. The *plasma alkaline phosphatase* if raised above 14 units denotes changes in the bones. The *calcium balance test* is most important in proving that the excretion of calcium in the urine is above normal. With an intake diet of 150 mg. of calcium given daily for five days a urinary excretion of 180 to 190 mg. daily is suggestive of hyperparathyroidism. When the excretion is over 200 mg. it is conclusive.

**Differential Diagnosis.** *Multiple Myeloma.*—Multiple punched-out radiotranslucent areas in bones. Pseudoglobulins found in the serum in 90 per cent. of cases; their presence being demonstrated by electrophoresis. Bence-Jones' protein in the urine in 50 per cent. of cases. Myeloma cells in bone marrow shown by sternal puncture. Biopsy of an accessible myeloma, e.g. in a rib. *Osteomalacia.*—Bones poorly calcified due to lack of vitamin D in adults, usually women during pregnancy. Deformities and pathological fractures occur. Evidence of malnutrition. *Thyrotoxicosis.*—There may be some decalcification of bones and an increase in serum calcium. *Cushing's Syndrome.*—Again decalcification of bones may occur and lead to collapse of some of the vertebral bodies. Other evidence of the disease is easy to find (Chap. 72). *Secondary Tumours of Bone.*—Presence of primary growth often demonstrable. *Osteitis Deformans* (Paget's Disease of Bone).—Great thickening and deformity of bone. Characteristic enlargement of the skull. Serum calcium and phosphorus: normal levels. *Osteogenesis Imperfecta* (Fragilitas Ossium).—A rare heredito-familial disorder found in children. Blue sclerotics. Rarefied bones. Multiple fractures. Tends to be self-limiting. Serum calcium and phosphorus: normal levels.

### SURGICAL TREATMENT

When the diagnosis of hyperparathyroidism has been established, operation must not be delayed, for continued excessive calcium loss causes progressive deterioration in the patient's condition. Surgical treatment consists in removal of an adequate amount of parathyroid tissue, whether there be a tumour, hyperplasia or hypertrophy. The condition may be caused by a single tumour, but when one tumour has been removed search is continued for other tumours. If a second tumour is missed the hypercalcaemia continues, and there is very little improvement. If a tumour is not present in the neck or behind the clavicles, the parathyroids are examined for evidence of hypertrophy or hyperplasia. If they are considered to be *enlarged* or *hyperplastic* two or more are removed. If no parathyroid abnormality is discovered in the neck, further search must be directed to the mediastinum (one in four tumours are found in this situation). The anterior mediastinum, the region behind the clavicles

has to be continued for a longer period it is likely that insufficient parathyroid tissue remains to maintain an adequate calcium balance, but, nevertheless, the tetany is usually easily controlled, and the need for calcium therapy gradually diminishes, presumably as hypertrophy of remaining parathyroid tissue occurs. Sometimes in mild hypoparathyroidism the blood calcium falls to *tetany level* only during menstruation, pregnancy and lactation (*latent tetany*). *Chronic tetany* may follow a severe acute phase in the very rare instance when most or all of the parathyroid tissue has been removed. The paresthesiæ and spasms are severe and incapacitating, and need intensive therapy to keep them under control. Parathyroid tetany usually occurs on the first or second post-operative day, and consists of paresthesiæ in the extremities followed by stiffness of the fingers and toes. These premonitory signs are followed by the classical carpopedal spasms; the hands stiffen into the so-called obstetric position in which the digits are straight and the tips of fingers and thumb are pressed tightly together. The toes are plantar flexed and adducted. Painful muscle spasms arise elsewhere, especially in the extensors of the knees and flexors of the elbows. *Chvostek's sign* or twitching of the facial muscles when the facial nerve is tapped in front of the ear denotes increased neuromuscular excitability. *Trousseau's sign* is the production of carpal spasm by firm pressure and occlusion of the arterial circulation above the elbow.

**Treatment of Tetany.** Parathormone replacement therapy is unsatisfactory in acute tetany, as several hours elapse before any response is obtained, even if given intravenously. Neither is it indicated for chronic tetany as its effect tends to wear off and it may give rise to decalcification of bone. Calcium salts raise the serum calcium level and quickly abolish tetany. Thus mild tetany may be satisfactorily treated by giving 4 gm. of calcium lactate by mouth three or four times daily, and severe tetany quickly responds to a slow intravenous injection of 20 ml. of a 10 per cent. solution of calcium gluconate. The effect is rapid and lasts several hours. Large doses of calcium are then given by mouth, although further injections of calcium may be necessary if there is a recurrence of spasms. *Calciferol* (vitamin D) 100,000 to 500,000 I.U. or the related steroid substance A.T. 10 (3 to 10 ml.) is given daily by mouth until the blood calcium rises to a satisfactory level; at such time the daily dose is replaced by a weekly maintenance one. It may be necessary to increase the dosage above the maintenance level during periods of excessive calcium loss such as menstruation, pregnancy or lactation. *Diet.*—A comparatively normal diet, though low in phosphorus, is recommended.



from pyelonephritis and uræmia. After operation, the symptoms soon begin to disappear. Recalcification of the skeleton gradually takes place, and ultimately the bones become as dense as normal. Although there is no further danger of pathological fractures, any deformities already present remain. Kidney function does not return to normal if renal damage is present, or if there are widespread deposits of calcium in the renal parenchyma. Soft plaque-like renal calculi may disintegrate, but surgical removal is required for the great majority of renal calculi.

### HYPOPARATHYROIDISM

#### (Parathyroid Tetany)

*Hypoparathyroidism* is most commonly caused by removal or injury of the parathyroid bodies during operations on the thyroid gland, or it may follow operations for hyperparathyroidism. Rarely it arises spontaneously due to atrophy of the parathyroids. The *clinical manifestation* is tetany. There is a fall in the level of serum calcium and a rise in the plasma phosphorus with diminished calcium excretion in the urine. The serum calcium (normal = 9 to 11 mg. per 100 ml.) is diminished, and may be as low as 5 mg. per 100 ml. or even lower. The plasma phosphorus (normal = 3 to 5 mg. per 100 ml.) is raised to 6 mg. per 100 ml. or more. Tetany following operations on the thyroid gland is more likely after total or near-total thyroidectomy. During such operations there is a greater likelihood of inadvertently removing the parathyroids, injuring them or interfering with their blood supply. The danger is also greater in operations for recurrent goitre, for the anatomy may then be abnormal; fibrous tissue has been laid down, and some of the parathyroids may have been removed or their blood supply interfered with at the original operation. During operations on the thyroid gland, care must therefore be taken to recognise and safeguard the parathyroid bodies. If a parathyroid gland is inadvertently removed it is immediately implanted into the sternomastoid muscle although there is no proof that such implants survive. The *parathyroid identity test* (Fig. 485) mentioned earlier is based on the inherent liability of parathyroid tissue to bruise easily at the slightest touch. Therefore tissue reacting in this way must be conserved.

*Transient tetany* is of little consequence, and occurs after about 1 per cent. of thyroid operations. It manifests itself the day following operation; the patient complains of tingling of the fingers and toes, often associated with a positive Chvostek's sign. This transient tetany may be caused simply by trauma or interference with the parathyroid blood supply. It responds quickly to calcium by mouth. Tetany may still be transient, even if some of the parathyroid bodies have been removed, for it seems that the calcium balance can be maintained with only two or perhaps only one of them. In transient tetany the symptoms do not return when calcium therapy is withdrawn after four or five days. When treatment

production begins and the sex of the patient. If the disorder begins in embryonic life *pseudohermaphroditism* develops in the female and *macrogenitosomia praecox* in the male. If it begins in infancy or childhood it results in abnormal physical and sexual development in either sex. "Little girls become little boys and little boys become little men" (Fig. 489). Excessive production of androgens in women causes hirsuteness of the face and body in the male distribution, amenorrhoea, regression of breasts, acne, baldness, and deepening of the voice (Figs. 490, 491 and 492). Although the advanced disease is easily diagnosed, a mild syndrome needs laboratory tests for differentiation from the commonplace fat hairy woman with normally functioning adrenals. In men there is over-masculinisation.

**Cushing's Syndrome.** This is caused primarily by excessive production of *glucocorticoids* (hydrocortisone and cortisone), affecting carbohydrate and protein metabolism. The underlying lesion may be hyperplasia, an adenoma or carcinoma of the adrenal cortex. An adenoma or carcinoma of an adrenal gland inhibits ACTH formation in the pituitary gland and thus causes atrophy of the opposite adrenal, and it has been found histologically that there is hyalinisation of the basophil cells of the pituitary gland. Cushing's syndrome occurs five times more often in women than in men. There is obesity, with fat distribution most marked in the supraclavicular regions, on the face ("moonface") and at the back of the neck ("buffalo hump"), but with sparing of the extremities; wasting and weakness of muscles; fine paper-like skin, with purple striae over the abdomen and thighs, and with multiple ecchymoses and bruising; osteoporosis, resulting in dorsal kyphosis, often with compression fractures of vertebrae; diabetes mellitus; hypertension; and psychological instability (e.g. acute depression, irritability, or aggressiveness). When the diagnosis has been made, it still remains to establish the cause, i.e. whether due to hyperplasia or a tumour of the adrenal cortex, or to pituitary dysfunction or a pituitary adenoma (*rare*). The classical adrenogenital and Cushing's syndromes have just been described, but it should be appreciated that there is a gradation in the clinical picture from



FIG. 489. Adrenogenital syndrome (virilism) in a boy aged five years, resulting in excessive physical and sexual development (height 5 feet).

## CHAPTER 72

### THE ADRENAL GLANDS

EACH adrenal (suprarenal) gland is supplied by a main suprarenal artery, a direct branch of the abdominal aorta ; other smaller suprarenal arteries arise from the renal and phrenic arteries. Blood from the adrenals empties directly into the inferior vena cava on the right side and into the renal vein on the left.

The adrenal medulla manufactures adrenaline and noradrenaline. The adrenal cortex produces :—(a) An androgenic hormone, closely similar to testosterone, in both males and females ; (b) hydrocortisone and cortisone which influence carbohydrate and protein metabolism—these steroids are also known as glucocorticoids ; (c) aldosterone which influences sodium and potassium metabolism.

A pituitary hormone, corticotrophin (adrenocorticotrophic hormone, ACTH) controls adrenocortical activity. It follows that hypersecretion of corticotrophin (e.g. as with the rare basophilic pituitary adenoma) may cause adrenocortical hyperplasia, although the hyperplasia may arise independently of the pituitary. Conversely, inhibition of corticotrophin by administration of cortisone results in atrophy of the adrenal cortex. Over-production of adrenocortical hormones may be caused by a cortical tumour, either benign or malignant.

Estimation of the 17-ketosteroids excreted in the urine in twenty-four hours is a valuable test of androgenic steroid excretion. The normal amount excreted by adult males is 10 to 20 mg. in twenty-four hours, and from 5 to 15 mg. in females. A reflection of glucocorticoid secretion is the estimation of either the 17-ketogenic steroids (normal = 4.0 to 10.2 mg.) or of the 17-hydroxycorticoids (normal = 1.1 to 12.0 mg.) excreted in the urine in twenty-four hours. There is at present no reliable simple method of estimating the amount of aldosterone in the blood or urine.

#### ADRENOCORTICAL SYNDROMES

The type of syndrome caused by hypersecretion of adrenocortical hormones depends on which hormone is most affected. In hyperplasia and in benign tumours there is usually overactivity of one type of hormone. Occasionally in hyperplasia, but more often in malignant tumours there is a mixed hormone overactivity.

**The Adrenogenital Syndrome.** This results from oversecretion of androgenic hormones caused by hyperplasia or a tumour of the adrenal cortex. The clinical picture depends on the age when the excessive

production begins and the sex of the patient. If the disorder begins in embryonic life *pseudohermaphroditism* develops in the female and *macro-genitosomia praecox* in the male. If it begins in infancy or childhood it results in abnormal physical and sexual development in either sex. "Little girls become little boys and little boys become little men" (Fig. 489). Excessive production of androgens in women causes hirsuteness of the face and body in the male distribution, amenorrhoea, regression of breasts, acne, baldness, and deepening of the voice (Figs. 490, 491 and 492). Although the advanced disease is easily diagnosed, a mild syndrome needs laboratory tests for differentiation from the commonplace fat hairy woman with normally functioning adrenals. In men there is over-masculinisation.

**Cushing's Syndrome.** This is caused primarily by excessive production of *glucocorticoids* (hydrocortisone and cortisone), affecting carbohydrate and protein metabolism. The underlying lesion may be hyperplasia, an adenoma or carcinoma of the adrenal cortex. An adenoma or carcinoma of an adrenal gland inhibits ACTH formation in the pituitary gland and thus causes atrophy of the opposite adrenal, and it has been found histologically that there is hyalinisation of the basophil cells of the pituitary gland. Cushing's syndrome occurs five times more often in women than in men. There is obesity, with fat distribution most marked in the supraclavicular regions, on the face ("moonface") and at the back of the neck ("buffalo hump"), but with sparing of the extremities; wasting and weakness of muscles; fine paper-like skin, with purple striae over the abdomen and thighs, and with multiple ecchymoses and bruising; osteoporosis, resulting in dorsal kyphosis, often with compression fractures of vertebrae; diabetes mellitus; hypertension; and psychological instability (e.g. acute depression, irritability, or aggressiveness). When the diagnosis has been made, it still remains to establish the cause, i.e. whether due to hyperplasia or a tumour of the adrenal cortex, or to pituitary dysfunction or a pituitary adenoma (*rare*). The classical adrenogenital and Cushing's syndromes have just been described, but it should be appreciated that there is a gradation in the clinical picture from



FIG. 489. Adrenogenital syndrome (virilism) in a boy aged five years, resulting in excessive physical and sexual development (height 5 feet).



FIG. 490. Adrenogenital syndrome. An advanced stage in an adult female. (*Dr. Raymond Greene's case.*)

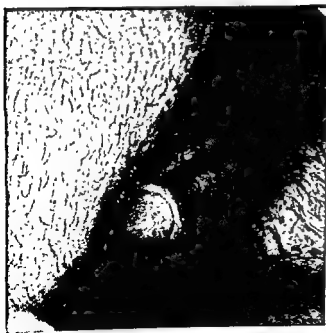


FIG. 491. Hypertrophy of the clitoris in the patient shown in Fig. 490.

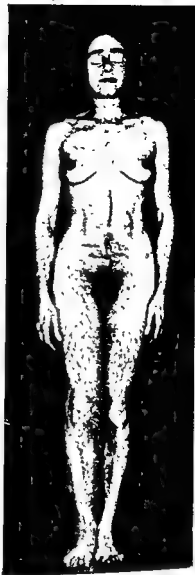


FIG. 492. Adrenogenital syndrome (a lesser degree of virilism), showing overgrowth and masculine distribution of hair.

the typical adrenogenital syndrome at one extreme to the typical Cushing's syndrome at the other.

**Aldosteronism.** This is due to increased production of aldosterone. *Primary aldosteronism*, Conn's disease, is usually produced by an adenoma of the adrenal cortex. Clinically, the disorder is characterised by mild hypertension and attacks of *hypokalaemic paresis* or *paralysis*. Secondary or compensatory aldosteronism is caused by sudden reduction of blood volume (e.g. in surgical shock), or in clinical disorders in which there is severe oedema (e.g. congestive heart failure).

**Investigations.** *Urinary Steroids.*—A rise in the urinary excretion of 17-hydroxycorticoids (oxysteroids) is very suggestive of Cushing's syndrome, whereas a rise in the 17-ketosteroids (near normal in Cushing's syndrome) is indicative of the adrenogenital syndrome. (Note.—There is an increased 17-ketosteroid excretion in the very rare masculinising tumour of the ovary, the arrhenoblastoma.) A rise in urinary aldosterone occurs in aldosteronism, but its assay is difficult and tedious. *Blood examination* may reveal a low eosinophil count in increased glucocorticoid formation. *Radiography*, especially tomography, may reveal an adrenal tumour above one of the kidneys. An *intravenous pyelogram* may show renal depression caused by an adrenal tumour. X-rays of the skeleton may show osteoporosis, and resultant compression of vertebral bodies and fractures of ribs. *Extraperitoneal pneumography* (perirenal insufflation) is valuable in outlining an adrenal tumour or hypertrophy. A needle is either passed between the rectum and coccyx or directly into the perirenal fascia and oxygen is injected into the extraperitoneal space, thereby defining the kidneys and adrenals. The value of the procedure is appreciably enhanced when used in conjunction with intravenous pyelography (Fig. 493). It is, however, important to realise that adrenocortical overactivity may be present without enlargement of either adrenal gland.

## TREATMENT

**Cushing's Syndrome.** *Adrenalectomy* is the treatment of choice in cases of adrenal tumour and hyperplasia (Figs. 494 and 495). The operative procedure is urgently indicated when the condition is rapidly progressive and accompanied by diabetes and bone changes. The prognosis is good for both hyperplasia and adenoma, but poor for carcinoma which gives rise to early secondary deposits in the liver and lungs. *Hypophysectomy* or irradiation of the pituitary gland is indicated in Cushing's syndrome when it is activated by oversecretion of the pituitary or by a pituitary adenoma.

**The Adrenogenital Syndrome.** In adults, as exemplified by virilism in females, this disorder is more commonly caused by adrenal hyperplasia than by a tumour. The latter is typically soft and yellow, and is potentially malignant and inclined to be invasive. *Adrenalectomy* is

indicated in those cases with tumours, and in the mixed type of adrenocortical overactivity due to hyperplasia in which Cushing phenomena predominate. *Suppression* therapy with daily cortisone acetate is indicated in the presence of adrenal hyperplasia when the *androgenic phenomena* (*virilism*) predominate. The results of medical therapy are good in the pure form of the adrenogenital syndrome, particularly when it occurs in children. In children, the adrenogenital picture is very similar to that in adults except that the cause is far more often an adenoma



FIG. 493. Extraperitoneal pneumography (perirenal insufflation) showing the outline of a normal adrenal on the right (reader's left) and of an adrenal tumour on the left (reader's right).

or carcinoma of the adrenal. Treatment is the same as in adults. Pseudohermaphroditism in the female and macrogenitosomia in the male (the result of adrenocortical oversecretion *in utero*) are treated by suppression therapy (cortisone).

**Primary Aldosteronism.** This condition is treated by removal of the adrenal which contains the adenoma.

**Adrenalectomy.** The lumbar incision is preferred. The approach is through the bed of the twelfth rib on the side of the suspected tumour. In Cushing's disease or the adrenogenital syndrome, if the first adrenal gland explored contains a tumour, the adrenal on the opposite side need not be explored, for it is almost certainly atrophic. Conversely, if the

first adrenal explored is atrophic, the opposite side must be explored for a tumour. If the first adrenal is large, its fellow will also be large; this necessitates removal of the first followed by total or subtotal (nine-tenths) removal of the other adrenal. If the first adrenal explored is normal in size it is likely that its fellow is also normal, although both are hyper-secreting. If the disorder is advanced, both adrenals are removed,



FIG. 494. Cushing's syndrome. Patient before adrenalectomy.



FIG. 495. The same patient as in Fig. 494 three months after bilateral adrenalectomy.

whereas if less severe, it is justifiable to leave behind one-tenth of one adrenal. Total adrenalectomy is preferable in the advanced Cushing's syndrome as:—(1) Regrowth of the remnant might lead to a recurrence of the condition; and (2) it is easier and simpler to control patients by complete substitution hormone therapy than to attempt to supplement the varying activity of an adrenal remnant.

**Management of Adrenalectomy.** Cortisone and hydrocortisone have done much in combating shock and making surgery safer. Two hundred milligrams of cortisone, in divided doses, are given each day for two days before operation and 10 mg. of DOCA the evening before and morning of



operation. The above daily dose of cortisone is continued until the blood pressure is stable for twenty-four hours, and is then lessened by 25 mg. daily until a maintenance dose of 25 to 50 mg. per day is reached. Up to 3 gm. of sodium chloride, daily by mouth, may also be required. During operation an intravenous drip of 5 per cent. dextrose in saline is given and hydrocortisone is added if necessary; intravenous noradrenaline is used to counteract any sudden fall in blood pressure. When an adrenal tumour is removed, the opposite adrenal is likely to be atrophic. In these circumstances, substitution therapy is essential to avoid acute adrenocortical failure.

*Results.* The changes following adrenalectomy are very satisfactory; the signs and symptoms of the diseases disappear quickly.

### TUMOURS OF THE ADRENAL MEDULLA

(1) *Chromaffin tissue tumour* (phaeochromocytoma) of the adrenal medulla is commonly accompanied by an excess production of pressor substances and is characterised by hypertension.

(2) *Sympathetic nervous tissue tumours* are discussed under The Autonomic Nervous System, Chap. 60.

**The Pheochromocytoma.** This chromaffin tissue tumour is usually benign and most often unilateral, but may be bilateral. It arises in males or females, usually between the ages of twenty and forty. The tumour is usually small and impalpable although it may very rarely present as an abdominal tumour. It is lobulated and encapsulated, yellowish-brown on section and often shows signs of hæmorrhage and of cystic degeneration. The symptoms associated with a pheochromocytoma are due to release of adrenaline and noradrenaline into the blood stream. The more common syndrome is for the patient to have sustained hypertension, due to continuous release of pressor substances. Paroxysms of increased blood pressure may then occur, and the systolic pressure may rapidly rise to as much as 300 mm. of mercury. This rise may last for a few minutes to several hours, and may not recur for several weeks. As the condition increases in severity, the attacks become more frequent, and are readily set off by fear, physical and mental strain, or other factors normally exciting production of noradrenaline. The paroxysms are characteristically accompanied by anxiety, headaches, sweating, palpitation, anginal pain, nausea and vomiting. In the less common syndrome, the patient has a normal blood pressure between attacks.

**Investigations.** Various tests are undertaken to prove the presence of, or otherwise, of a pheochromocytoma. Histamine phosphate injected intravenously causes a fall in blood pressure in normal people, and a rise in pressure, although not invariably so, in patients with a pheochromocytoma. A false positive result is also possible. Phentolamine (Rogitine) may be administered as a test, for it lowers the blood pressure created by adrenaline. Pressor substances are increased in the urine in the presence

of a pheochromocytoma and can be assayed. *Radiography* before and after perirenal insufflation of oxygen is valuable and is further aided by tomography and intravenous pycnography [blood pressure lowering agents must be readily available as perirenal insufflation, by creating pressure on the tumour, may produce a hypertensive attack].

**Treatment.** Removal of the tumour is carried out. A rapid rise of blood pressure may occur during the induction of the anæsthetic, or later, when the tumour is being handled; such a rise of blood pressure must be guarded against by the administration of a slow intravenous infusion of 5 per cent. dextrose, by means of which 2 to 10 mg. of Rogitine could be quickly given should the blood pressure rise above 150 mm. of mercury. A rapid fall of blood pressure, shock and collapse may occur immediately after removal of the tumour. This is controlled by means of intravenous noradrenaline.

As pheochromocytomas may arise:—(a) In one or both adrenals; (b) outside yet close to an adrenal; (c) along the sympathetic chain; or (d) on the aorta, it is essential for all these areas to be explored. Such a wide exploration can best be undertaken through a transverse upper abdominal incision. Both recti are divided. The left adrenal is first explored by palpation and if a tumour is felt or suspected, the posterior part of the peritoneum is exposed above the kidney and in front of the adrenal while the spleen is gently retracted downwards. The peritoneum is then incised over the tumour which is removed by blunt dissection. The right adrenal region is next exposed by retracting the liver upwards, the kidney downwards and the intestines medially. A tumour, if present, is dissected free and removed after incising the peritoneum over it. The abdominal cavity is next explored with particular attention to the sympathetic ganglia surrounding the aorta. After operation, the symptoms are relieved and the blood pressure returns to normal, or near normal, in more than half the cases.

#### ADRENALECTOMY FOR METASTATIC OR INOPERABLE CANCER OF THE BREAST

Advanced cancer of the breast is classified into that which is to a great extent dependent on the production of hormones for its growth (hormone-dependent) and that which is not (non-hormone-dependent). There are at present no means of determining before operation which tumours are hormone-dependent. The *hormone-dependent* type of growth is the one present in nearly half those *premenopausal* women with metastatic breast cancer. This is shown by regression of the growth following oöphorectomy in this proportion of cases. *Total adrenalectomy* is indicated in those patients with metastasis from cancer of the breast, whose hormone-dependent tumours have become reactivated after a temporary remission produced by oöphorectomy. Adrenalectomy is of no value in those patients who fail to respond to oöphorectomy (non-hormone-dependent).

Combined oöphorectomy and adrenalectomy is recommended for post-menopausal patients with advanced breast cancer. These cases do not respond to oöphorectomy alone.

These procedures are indicated as palliative treatment; they give marked relief from the crippling pain of secondary deposits in the bones, and there is temporary regression of secondary deposits in the skin and elsewhere.

*Hypophysectomy* has given benefit to patients with advanced cancer of the breast whose symptoms have relapsed after relief from oöphorectomy and adrenalectomy. It appears, therefore, that some breast cancer is dependent for growth on pituitary hormones and that the therapy of the future will be more and more directed to ablation of the pituitary gland by means of surgical extirpation or radioactive implants.

**Pituitary Tumours** (see section on *Neurosurgery*, Chap. 53).

## SECTION IX

### CHAPTER 73

### THE BREAST

by

BASIL H. PAGE

THE essential components of the breast are the mammary disc and the nipple. The mammary disc consists of a fibrous stroma in which lies the glandular tissue consisting of alveoli arranged in clusters (forming the lobules of the breast) and opening into ducts. The smallest ducts unite to form larger ducts, and these in turn unite with other ducts as they all converge on the centre of the mammary disc until from fifteen to twenty main ducts are formed. As these approach the nipple they expand into ampullæ, but become narrow again as they run through the nipple and emerge on the skin. The fibrous tissue which comprises the stroma of the breast is very tough and dense, but nevertheless supple. A variable amount of fat is interspersed through it. The skin immediately surrounding the nipple (the areola) is pigmented, contains numerous sebaceous glands and lies directly on the mammary disc without the intervention of any subcutaneous fat. The mammary disc is covered by a layer of fascia from which fine septa pass to the skin (the suspensory ligaments of Astley Cooper). The shape and size of the breast depend partly on the bulk of the mammary disc, but mainly on the amount, distribution and consistency of the subcutaneous fat which varies greatly between one individual and another. There is very little fat behind the deep surface of the mammary disc.

The arterial supply of the breast is derived mainly from the axillary artery superolaterally and the internal mammary artery medially. The intercostal arteries make a small contribution. The veins follow the general lines of the arteries, but a special feature, often conspicuous to the surgeon, is the anastomotic plexus of veins on the surface of the mammary disc.

The lymph drainage (Fig. 496) is one of the most important of all the anatomical features because of the mortal part it plays in the spread of cancer. There are two main groups of glands into which the lymph drains directly from the breast, the axillary and the internal mammary. The axillary glands are the more numerous; the internal mammary glands, which drain mainly the medial part of the breast, lie behind the sternum

Combined oöphorectomy and adrenalectomy is recommended for post-menopausal patients with advanced breast cancer. These cases do not respond to oöphorectomy alone.

These procedures are indicated as palliative treatment; they give marked relief from the crippling pain of secondary deposits in the bones, and there is temporary regression of secondary deposits in the skin and elsewhere.

Hypophysectomy has given benefit to patients with advanced cancer of the breast whose symptoms have relapsed after relief from oöphorectomy and adrenalectomy. It appears, therefore, that some breast cancer is dependent for growth on pituitary hormones and that the therapy of the future will be more and more directed to ablation of the pituitary gland by means of surgical extirpation or radioactive implants.

**Pituitary Tumours** (see section on *Neurosurgery*, Chap. 53).

chest wall, whereas with the patient erect the breast hangs down with the mammary disc folded on itself, presenting to the fingers a double thickness which confuses perception and complicates interpretation. With the patient lying, the examiner's fingers systematically feel every part of both breasts, beginning with the normal breast for comparison, gently compressing the single thickness of the mammary disc against the chest wall. The palmar surface of the terminal phalanges should be used, for this is the most sensitive organ of palpation. The flat of the hand is a relatively crude instrument. In palpating the breast, the main objective is usually the search for a lump. This is difficult because the normal breast is composed of gland tissue in a fibrous stroma, and is therefore nodular. Sometimes the nodules are so soft and fine that they are hardly perceptible; sometimes, even in a normal breast, they are large and firm and very obvious, but they are diffusely scattered throughout the breast and of uniform consistency. The more nodular the breast the more difficult is the search, and only experience will bring assurance in answering the question "Is there a lump in this breast?" i.e. a lump which is not due to a variation of normal nodularity. Because there is no subcutaneous fat under the areola the mammary disc can be felt here much more clearly than elsewhere, and the normal nodularity is especially obvious. Once a lump has been detected in the breast, its exact nature must be established beyond any possibility of doubt, and this usually means histology.

The lymph glands of the axillæ and supraclavicular fossæ are always examined. The axillary glands are most easily felt with the palmar aspect of the examiner's fingers directed medially. The patient's arm should be slightly abducted at the shoulder with all the muscles relaxed. The examiner's left hand is used for the right axilla and the right hand for the left axilla. The liver is always palpated if carcinoma is suspected.

*Supernumerary nipples*, usually rudimentary, are common, and may be situated anywhere along the embryonic "milk line" which runs downwards and slightly medially from the axilla over the chest, abdomen and groin to the medial aspect of the upper part of the thigh. They may occasionally require removal for cosmetic reasons. *Ectopia of breast tissue* occurs less frequently. It also is found along the "milk line" and may or may not be accompanied by an ectopic nipple. Its chief importance is that it may give rise to a painful swelling particularly if lactation occurs. It may rarely be the site of carcinoma. Ectopic breast tissue should be excised if it causes symptoms.

The natural form of the nipple varies greatly. It may be large and prominent, or flat, or even indrawn. It is important to distinguish clearly between lifelong abnormalities and recently acquired changes, for a change in the form of the nipple is usually the result of carcinoma. The female breast enlarges at puberty due to increase in the size of the mammary disc (including growth of the glandular elements) and deposition of

and the costal cartilages. The inferior deep cervical glands (the "supra-clavicular" glands) do not usually drain lymph directly from the breast except from the subcutaneous tissues of its upper part, but they receive some efferents from the axillary glands. The medial inferior deep cervical group adjoin the uppermost internal mammary glands behind the sternoclavicular joint.

The lymph vessels in the mammary disc drain at first towards its anterior surface, mainly converging on the nipple, but some run directly to the surface of the breast more peripherally. On the surface of the breast there is a subcutaneous plexus of lymphatic vessels especially well

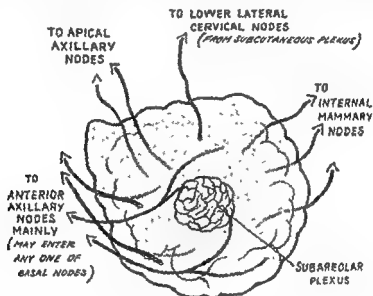


FIG. 406. The routes of lymphatic drainage from the breast. (From "The Breast," by F. D. Sauer, John Wright & Son Ltd.)

developed under the areola. In the subcutaneous plexus the lymph drains over the surface of the breast to the plexus lying on the surface of the pectoral fascia and thence to the axillary or internal mammary glands.

**Examination of the Breast.** The patient should be stripped to the waist so that both breasts can be compared, and the neck, axillae and upper abdomen are all accessible. A good light is essential. For most of the examination the supine position is best, but additional information may be obtained with the patient sitting upright. The special points to be noted on inspecting are the symmetry of the breasts, any visible lump and the condition of the nipple. With the patient sitting upright, the relative height of the nipples and the mobility of the breasts can be observed. By comparing the breasts as they assume the pendant position, unequal changes of contour may be revealed. The great advantage of the supine position for palpation is that the breast spreads over the chest so that the mammary disc can be felt as a single thickness against the

the early stages of the nipple affection, there is no evidence of a tumour of the breast. The disease of the nipple has certain characteristic features both clinical and histological. Clinically there is at first a slight serous discharge with a tendency to incrustation. A raw surface develops and the nipple is slowly destroyed. Histologically, the typical feature is the presence of large round cells with vacuolated cytoplasm in the epidermis. The relationship between the disease of the nipple and the subsequent tumour of the breast is not understood. It may be that the eczematous process spreads up the ducts into the breast substance and there sets up a glandular carcinoma. A precisely similar condition sometimes affects the

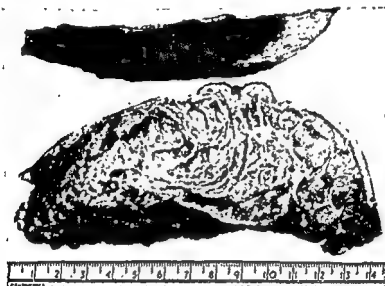


FIG. 497. Paget's disease of nipple and carcinoma of breast. The carcinoma lies deep in the breast above the 6 cm. mark in the scale.

skin of the glans penis and may be associated with a carcinoma of the urethra.

The diagnosis of Paget's disease presents difficulties. In the early stages it has to be distinguished from simple eczema. Absence of destruction of the nipple, a short history and response to treatment are all points in favour of simple eczema. Paget's disease is a chronic condition the life of which is measured in months or even years, and it does not respond to treatment, but is steadily progressive. If there is any doubt, biopsy must be done. A different problem is encountered when there is an obvious tumour in the breast. The difficulty in these cases is to know whether the affection of the nipple is not simply due to secondary involvement by the underlying carcinoma. The history may help. In practice the distinction is not important because at this stage the only thing that matters is the treatment of the tumour of the breast. The treatment of



subcutaneous fat. This development is due to hormonal stimuli. The most important secretions are those of the ovaries (especially oestrogens), but other sources contribute including the adrenal, the pituitary and the thyroid. The size of the breast varies greatly and, in spite of the importance of hormonal factors in bringing about pubertal development, most women whose breasts are abnormally large or small are not suffering from recognisable endocrine disorder. In some cases, unduly small breasts (*hypomastia*) are due to hypogonadism and respond to treatment with oestrogens. Overgrowth of the breasts (*macromastia*) is very seldom due to endocrine disease. When large breasts are unsightly, or cause discomfort by their weight, some relief may be obtained by means of an efficient brassiere, but in some cases reduction of the size of the breast by plastic surgery may be justifiable.

**Affections of the Nipple.** The chief importance of flat or indrawn nipples lies in the interference they may cause with suckling, and in the differential diagnosis of conditions in which the nipple may be affected secondarily, particularly carcinoma of the breast. The nipple is incidentally subject to any of the diseases which may affect skin, particularly rugose skin; warts (papillomas) and eczema are common. The nipple is one of the classical sites of the rare extra-genital syphilitic chancre.

**Discharge from the Nipple.** This is a fairly common complaint. It may be clear, like serum, or turbid, and its colour may be yellow, green, brown, red or almost black. The red and brown colours are due to blood; the green may be due to infection with *Pseudomonas pyocyanea*. The discharge is usually unilateral and emerges from one orifice, or the same few orifices on every occasion in the same patient. Pressure on the part of the areola corresponding with the duct orifice may reproduce the discharge. The symptom is most common in middle-aged women. Occasionally the cause is an underlying carcinoma of the breast, and this is always the most important possibility. Most often no clinical evidence of localised disease is found. Sometimes an *intraduct papilloma* (papilliferous adenoma) is the cause.

In the management of discharge from the nipple, the first thing is to find out if there is a lump in the breast. If there is no lump, surgical treatment is not indicated, and if these cases are kept under periodic observation it is usual for no sign of disease to develop even though the discharge continues. If a lump is found in the breast, operation is absolutely indicated. Exploration may reveal an *intraduct papilloma* or an area of fibro-adenosis with cysts. Sometimes it is possible to introduce a cannula into the duct and inject contrast medium for radiography; an *intraductal papilloma* produces a filling defect.

**Page's Disease of the Nipple.** This is a rare but important and interesting condition. The essential clinical facts are that a peculiar eczematous condition of the nipple is followed, sometimes after the lapse of years, by the development in the breast of a carcinomatous tumour (Fig. 497). In

the early stages of the nipple affection, there is no evidence of a tumour of the breast. The disease of the nipple has certain characteristic features both clinical and histological. Clinically there is at first a slight serous discharge with a tendency to incrustation. A raw surface develops and the nipple is slowly destroyed. Histologically, the typical feature is the presence of large round cells with vacuolated cytoplasm in the epidermis. The relationship between the disease of the nipple and the subsequent tumour of the breast is not understood. It may be that the eczematous process spreads up the ducts into the breast substance and there sets up a glandular carcinoma. A precisely similar condition sometimes affects the



FIG. 497. Paget's disease of nipple and carcinoma of breast. The carcinoma lies deep in the breast above the 6 cm. mark in the scale.

skin of the glans penis and may be associated with a carcinoma of the urethra.

The diagnosis of Paget's disease presents difficulties. In the early stages it has to be distinguished from simple eczema. Absence of destruction of the nipple, a short history and response to treatment are all points in favour of simple eczema. Paget's disease is a chronic condition the life of which is measured in months or even years, and it does not respond to treatment, but is steadily progressive. If there is any doubt, biopsy must be done. A different problem is encountered when there is an obvious tumour in the breast. The difficulty in these cases is to know whether the affection of the nipple is not simply due to secondary involvement by the underlying carcinoma. The history may help. In practice the distinction is not important because at this stage the only thing that matters is the treatment of the tumour of the breast. The treatment of

Paget's disease before a lump is palpable is radical mastectomy. When a tumour has appeared, its stage of development decides treatment.

### INFECTIVE CONDITIONS OF THE BREAST

The skin and subcutaneous tissue of the breast are subject to all the diseases which may affect these tissues anywhere else. The large sebaceous glands of the areola are particularly liable to infection and may cause *areolar abscesses*. The history, physical signs and course of this condition usually make the diagnosis obvious. The treatment is incision and drainage.

**Puerperal Mastitis.** A few days after delivery when lactation is commencing, both breasts become engorged, slightly painful and tender. There may be slight fever. This is not an infective condition, and subsides spontaneously if normal suckling is possible. Infective mastitis develops in the second week or later and usually affects only one of the breasts which becomes swollen and painful with redness and oedema of the skin. There is considerable fever, usually 102° F. (39° C.) or more. In some of these patients there has been difficulty with suckling so that the breast has not been emptied regularly; the nipple may have become cracked. The infective organism is usually the *Staphylococcus aureus* (pyogenes) and the route of infection may be up the ducts from the nipple or by the blood stream. Pain makes suckling difficult, and this leads to further swelling of the breast and aggravates the infection. If effective treatment is not given, suppuration occurs and spreads through the breast. Abscesses may form. Sometimes there is septicæmia or pyæmia. The diagnosis is usually straightforward because of the characteristic circumstances. Early engorgement or "milk fever" can usually be distinguished. The diagnosis of *mastitis carcinomatosa* does not arise until anti-infective treatment has failed. The first essential in treatment is the prompt administration of antibiotics. Penicillin (250,000 units intramuscularly six-hourly) has hitherto been the standard agent, but the incidence of infection in hospital with penicillin-resistant organisms is becoming so high that other antibiotics must often be used. A good deal can be done to prevent mastitis by attention to the nipples and by efficient management of suckling in the early stages of lactation. Once mastitis has developed, suckling should be continued if pain and the general condition of the patient allow it or at least the breast should be regularly emptied by pump or manual expression. Between feeds the breast should be firmly bound. If breast feeding has to be abandoned, lactation should be promptly suppressed by administration of oestrogens.

**Breast Abscess.** In the great majority of cases breast abscess follows acute puerperal mastitis. If mastitis fails to resolve, an abscess forms in the stroma of the mammary disc. This tough tissue resists dissolution, and the suppurative process cannot clear space for a single abscess cavity, consequently the pus lies in several communicating loculi. The toughness

of the breast tissue also delays the pointing of the abscess on the surface, and the diagnosis should therefore usually be made before fluctuation has developed. The breast is enlarged, oedematous, red, indurated and tender. These changes are concentrated at one point, where there may be an area of softening or even fluctuation. There is usually fever, even high fever (over  $103^{\circ}$  F. or  $39^{\circ}$  C.), but sometimes even a large abscess may not cause fever, and this is particularly liable to happen if the patient has received antibiotics.

In the treatment of breast abscess, evacuation of the pus is essential. To try chemotherapy alone is useless and harmful. An ample radial incision is made, laying the abscess wide open. A finger is introduced and the septa between the loculi gently broken down. A drain is inserted and the breast firmly bound. Dressings should be infrequent; the first one is done between the second and the fourth day. An alternative method of treatment is to aspirate the pus with a wide-bore needle and replace the pus with a solution of penicillin. Successful cases have been reported, but the method is painful and unreliable. Once an abscess has formed it is usually necessary to discontinue suckling and suppress lactation, not only on the mother's account, but in order to avoid infecting the infant.

In the differential diagnosis of breast abscess there are only two common problems. The first occurs when there is no doubt about the diagnosis of mastitis, but when it is difficult to know if an abscess has formed. The physical signs alone may decide the issue, especially when the inflammation is concentrated in a small area. Sometimes the duration of the symptoms makes the diagnosis clear, for mastitis usually resolves rapidly or leads to abscess formation, so that persistent signs of mastitis usually signify the presence of an abscess. The other problem occurs in those cases in which diffuse signs are accompanied by much induration and yet there is little tenderness and perhaps no fever. Such evidence may suggest *mastitis carcinomatosa*, the exceedingly swift and malignant form of carcinoma which very occasionally occurs in the puerperium. Although this possibility must be borne in mind, it must be remembered that it is rare, whereas atypical mastitis and breast abscesses are common. Indeed the use (or rather the misuse) of antibiotics has greatly increased the incidence of this sullen, subacute type of suppurative mastitis. The solution of the problem is straightforward. The breast should be incised and in most cases pus is found. If any doubt remains tissue is taken for microscopy.

**Galactoceles.** This is a rare condition occurring during lactation, and is caused by the blockage of a main duct by inflammation or scarring. The obstructed segment becomes distended with milk and a swelling forms. There is often some associated subacute inflammation, and a breast abscess may be suspected. Aspiration may be sufficient to cure, but incision combined with suppression of lactation may be necessary.

**Tuberculosis.** This rarely affects the breast, but may do so in two ways.

Paget's disease before a lump is palpable is radical mastectomy. When a tumour has appeared, its stage of development decides treatment.

### INFECTIVE CONDITIONS OF THE BREAST

The skin and subcutaneous tissue of the breast are subject to all the diseases which may affect these tissues anywhere else. The large sebaceous glands of the areola are particularly liable to infection and may cause *areolar abscesses*. The history, physical signs and course of this condition usually make the diagnosis obvious. The treatment is incision and drainage.

**Puerperal Mastitis.** A few days after delivery when lactation is commencing, both breasts become engorged, slightly painful and tender. There may be slight fever. This is not an infective condition, and subsides spontaneously if normal suckling is possible. Infective mastitis develops in the second week or later and usually affects only one of the breasts which becomes swollen and painful with redness and œdema of the skin. There is considerable fever, usually  $102^{\circ}\text{F}$ . ( $39^{\circ}\text{C}$ .) or more. In some of these patients there has been difficulty with suckling so that the breast has not been emptied regularly; the nipple may have become cracked. The infective organism is usually the *Staphylococcus aureus* (pyogenes) and the route of infection may be up the ducts from the nipple or by the blood stream. Pain makes suckling difficult, and this leads to further swelling of the breast and aggravates the infection. If effective treatment is not given, suppuration occurs and spreads through the breast. Abscesses may form. Sometimes there is septicæmia or pyæmia. The diagnosis is usually straightforward because of the characteristic circumstances. Early engorgement or "milk fever" can usually be distinguished. The diagnosis of *mastitis carcinomatosa* does not arise until anti-infective treatment has failed. The first essential in treatment is the prompt administration of antibiotics. Penicillin (250,000 units intramuscularly six-hourly) has hitherto been the standard agent, but the incidence of infection in hospital with penicillin-resistant organisms is becoming so high that other antibiotics must often be used. A good deal can be done to prevent mastitis by attention to the nipples and by efficient management of suckling in the early stages of lactation. Once mastitis has developed, suckling should be continued if pain and the general condition of the patient allow it or at least the breast should be regularly emptied by pump or manual expression. Between feeds the breast should be firmly bound. If breast feeding has to be abandoned, lactation should be promptly suppressed by administration of œstrogens.

**Breast Abscess.** In the great majority of cases breast abscess follows acute puerperal mastitis. If mastitis fails to resolve, an abscess forms in the stroma of the mammary disc. This tough tissue resists dissolution, and the suppurative process cannot clear space for a single abscess cavity, consequently the pus lies in several communicating loculi. The toughness

that fibro-adenosis is a preneoplastic and particularly a precarcinomatous condition. The argument from histology is inconclusive and the debate continues. The clinical evidence does not support the conclusion that fibro-adenosis is precarcinomatous, and the surgical attitude to the condition is based on the clinical evidence.

The chief symptom of fibro-adenosis is pain in the breast occurring usually in the premenstrual phase. It often affects both breasts, but one is usually worse than the other. Quite commonly the pain radiates beyond the confines of the breast, over the chest and shoulder. Another symptom is the discovery by the patient of a lump or nodularity in the breast. Occasionally there is discharge from the nipple. The age at which these symptoms occur is between twenty and fifty years. Pain is the common symptom in the younger patients (twenty to thirty-five years) and a lump (often a cyst) in the older group. Fresh symptoms after the menopause are rare.

The physical signs in the patients who complain of pain are a diffuse, firm nodularity and tenderness. The changes are often more marked in one part of the breast, usually the upper and outer quadrant. Both breasts are usually affected, but one more than the other. In the examination of these cases, great care and sound judgement are required in answering the question, "Is there a lump?" The axillary glands are often unusually easy to feel.

The treatment of fibro-adenosis is chiefly symptomatic. One of the most important points is to reassure the patient that the condition is harmless. The relief of pain is difficult. A well-fitting brassiere may help. Endocrine treatment might be expected to be effective as the cause of fibro-adenosis is probably an endocrine disturbance, but the results are very disappointing. This is hardly surprising, for the endocrine control is likely to be a complicated and delicate balance between at least three hormones (oestrogens, progesterone and androgens), and artificial administration would not be expected to restore and maintain the balance once it is lost. In practice, the best results are obtained with androgens, which are most conveniently applied by inunction of ointment (3 to 10 mg. testosterone daily). Short-wave diathermy may bring temporary relief. If there is a local lump it is, of course removed chiefly to establish the diagnosis, but its excision may greatly relieve the patient by removing a cause of worry.

**Cysts of the Breast.** These are common and are almost always due to fibro-adenosis. Small cysts, not detectable as distinct lumps clinically, occur in all varieties of fibro-adenosis, including those in young women, but cysts producing clinical manifestations are usually found between the ages of thirty-five and fifty. About half the patients have found a lump in the breast and the other half complain of pain. The physical signs are those of fibro-adenosis together with a local lump most commonly in the upper and outer quadrant. The lump may be obviously fluctuant

A hæmatogenous focus may arise in the breast itself, or the breast may be involved secondarily by infection spreading from the chest, particularly from a mediastinal gland. In either case, typical tuberculous granulation tissue forms, and caseation may ensue with subsequent liquefaction and formation of tuberculous "pus." Sinuses may appear and the characteristic bluish, anæmic granulations at their orifices may strongly suggest the diagnosis. But even at this latestage, and much more so earlier, the chronic induration of part of the breast simulates carcinoma. Biopsy is always necessary.

**Fibro-adenosis.** This is a very common disorder of the breast which takes a variety of forms. Many other terms have been used for it, such as chronic mastitis, cystic mastitis, interstitial mastitis, cysto-adenoma papilliferum, involution cysts, cystic disease of the breast, Schimmelbusch's disease, mazoplasia and chronic cystipherous epithelial hyperplasia. Most of these terms are inappropriate because they imply pathological processes which have no part, or no essential part, in this condition; the term fibro-adenosis is objective and non-committal.

The female breast, almost throughout life, is subject to periodic change, an ebb and flow of cellular activity regulated by the endocrine system. The first manifestation of this tendency may show itself in the first few days of life when the breasts may swell, a condition termed *mastitis neonatorum*, and presumed to be due to the residual effects of maternal oestrogens. At puberty the breasts enlarge due to proliferation of the glandular elements and increase of the stroma. From this time onwards, growth and retrogression alternate at every menstrual period; the flow of activity is premenstrual and the ebb intermenstrual. These changes take place on a far greater scale during pregnancy and lactation. At the menopause the regular cycles cease and involution sets in, but it is a gradual and rather irregular process. These cyclical cellular changes do not affect every part of the breast to the same extent at every cycle, but in the healthy breast they are fairly evenly spread. In the healthy breast also, a fairly even balance is maintained between involution and evolution. When this balance is upset or when the distribution of the changes is uneven, fibro-adenosis develops. There is an irregular increase of the fibrous tissue of the stroma, and the glandular elements show proliferation of alveoli and hyperplasia of the epithelium lining them and the ducts. Cells may be cast off into the lumen. A common sequel is the formation of cysts due to overgrowth of the alveoli or obstruction of their ducts. The cause of fibro-adenosis is unknown, but it is presumed to be due to a disorder of hormone control.

The extreme degrees of epithelial hyperplasia which occur in fibro-adenosis closely resemble under the microscope some of the less malignant forms of neoplasm such as intraduct carcinoma. Alveolar proliferation combined with stromal overgrowth resembles under the microscope the common benign neoplasm, fibro-adenoma. It has therefore been suggested

that fibro-adenosis is a preneoplastic and particularly a precarcinomatous condition. The argument from histology is inconclusive and the debate continues. The clinical evidence does not support the conclusion that fibro-adenosis is precarcinomatous, and the surgical attitude to the condition is based on the clinical evidence.

The chief symptom of fibro-adenosis is pain in the breast occurring usually in the premenstrual phase. It often affects both breasts, but one is usually worse than the other. Quite commonly the pain radiates beyond the confines of the breast, over the chest and shoulder. Another symptom is the discovery by the patient of a lump or nodularity in the breast. Occasionally there is discharge from the nipple. The age at which these symptoms occur is between twenty and fifty years. Pain is the common symptom in the younger patients (twenty to thirty-five years) and a lump (often a cyst) in the older group. Fresh symptoms after the menopause are rare.

The physical signs in the patients who complain of pain are a diffuse, firm nodularity and tenderness. The changes are often more marked in one part of the breast, usually the upper and outer quadrant. Both breasts are usually affected, but one more than the other. In the examination of these cases, great care and sound judgement are required in answering the question, "Is there a lump?" The axillary glands are often unusually easy to feel.

The treatment of fibro-adenosis is chiefly symptomatic. One of the most important points is to reassure the patient that the condition is harmless. The relief of pain is difficult. A well-fitting brassiere may help. Endocrine treatment might be expected to be effective as the cause of fibro-adenosis is probably an endocrine disturbance, but the results are very disappointing. This is hardly surprising, for the endocrine control is likely to be a complicated and delicate balance between at least three hormones (oestrogens, progesterone and androgens), and artificial administration would not be expected to restore and maintain the balance once it is lost. In practice, the best results are obtained with androgens, which are most conveniently applied by inunction of ointment (3 to 10 mg. testosterone daily). Short-wave diathermy may bring temporary relief. If there is a local lump it is, of course removed chiefly to establish the diagnosis, but its excision may greatly relieve the patient by removing a cause of worry.

**Cysts of the Breast.** These are common and are almost always due to fibro-adenosis. Small cysts, not detectable as distinct lumps clinically, occur in all varieties of fibro-adenosis, including those in young women, but cysts producing clinical manifestations are usually found between the ages of thirty-five and fifty. About half the patients have found a lump in the breast and the other half complain of pain. The physical signs are those of fibro-adenosis together with a local lump most commonly in the upper and outer quadrant. The lump may be obviously fluctuant



if it is large or near the surface of the breast. Often, however, the cyst is smaller and lies buried in the mammary disc; fluctuation then cannot be elicited. The lump may feel elastic (resembling a fibro-adenoma), but a very tense cyst, lying deep in nodular breast tissue, may be indistinguishable from a carcinoma. A cyst does not seem to move freely in the breast tissue.

Occasionally a cyst of the breast is associated with carcinoma in one of two ways. Firstly, a papilliferous carcinoma may grow from the lining of a cyst. Clinically, there is usually no indication of this possibility unless the cyst is emptied. Secondly, there is the rare cyst formed by liquefaction of the centre of a carcinoma or sarcoma.

The clinical diagnosis of a cyst must always be confirmed. Aspiration with a needle and syringe can demonstrate the fluid contents, but this test is not complete until the cyst has been emptied and further palpation has then established that the lump has completely disappeared. Omission to complete the test in this way may be failure to discover an intracystic tumour. Aspiration is also a convenient method of treatment, and in only one-third of cases does the cyst refill. If the diagnosis of a cyst is not confirmed by aspiration, operation is necessary. Although it has the advantage of preventing recurrence of the original cyst it does not prevent cysts forming in other parts of the breast due to the widespread fibro-adenosis. Operation consists of the excision of the segment of the mammary disc containing the cyst usually through a radial incision. The tissue removed is always examined microscopically.

**Fat Necrosis.** This is an uncommon condition, important chiefly because it may simulate carcinoma. It may involve the subcutaneous fat over the breast or the mammary disc itself. The cause is not fully understood, but there is a history of trauma in about 40 per cent. of cases. Whatever the cause, the onset is an area of necrosis followed by saponification of the fat and surrounding fibrosis. Macroscopically the cut surface is not concave (cf. carcinoma) and shows dull white areas and a pinkish periphery. Clinically there is a firm, well-defined area in the breast which is attached to the skin in half the cases and there may even be peau d'orange and retraction of the nipple. Treatment consists of excision of the affected area, chiefly in order to confirm the diagnosis by histology.

### BENIGN TUMOURS

Benign connective tissue tumours occasionally occur in the breast or immediately adjacent to it, e.g. lipoma, fibroma, myoma, osteoma, chondroma and angioma. They are rare compared with the common epithelial tumour. This is essentially an adenoma, but there is always associated overgrowth of the fibrous stroma around the glandular elements, and the tumour is usually called a *fibro-adenoma*. In the commonest variety, the fibrous stroma predominates, and Turnbull called

it *adenofibroma*. Histologically, these tumours closely resemble fibro-adenosis; the most striking difference is in a macroscopic feature, the complete encapsulation of the fibro-adenoma which enables it to be "shelled out." These tumours occur between puberty and about the age of thirty-five years. The history is that of the accidental discovery of a lump. On examination it is oval, clearly defined, elastic or hard and freely movable. Treatment is removal (Fig. 498).

There is a rare type of fibro-adenoma sometimes called the soft fibro-adenoma. It grows rapidly and attains a large size; it may even cause



FIG. 498. Large fibro-adenoma of left breast. (Mr. K. A. Moore's case.)

pressure necrosis of the overlying skin. Treatment is the same as for the common, smaller, hard fibro-adenoma.

**Benign Papillary Tumours.** These are adenomas, but are often called papillomas on account of their form. They occur in the ampullæ of the main ducts and also in cysts. They frequently cause bleeding from the nipple, but are sometimes an incidental finding within a cyst. The treatment is local excision. See also under *Discharge from the Nipple*.

### MALIGNANT TUMOURS

(A) **Tumours near the Borderline of Malignancy.** The breast not infrequently presents the difficult problem of a tumour or tissue whose cells show features suggesting carcinoma, but without any evidence of infiltration. Clinically these cases usually present as a lump in the breast, frequently with concomitant evidence of fibro-adenosis. The lump is

clinically indeterminate, but consistent with carcinoma. The cells lining the ducts and alveoli are larger than normal, stain deeply and are arranged in more than two layers. Mitotic figures show evidence of active cell division. The cells, nevertheless, remain on the right side of the basement membrane. The terms *intraduct carcinoma* or *carcinoma in situ* are used in such cases. Sometimes the epithelial hyperplasia has led to the casting off into the lumen of the ducts of masses of cells which form inspissated plugs. These plugs can be squeezed out of the cut ends of the ducts like blackheads, and Bloodgood coined the term "comedo carcinoma." A similar appearance may be seen sometimes in infiltrating carcinoma. Tumours with equivocal evidence of malignancy present a difficult problem to both surgeon and pathologist, and treatment cannot be standardised; usually a simple mastectomy is required.

Very rarely, doubtful evidence of malignancy is found, not in the epithelial but in the stromal elements of a breast tumour. An example is the adeno-myxo-fibro-sarcoma in which the stroma grows rapidly and sometimes undergoes cystic degeneration (*Brodie's serocystic tumour*). The overlying skin may slough so that the tumour fungates. The stroma shows atypical changes microscopically, and there is local infiltration, but metastasis does not occur and excision is not followed by recurrence. Pure sarcoma of the breast is extremely rare.

(B) **Carcinoma.** Malignant tumours of the glandular tissue of the breast are extremely common. Their cause is still unknown in spite of much research both on animals and in the human. A little is known, however, about several factors which may play a part in the causation of human breast cancer. It is probably true that there is a genetic factor and that a woman whose near relations have had breast cancer is herself a little more liable than the average to acquire the disease. Hormones are known to play a part in the causation and control of cancer, but very little is known about the working of this influence. There is at least suggestive evidence that oestrogens administered artificially may sometimes cause human breast cancer. Nothing has been discovered in cancer research in the human about the remarkable carcinogenic factor which is known to be transmissible by milk in mice. The maximum age incidence of human breast cancer is between forty and forty-five years, but it drops a little after this age. It is comparatively rare before thirty. There is no convincing evidence that trauma, marriage, pregnancy or lactation are causative factors.

Carcinoma of the breast may be classified histologically according to the shape of the cells, their arrangement, the products of their activity, and the amount of stroma. The first two considerations may give some indication of the degree of malignancy of the tumour. Commonly the cells are polygonal or spheroidal and have little regular arrangement, appearing in columns or sheets. Rarely, the cells are grouped regularly into alveoli; these are *adenocarcinomas* and have a favourable prognosis.

Occasionally the epithelial cells produce large quantities of mucus and form the *mucous* or *colloid carcinoma* which also has a relatively good prognosis. Myxomatous degeneration of the stroma, on the other hand, produces the very malignant *carcinoma myxomatoides*. When there is scant stroma the tumour is soft and cellular and is called *medullary*. Such tumours grow rather rapidly and become bulky, but the outlook is more favourable for them than for the commonest of all the breast tumours, the *scirrhous carcinoma*, in which the fibrous stroma predominates over the epithelial cells and gives rise to some of its most characteristic physical signs. A small number of carcinomas have a *papillary* form.

Carcinoma of the breast spreads like any other malignant epithelial tumour. By direct spread it may involve the skin, producing an ulcer or a fungating mass; or it may spread deeply and fix the breast to the pectoral muscle or even the ribs. Lymphatic spread is probably responsible for most of the metastatic deposits and is extremely common. It may take several forms, of which the commonest is probably *embolic*; cancer travels along the lymphatic vessels to the nearest lymph glands and there forms a secondary deposit; there is no trace of cancer along the route. The glands first affected are in the axilla, except in tumours of the medial half of the breast which may spread first to the internal mammary glands. The supraclavicular glands are usually involved by secondary spread from one or other of these primary groups.

Lymphatic *permeation* also occurs, a process in which malignant cells "grow along" a lymphatic channel which is thus occupied along its length by a continuous chain of carcinoma. Such a continuous chain is seldom found and the explanation of its absence is either that it never existed or (according to W. S. Handley) that lengths of the chain were destroyed by reactive fibrosis.

The spread of cancer along the lymphatic vessels normally occurs in the direction of the normal flow of lymph. When the normal channels are blocked by growth, flow may occur in abnormal directions, producing some of the characteristic signs of advanced mammary cancer. If the subcutaneous lymphatic plexus becomes blocked, the flow of lymph from the epidermis and dermis is impeded and a firm œdematous swelling of the skin ensues. This area is punctuated by a regular pattern of tiny indrawn points (the ducts of skin glands and hair follicles) giving the appearance aptly called *pigskin* or *peau d'orange* (Fig. 499). Sometimes by retrograde flow cancer emboli lodge in the dermis itself, and plaques or even wide sheets of infiltration form at a distance from the primary tumour, the so-called *carcinoma en cuirasse*. Diversion of lymph flow by blockage of the normal channels may also explain some of the cases of involvement of both breasts by carcinoma. The peritoneal cavity may be invaded by tumour cells moving along the lymphatics downwards into the rectus sheath and thence into the falciform ligament. The liver is a common site for secondary deposits.

Secondary deposits frequently form in bones, particularly bones fairly near the breast such as the dorsal vertebræ, the upper end of the humerus and the ribs. Rather less frequently, more distant bones are involved. The route by which cancer cells travel to the bones is not known for sure. The route may be the full course of the pulmonary and systemic circulation, for this is the most obvious physiological current which could carry a cell from the breast to a distant part of the body. The greatest objection to this suggestion is that in the majority of patients when secondary deposits appear in the bones the lungs are free. Although 20 per cent. of people have a patent foramen ovale, paradoxical embolism is probably

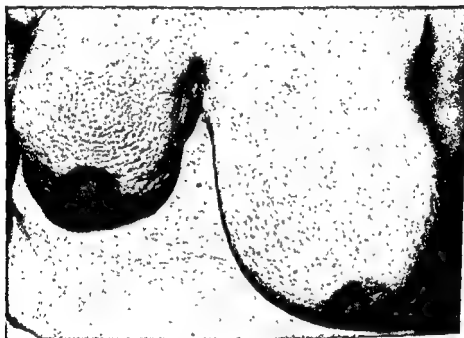


FIG. 490. Carcinoma of both breasts with marked *peau d'orange* of right breast.

far less common than this figure suggests. There is no direct lymphatic connection between the breast and the bones. The work of Batson on the vertebral veins suggests that there are irregular sluggish currents in venous backwaters which may carry cells long distances without their ever leaving peripheral venous "waters," let alone passing through the pulmonary circulation or into the arteries. This hypothesis would help to explain why the bones near the breast are most often affected, a fact which is quite inexplicable if the metastasis is by arterial embolism.

Untreated carcinoma of the breast does not kill the patient quickly. The average expectation of life is about three and a quarter years, and it is longer for medullary growths.

The usual symptom of a carcinoma of the breast is the accidental discovery by the patient of a painless lump. A common story is that the patient happens to come upon the lump while washing. Pain, which may

sometimes lead to the discovery of the lump, is a symptom in about 20 per cent. of cases. Sometimes it is a change in the nipple (e.g. retraction or ulceration) which first attracts the patient's attention. Occasionally the first indication of disease is due to a secondary deposit, e.g. a spontaneous fracture. The duration of the symptoms is very variable, and many patients neglect to seek medical advice out of fear or sometimes ignorance.

*Physical Signs.* Usually the general condition of the patient is unaffected when she first seeks advice. The physical sign is a lump in the breast, most commonly in the upper and outer quadrant. Quite often the

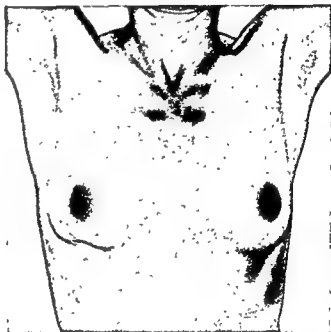


FIG. 500. Scirrhous carcinoma of right breast showing dimpling of the skin.

lump has certain features which make the diagnosis almost certain on clinical grounds alone, but in many of these cases the disease is advanced. The earlier and more curable the carcinoma, the less likely is it to have specific features. A carcinoma is typically hard, definite and not tender. Unlike a benign tumour, it is not freely movable in the breast tissue. All these features are obscured if the lump is small or buried deep in the breast, particularly if the breast is bulky or nodular. Two of the most characteristic features, namely tethering of the skin and retraction of the nipple, are due to contraction of the stroma which in turn causes an indrawing of attached elements. Tethering or dimpling of the skin (Fig. 500) is due to shortening of the ligaments of Cooper. It is a most valuable sign, which sometimes shows itself in an obvious constant dimple over the tumour, but sometimes it is only potential and its elicitation needs care. A good light is essential, and it is best directed obliquely.

By gentle pressure just beside the tumour the breast is moved slightly on the chest wall. The skin over the tumour is closely watched and a faint dimple can be seen to appear as the tumour moves relative to the skin. Retraction of the nipple is due to shortening of the fibrous septa which run to it from the part of the breast in which the tumour lies (Fig. 501). It is a later sign than dimpling of the skin.

Some important physical signs are due to extension of the carcinoma outside the limits of the breast. By extending deeply the growth involves the pectoral muscle or at least its fascial sheath. The sign is elicited by comparing the mobility of the breast when the muscle is relaxed and when it is contracted. In order to contract the muscle, the patient is told to press firmly with a hand against the hip.

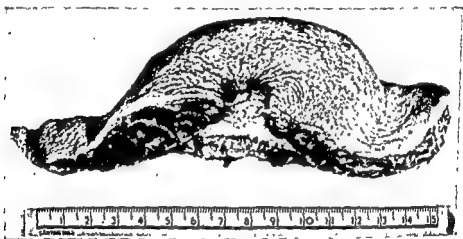


FIG. 501. Carcinoma of breast. The nipple is retracted by the tumour underlying it.

Further inward extension of the growth involves the ribs and fixes the breast irrespective of the state of contraction of the pectoral muscle. Ulceration, fungation (Fig. 502), peau d'orange and carcinoma en cuirasse are late and obvious physical signs. Enlargement of the regional lymph glands is extremely important evidence in assessing the extent of spread of a carcinoma, but it is often of little help in making the diagnosis, for the difficulty is most likely to arise in the relatively early case without glandular involvement.

On the basis of the physical signs, carcinoma of the breast may be classified according to the state of advancement of the disease. This staging is important in the assessment of operability and also in the evaluation of the results of treatment. Unfortunately, there is no general agreement of the details of a system, but most systems agree in outline. *Stage 1.*—The tumour is confined to the breast and if the skin is involved it is by direct extension and of smaller extent than the size of the tumour. The axillary glands are not palpable. *Stage 2.*—The axillary glands are enlarged, but are mobile. *Stage 3.*—The tumour involves the pectoral

muscle but not the ribs. Skin involvement may be wider than the tumour, but confined to the skin over the breast. Axillary glands are still mobile. *Stage 4.*—The tumour is fixed to the ribs; the skin may be involved beyond the limits of the breast. The axillary glands are no longer mobile and more distant glands may be palpably enlarged. In some schemes of classification there are only three stages which correspond roughly with stages 1, 2 and 4 above. Clinical staging is subject to error. In one series



FIG. 502. Fungating carcinoma of left breast.

carcinoma was found by histology in the axillary glands in 29 per cent. of cases placed in stage 1, whereas in 23 per cent. placed in stage 2, no carcinoma was found by histology in the axillary glands. Moreover, a clinical classification can take no account of the internal mammary glands and Handley has shown that they are involved in 14 per cent. of cases in which the axilla is free of growth.

In the clinical diagnosis of carcinoma of the breast, three types of problem arise. The first is the problem of deciding whether or not there is a lump in the breast. This difficulty arises when the lump is small or indefinite and when the breast is nodular or bulky. A decision must be made on clinical grounds alone and it must be final. The second type of



problem is presented by the lump which has no specific features of carcinoma. The common alternative possibilities are a fibro-adenoma, a cyst, and fibro-adenosis. Careful consideration of all the clinical data may make the diagnosis almost certain, but in all such cases the final decision depends on histology. In the third type of case, some of the characteristic features of carcinoma are present, such as dimpling of the skin or retraction of the nipple, or even fixation of the breast to the chest wall. In these cases, a diagnosis of carcinoma is almost certain, but there are some rare conditions such as fat necrosis, chronic infection (e.g. tuberculosis) and the effects of trauma, which can produce some of the physical signs typical of carcinoma, even of advanced carcinoma. Circumstances may suggest that the appearances of carcinoma are being mimicked by one of these rare conditions, but the diagnosis finally depends on biopsy.

*Biopsy.* In tumours of the breast, the diagnosis must always be confirmed by histology. Sometimes the clinical diagnosis is sufficiently definite to justify the performance of a radical operation without previous confirmation. Often the clinical diagnosis is suggestive but not certain, and it requires confirmation before the right treatment can be selected. In these cases, the patient is prepared for radical mastectomy and then the diagnosis is confirmed in one of several ways. When the tumour is not large it is excised with a margin of normal breast tissue. The tumour is then incised, and in many cases the macroscopic evidence gives sufficient confirmation of the diagnosis for a decision to be made on the type of operation. A harmless cyst may be revealed or the harsh, grating, slightly concave cut surface of a scirrhous carcinoma, or again the slightly convex, whorled cut surface of a fibro-adenoma with its obvious encapsulation. If the macroscopic evidence is doubtful, one of two courses may be taken. Microscopy by immediate frozen section is the ideal course if the pathologist is expert in this method as it allows treatment to be decided on and carried out forthwith. The alternative course is to close the wound and await the result of the study of paraffin sections. There is no evidence that a delay of a few days between biopsy and radical operation has any adverse effect on the patient's prospects if the lesion is found to be carcinoma. When the doubtful tumour is so large that its removal for biopsy almost entails removal of the breast a biopsy may be done by incising the tumour *in situ*, preferably with the diathermy knife. Needle biopsy is not generally considered satisfactory for the breast.

*Treatment.* Three main lines of treatment are available : (1) Surgery ; (2) radiotherapy ; and (3) hormonal methods.

*Surgery.* (A) *Radical Mastectomy.* This consists of the removal of the tumour, the breast, the pectoral muscles and fascia, together with the axillary lymphatic glands, all in one piece. It is the classical example of a radical cancer operation in which the tumour and its lymphatic drainage system are removed in continuity together with a wide margin of healthy

tissue. The operation (often associated with the name of the American, W. S. Halsted) has been extensively used for over sixty years. It causes considerable operative shock, and the mortality is 1 or 2 per cent. It is mutilating, but the deformity can be completely hidden under clothes. It is followed by œdema of the arm in about 14 per cent. of cases due to obstruction in the axilla of the lymphatics draining the arm. It often causes some stiffness of the shoulder joint.

(B) *Simple (or Local) Mastectomy.* This is the removal of the breast (including the nipple), without the pectoral muscle and without dissecting the axilla. It is sometimes done in combination with radiotherapy particularly in advanced cancer. Mutilation is slighter than after radical mastectomy, and the shoulder and arm are unaffected. Death due to the operation is rare.

(C) *Restricted Radical Mastectomy.* A simple mastectomy is combined with dissection of accessible axillary glands without removing the pectoral muscle. This operation also is usually employed in combination with radiotherapy. It avoids some of the mutilation and disability of radical mastectomy.

(D) *Extended Radical Mastectomy.* The standard operation of radical mastectomy may be extended by dividing the clavicle and removing the supraclavicular lymph glands, or by dividing the upper costal cartilages and removing the internal mammary glands, or both. The restricted radical operation has also been modified by including in its scope the internal mammary chain of glands. The results obtained by these operations (C and D) have not so far proved their superiority over the standard methods of treatment.

*Radiotherapy.* Although surgery remains the standard method of treatment in all cases deemed curable, the improved technique and equipment of radiotherapy have greatly extended its scope in carcinoma of the breast, and it is now much used in combination with surgery.

*Hormonal Methods.* The normal breast is known to be dependent on hormones for its structural development and functional activity. It is now known that about 50 per cent. of breast cancers are dependent on hormones for their continued growth. This is not to say that hormone treatment has ever cured cancer of the breast, so that it is never used in cases which might be cured by surgery or radiotherapy, but very advanced cancer can sometimes be checked or even made to regress by hormone treatment. The hormones chiefly responsible for maintaining the activity of cancer are œstrogens (produced mainly by the ovaries and the adrenals) and prolactin (produced by the pituitary), these two having a synergistic action. The baneful hormone influence may be reduced by removing the ovaries and adrenals, by administering androgens (to counteract the natural œstrogens), or by removing or destroying the pituitary. Success is also sometimes achieved by administering œstrogens (which act by suppressing pituitary function). The attack on the pituitary has been

used only recently, and insufficient data have been collected to evaluate its long-term results, but it is known that the other methods tend to lose their effect after about two years. When this happens further relief may be obtained by switching the attack to one of the other methods.

*Choice of Treatment.* For stage 1 carcinoma the best results of radical mastectomy have never been equalled by any other method of treatment. Gordon-Taylor has recorded 84 per cent. of ten-year cures. A good deal seems to depend on skilful, gentle technique. Radical mastectomy is the treatment for stage 1 cases.

In stage 2 the results of any form of treatment are very much inferior to the results in stage 1. Gordon-Taylor's figures for stage 2 are 29.4 per cent. of ten-year cures. Moreover, no one method has proved its superiority over the other methods. Radical mastectomy is advocated by the majority of surgeons, but a complementary course of radiotherapy is given as well, usually post-operatively. There is evidence that once the axillary glands are involved surgical clearance of the axilla may actually cause further spread of the carcinoma. For this reason there is a case for confining surgery to simple mastectomy and relying on radiotherapy alone for the axillary deposits. Alternatively a restricted radical mastectomy may be done and complementary radiotherapy given.

In stage 3 a very careful selection must be made, for surgery may easily make things worse. In many cases radiotherapy should precede surgery which should be restricted to simple mastectomy.

In stage 4 surgery has no place except sometimes to remove a fungating tumour by simple mastectomy when the activity of the carcinoma has been temporarily checked by radiotherapy. In this and more advanced stages, hormone treatment may be beneficial, particularly when there are secondaries in bones causing severe pain. No reliable method has yet been worked out by which the hormone-resistant cases can be distinguished from the hormone-dependant before treatment is given. The simplest hormone treatment is the administration of androgens (50 mg. testosterone propionate thrice weekly by intramuscular injection or methyl testosterone 100 mg. daily by mouth) or oestrogens (15 mg. daily by mouth), but more effective suppression of oestrogenic activity is obtained by removal of the ovaries and adrenals. Removal of the pituitary or its destruction by implantation of a source of  $\beta$ -radiation may reinstate hormone control which has gradually been lost after removal of the ovaries and adrenals. It may also be used as a first line of attack.

**The Male Breast.** The mammary disc in the male measures only 1 inch in diameter. It contains ducts, but no alveoli. The flatness of the mammary disc and the small amount of subcutaneous fat over it makes it an inconspicuous prominence.

Disorders of the male breast are far less common than those of the female. The commonest disorder is a slightly painful swelling of the

mammary disc usually called *mastitis* although it is not inflammatory. It occurs most commonly at puberty or in adolescence and causes a uniform slightly tender swelling of the disc. No treatment is indicated and the breast gradually returns to normal spontaneously. A similar condition occurs occasionally in adults, and in them it is important to establish the diagnosis with certainty because of the possibility of carcinoma. If the whole mammary disc is smoothly and evenly enlarged, and if

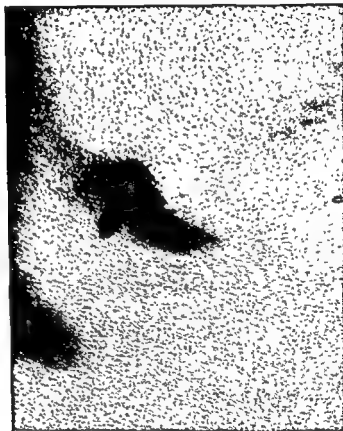


FIG. 503. Carcinoma of male breast (left).

it is tender, "mastitis" may be diagnosed, but if it does not show clear signs of subsiding within a month, the mammary disc should be excised.

*Carcinoma* is a hundred times less common in the male breast than in the female. The pathology, physical signs and treatment are the same as in the female, but the prognosis is worse (Fig. 503).

*Gynæcomastia* is enlargement of the male breast affecting both the mammary disc and the subcutaneous fat over it, so that it comes to resemble the female breast. It may occur on one or both sides. Two classes must be distinguished, those due to testicular disorders, and those in which the testes are normal. Testicular abnormalities which may cause gynæcomastia are of two types. In the first type, the testis is

clinically abnormal due to a variety of conditions, e.g. neoplasm (particularly teratoma), atrophy (following trauma, mumps, epidymo-orchitis, or unknown causes) or absence (orchidectomy). In the other type the abnormality of the testis is recognisable only by histology and there are other endocrine disturbances (Klinefelter's syndrome). Gynæcomastia with normal testes may be associated with a variety of conditions, particularly diseases affecting liver function (e.g. cirrhosis). Treatment of gynæcomastia should first be directed to the cause. If this is not curable the size of the breast may be reduced by excision of the mammary disc through a semi-circum-areolar incision.

## SECTION X

### PERIPHERAL VASCULAR SURGERY

by  
D. L. B. FARLEY

#### CHAPTER 74

### THE PERIPHERAL CIRCULATION. THE ARTERIES

THE *peripheral vascular system* includes the blood vessels and lymphatics supplying the extremities. The peripheral arterial circulation is modified by : (a) Arterial disease ; and (b) variations in tone of the muscle walls of the arteries. The tone of the arteries of the skin and subcutaneous tissues is largely controlled by the sympathetic nervous system, whereas the tone of the vessels which supply muscles is greatly influenced by chemical substances liberated during muscular activity. *Defective arterial supply*, commonly called arterial insufficiency, results in pain, loss of function, or gangrene. When tissues suffer from an insufficient blood supply the term *ischæmia* is applied. *Impaired venous return* (i.e. venous insufficiency) leads to peripheral œdema ; gangrene is extremely rare, but skin ulceration frequently occurs in the lower limbs. In *lymphatic obstruction or stagnation* peripheral œdema is even more marked, and again skin ulceration may result.

**Intravascular thrombosis** implies clot formation within a blood vessel or in the heart. It is favoured by injury or disease of the tunica intima, slowing of the blood stream and an increase in the coagulability of the blood. Clotting may be a slow or rapid process, and the largest vessels may become blocked. Thrombosis is a hazard even in the absence of obstruction, for either the whole or part of a clot may be swept by the blood stream to occlude a vessel beyond (*embolism*). A thrombosed vein often becomes partly or completely recanalised, but a thrombosed artery is usually permanently blocked.

**Embolism.** The Greek word embolus means a peg or stopper. The most common embolus is a detached portion of blood clot. Other forms of emboli include atheromatous plaques, part of a vegetation in endocarditis, fragments of tumour, fat particles and air bubbles. Foreign bodies, liquor amnii and parasitic ova may act in this way extremely rarely. Arterial emboli lodge further down the artery or in one of its

branches, most commonly at a bifurcation. Emboli from systemic veins pass through the heart before becoming arrested. Such emboli usually enter the pulmonary system after negotiating the right auricle and ventricle; on exceptional occasions, emboli pass through the foramen ovale thus finding a way into the peripheral arterial system. These are called "*paradoxical emboli*." If a vessel is not completely blocked by an embolus, thrombosis around it soon completes the obstruction. Peripheral arterial embolism is discussed under acute occlusion. Fat and air embolism are dealt with under venous trauma.

*Pulmonary embolism* is most often due to detachment of part of a venous clot (see under thrombophlebitis and phlebothrombosis). Although the original embolus may be large, it may however become broken up in its passage through the heart so that multiple emboli are released into the pulmonary circulation. If a large clot lodges in the pulmonary artery, collapse is usually dramatic, with acute dyspnoea, pain in the chest, venous congestion and cyanosis. Prior to the collapse, a patient has often just asked for a bedpan or his bowels may have been moved precipitously. More peripheral, usually multiple emboli produce only moderate shock and dyspnoea, but pain on breathing from pleural irritation and the expectoration of blood-stained sputum later result from the pulmonary infarction that develops. Very small multiple emboli result in patches of pulmonary collapse and pneumonitis, often indistinguishable clinically and radiologically from bronchopneumonia. *A patient who has survived one attack of embolism has a 25 per cent. chance of a further episode.*

*Treatment of Pulmonary Embolism.* In severe cases, immediate treatment includes oxygen, morphia (16 mg. or  $\frac{1}{4}$  gr.) or pethidine (75 mg.), and atropine (1.0 mg. or  $\frac{1}{80}$  gr.). Trendelenberg's heroic operation consists of immediate thoracotomy and extraction of the clot from the pulmonary artery. As in the early stages it is impossible to know whether or not recovery will take place spontaneously, such a hazardous procedure is rarely undertaken. Anticoagulant drugs should be given in order to reduce the likelihood of further emboli. Antibiotics are indicated to lessen the risk of superadded pneumonia.

*Gangrene* is necrosis or death *en masse* of a part of the body, usually part of a limb or internal organ. It is most commonly due to ischaemia. The types of gangrene together with some examples of each are as follows:

(1) *Ischaemic.* Sudden arterial interruption, such as traumatic division or acute occlusion from embolism. Increasing pressure on an artery, e.g. from a haematoma, or a tight plaster bandage. Progressive arterial obliteration, as in atherosclerosis. "Vasomotor" gangrene, e.g. in the Raynaud phenomenon.

(2) *Infective.* Gas gangrene, cancrum oris.

(3) *Toxic.* Diabetes, ergot poisoning.

(4) *Direct Injury.* Severe crushing injuries, burns, frost-bite, bedsore, constricting plaster casts.

(5) *Venous.* Deep venous thrombosis.

Gangrene is usually described as being of two varieties, dry and moist, but there may be a combination of both types. Gangrene does not always result from a single cause. For instance, in diabetes mellitus, gangrene is often due to a combination of atherosclerosis, lowered resistance to septic inflammation, and trophic change in the limbs from neurological defects. Again, the degree of atherosclerosis that in an old person leads to gangrene may prove harmless to a younger individual. This type of gangrene ("senile" gangrene, Fig. 504) results from a combination of causes, including peripheral atherosclerosis, reduced cardiac output due to age, and the presence of any other chronic disease, e.g., chronic nephritis. Ischæmic gangrene is discussed again later.

## THE ARTERIES

### ARTERITIS

Acute arteritis may follow septic wounds and lead to secondary hæmorrhage. As it can occur in septic cavities and in the vicinity of an abscess care must be taken to avoid contact between a drainage tube and an artery for fear of necrosis of the arterial wall. Secondary hæmorrhage is becoming rarer with the use of antibiotics for infected wounds. Arteritis at the site of a septic embolism may lead to dilatation of the vessel (*mycotic aneurysm*).

**Chronic Arteritis.** (1) *Arteriosclerotic degeneration* is the commonest type of chronic arteritis seen clinically. It is of three types: atherosclerosis, medial arteriosclerosis and diffuse hyperplastic sclerosis. *Atherosclerosis* is fully discussed under arterial occlusion. *Medial Arteriosclerosis*.—Some medial thickening is a natural accompaniment of age. Excessive degeneration may be followed by the deposition of calcium and even bone. As a result, arteries may become visualised on radiography throughout their whole length; the calcium is laid down in rings (*Mönckeberg's sclerosis*). Such vessels act as functioning conduits, however, and therefore do not produce symptoms. *Localised medial*

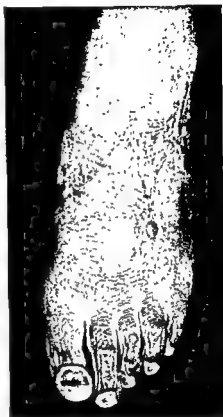


FIG. 504. Senile gangrene of the foot.  
(Courtesy of Professor C. G. Rob.)



sclerosis is seen in *atheromatous* (atherosclerotic) arteries, and in Great Britain it is the commonest cause of aneurysmal dilatation. Localised *mucinoid* degeneration of the media is a cause of dissecting aneurysm of the aorta. *Diffuse Hyperplastic Sclerosis*.—In this disease the arterioles develop hypertrophy of the muscle coat. It is usually accompanied by hypertension. (2) *Thrombo-angiitis obliterans* (Buerger's disease) is discussed under arterial occlusion. (3) *Syphilis* of the larger arteries may lead to aneurysm formation.



FIG. 505. Arteriogram in atherosclerosis in the leg, with occlusion of the superficial femoral artery. Note the characteristically wavy appearance of the collateral vessels. The artery immediately above and below the block has an irregular outline, whereas the profunda branch is normal. (Courtesy of Dr. C. Foster Cooper.)

### ARTERIAL OCCLUSION

Although occlusion implies complete obstruction, it is useful to consider vessels as being partly or completely occluded. A combination of both kinds of obstruction is usually seen in the obliterative types of arterial disease (Fig. 505). Arterial occlusion may be acute or insidious in onset, and any artery may be affected. The disease, which may be spasmodic in nature or due to structural narrowing of the arterial lumen, may be localised to one artery or widespread throughout the arterial tree. The symptoms of occlusion, whatever its cause, are due to ischaemia, and their severity depends on the degree of compensation provided by the collateral circulation. If the latter is efficient, no symptoms develop; if it is inadequate, pain, loss of function, ulceration or gangrene result. The ability of the collateral channels to maintain adequate nutrition depends on the anatomy of the part, the time allowed for their compensatory dilata-

tion, and their freedom from disease. The patient's general condition however is also of importance. Ischaemia is intensified, for example, by a fall of blood pressure, or, in chronic cases, by the presence of anaemia or diabetes mellitus. Complete ischaemia results in skin death in approximately twenty-four hours, but blisters may appear in twelve hours. Muscular necrosis occurs in eight hours, but peripheral nerves survive nearly as long as skin, although they cease to function after fifteen to twenty minutes. *If the limb is cooled all these times are prolonged.* Atrophy

and contracture of the muscles (*Volkman's ischaemic fibrosis*), or post-ischaemic neuritis may follow a period of acute ischaemia insufficiently severe to produce gangrene. In parts of the body where no large collateral arteries abound, small vessels open up and their walls hypertrophy. They may be felt to pulsate. Full development of the collateral circulation may take hours or weeks, and a slowly increasing arterial occlusion is more easily compensated for than an acute arrest. For this reason, any artery, including the aorta, may gradually become occluded without the development of major symptoms. Unfortunately, the commonest obliterating diseases are progressive, and collateral vessels often become blocked later.

Arterial ligation in healthy young persons demonstrates that, apart from aortic ligation, which is fatal, some major peripheral vessels may be acutely occluded without risk of ischaemia. Exceptions are the *common femoral artery*, the *internal carotid*, the *popliteal*, and the *axillary distal to its subscapular branch*. The arm is more generously supplied with collaterals than the leg, and gangrene is therefore relatively less likely. When the distal arteries are occluded, a collateral circulation is much less easily provided (Fig. 506), and as a general rule the more distal the disease the greater is the chance of gangrene.

In the study of arterial occlusion it is important to remember that the effects of obstruction depend on the anatomical situation of the lesion rather than on the nature of the pathological process. Diagnosis of the site of occlusion is therefore of prime importance. It is also of value in establishing the pathology of the condition. Atherosclerosis, for instance, affects the aorta and major peripheral arteries, whereas vasomotor diseases involve the most distal vessels, and thromboangiitis obliterans affects both medium-sized and small arteries.

#### CHRONIC ARTERIAL OCCLUSION

**Atherosclerosis.** This is a generalised, degenerative disease which affects the main arteries, the coronary and the intracranial arteries. It



FIG. 506. Arteriograms showing occlusion of the small distal arteries in Buerger's disease. At the ankle, mainly collateral vessels are apparent. No vessels can be seen in the foot. (Courtesy of Professor C. G. Rob. Arteriography by Dr. E. Rohan Williams.)

accounts for the majority of cases of peripheral arterial occlusion. Atherosclerosis commonly affects the aorta and its branches, extending distally to the renal, brachial and upper tibial arteries. The greatest incidence of this disease is in people over fifty, and it is more common in men than women. Hypertension occurs in a little under one-third of cases. Atherosclerosis is ten times more common in patients with diabetes mellitus, in which disease it tends to develop at an earlier age. From the distribution of the disease it is apparent that mechanical strain is an ætiological factor, since the major arteries sustain a high pressure, the coronary arteries are never at rest and the cerebral vessels have thin walls; atheromatous change is also particularly marked where branches leave the main stem.

Lipid material, mostly cholesterol ester, is deposited deep in the tunica intima. As it accumulates it extends to the surface and forms a plaque in the wall of the vessel. Plaques may coalesce and ulcerate. Thrombosis readily takes place in such situations and calcification often follows. In the early stages, absorption of the clot, leading to fatty changes, together with restoration of the continuity of the intimal surface, gives an appearance similar to the original atheroma. Although it is impossible to be certain how much the absorption of intramural thrombi contributes to the ætiology, it certainly plays a part in advancing the disease as well as being one of its main complications. Advance of the disease, together with secondary fibrosis, thrombosis and calcification, leads to severe narrowing of the lumen, and later its obliteration. Localised areas of atrophy in the medial coat are often seen. The size of the vessel is no bar to complete obliteration for even the aorta may lose its patency.

Although a vessel may be extensively diseased, segments of complete occlusion are often quite short and may be only a few centimetres in length. Many patients, for instance, are shown on arteriography to have a localised block in the lower part of the superficial femoral artery. Recanalisation of occluded arteries rarely occurs to any useful extent. Atherosclerosis is slowly progressive and amputations for gangrene are often necessary. Coronary thrombosis is responsible for the death (immediate or delayed) of many patients, and a large proportion of patients with peripheral atherosclerosis are found to have evidence of coronary artery disease. This association is of the greatest importance when surgical treatment is being considered.

*Symptoms.* The symptoms are due to impaired blood flow and include those of chronic ischæmia shortly to be described. Episodes of acute ischæmia may however occasionally result from thrombosis or from an embolus (detached from a plaque of atheroma).

**Thrombo-angiitis Obliterans** (Syn. Buerger's Disease). Thrombo-angiitis obliterans is far less common than atherosclerosis, and usually arises before the fortieth year. Men are affected almost exclusively. The disease is almost entirely confined to tobacco smokers, and if the habit is

broken the condition tends to run a more favourable course. It is no longer thought that either Russians or Jews are unduly susceptible. The disease consists of segmental occlusion of arteries and veins by a low-grade inflammation which binds the arteries and veins together. Adjacent nerves are rarely affected. The arterial muscular coat is fairly well preserved, and aneurysm formation is not a feature. Suppuration is never seen. The disease may start in the arm, but the anterior and posterior tibial arteries are usually the first affected. Small distal arteries eventually become involved (Fig. 506) and gangrene often results. Superficial thrombophlebitis is seen in over a third of cases. On microscopy, both artery and vein show partial occlusion with extensive perivascular fibrosis. Whereas the main arterial structure is fairly well preserved, the intima is thickened by endothelial proliferation and all coats are infiltrated with lymphocytes and plasma cells. A thrombus is always present and is usually well organised; recanalisation occurs to a limited extent. The disease is usually progressive, and spreads both distally and centrally. Amputation of one or more limbs may be necessary, but the prognosis with regard to life is good.

*Diagnosis.* The majority of symptoms are due to ischæmia. Pain at rest is particularly prominent. Gangrene may occur in any limb. Superficial thrombophlebitis may be observed. If deep vein thrombosis is extensive there may be cyanosis and œdema of the lower part of the leg. Fungus infection of the feet is often present. The earlier age of onset together with the presence of thrombophlebitis or of gangrene in the upper limb usually differentiates this disease from atherosclerosis. Unlike the latter, thrombo-angiitis obliterans is hardly ever associated with diabetes or hypertension. Biopsy of an area of thrombophlebitis may show changes on microscopy compatible with the diagnosis.

**Chronic Arterial Trauma.** Occlusion of the axillary artery may follow the use of crutches, and the subclavian artery may become thrombosed in cases of cervical rib. As the occlusion is gradual and well localised, the collateral circulation is usually sufficient to prevent gangrene, but lesser degrees of ischæmia may be apparent. Embolism from dislodgement of clot occasionally causes digital gangrene. Primary occlusion of the popliteal artery, which is most common in males, may be favoured by the manner in which the femoral artery enters the popliteal fossa, for the vessel undergoes intermittent compression at the adductor opening during exercise (see also the anterior compartment syndrome, Chap. 61).

**Post-traumatic Occlusion, Post-embolic Occlusion, and Occlusion following Thermal Injuries.** Chronic ischæmia symptoms may develop after arterial trauma, arterial embolism, frost-bite or trench-foot as a result of major arterial damage during the acute phase.

**Manifestations of Chronic Ischæmia.** Pain may be continuous or intermittent. Intermittent pain includes intermittent claudication, the pain of ischæmic neuritis and attacks of the Raynaud phenomenon to be

described later. *Rest pain* refers to continual pain, unrelieved by rest, resulting from severe generalised arterial deficiency and often heralding the onset of gangrene. It may be intense and occupy the whole of the patient's attention. The onset of gangrene does not usually lead to a significant reduction of the pain. *Intermittent Claudication*.—Originally meaning a "limp," claudication is now used to imply a state of crippling pain. It arises in muscle and is produced by muscular activity in the presence of anoxia. Anæmia may contribute to its development and even cause it. The pain is cramp-like and follows a feeling of tiredness in the muscles. The time it takes to develop after exercise and the distance travelled are called the "claudication time" and "claudication distance," respectively. Further deterioration in the vessels is reflected by a reduction in this time and distance. The pain disappears after cessation of effort, and in mild cases it may pass off even though exercise is continued, owing to collateral vascular dilatation. The situation of the pain depends on which arteries are diseased. As femoral and popliteal occlusion are the most common, claudication is most often situated in the calf of the leg. Intermittent claudication may, however, occur in the sole of the foot, and thus be confused with foot strain, or it may occur in the buttock or thigh, when it may be mistaken for sciatica. The site of pain does not betray the extent of the disease, since the patient rests as soon as pain is felt, thus preventing its development in other situations. When muscular atrophy results from spread of the disease claudication disappears. *The pain of ischæmic neuritis* is usually lancinating and shoots up and down the leg, but does not usually bear an accurate relation to the distribution of the involved nerves. *Muscular Weakness*.—Although rarely a presenting symptom, a degree of muscular atony and wasting may be found on examination.

*Skin Changes from Impaired Nutrition*. The subjective temperature of a limb is dependent on the skin blood flow. Patients therefore recognise an ischæmic limb as cold. Skin sensibility is altered; it may be hyper- or hypo-æsthetic, particularly in the digits. Infection commonly complicates ischæmia. Thus tinea pedis or smouldering paronychia may be an early symptom. Minor injuries heal poorly, and abrasions lead to ulceration. The onset of gangrene in approximately 50 per cent. of cases follows a mild injury or a minor surgical procedure on the foot.

*Gangrene*. In the obliterating vascular diseases, gangrene is often dry, but there may be a combination of both dry and moist types. *Dry gangrene* is commonly seen in the dry shrunken feet of elderly patients with atherosclerosis (Fig. 504). The dead tissue shrivels further owing to loss of moisture by evaporation, and becomes black from the deposition of blood pigment. As a result of further thrombosis in neighbouring vessels, the gangrene slowly spreads up the limb until more healthy blood vessels are reached. Here the growth of granulation tissue becomes apparent as a red line bordering on the dark area. This is known as the

"line of demarcation." After a period of time, the dead part or eschar may separate spontaneously. At the level of separation, the skin, which is nourished by vessels of very small calibre suffers more than the deeper structures and therefore the stump tapers to a point. *Moist gangrene* follows episodes of acute occlusion as the limb is full of blood at the time of arterial arrest. The limb is swollen and later may develop a foul odour from secondary infection. Capillary anoxia leads to transudation of more fluid into the tissues, thus adding to the wetness of the part. Constitutional symptoms may be severe, and bacteraemia or septicæmia may prove fatal. In this type of gangrene, if separation results, the skin suffers less than the deeper tissues and the stump is therefore flat.

**Clinical Examination.** Chronic peripheral ischaemia is usually part of a generalised disease; a general medical examination is therefore always necessary. Healthy limbs vary considerably in temperature and colour; therefore a comparison should always be made between extremities. A generalised atrophy of the limb is seen in cases of extensive atherosclerosis. The patient with claudication should be made to indicate clearly the distribution of the pain. Prominent tortuous arteries in the temples or in the arms are seen with medial sclerosis, but they usually function well and are not especially related to occlusive arterial disease.

**Colour.** This depends on: (a) The amount of blood flowing; (b) the oxygen content of the blood; and (c) the external temperature. A good flow is shown by a warm, pale skin, with visible and palpable veins. Cyanosis denotes a poor flow, as does reddening, if the skin is cold. *Buerger's Test.*—The normal leg shows only slight pallor of the foot on elevation, and normal colour returns quickly on lowering the limb. Limbs with an impaired blood supply become white on elevation owing to poor arterial filling; on lowering, colour returns slowly and may be either red or blue from capillary stagnation ("dependent rubor"). During the performance of this test, filling of the veins should be noticed when the limb is lowered; they normally become distended in five or six seconds.

**Skin Changes.** The skin is often smooth and hairless, and the normal calluses of the sole are often absent. There may be atrophy of the digital pulp. Trophic ulceration may be noted and onychogryphosis or chronic paronychia may be apparent.

**Gangrene.** The site and extent of gangrene is easily noted, but the upper limit of the gangrene may be well distal to the level of the arterial block.

**Skin Temperature.** This is best felt with the back of the hand. It is essential for both limbs to be exposed for a period to room conditions before the temperature is estimated; in these circumstances a lowering of temperature on one side implies a degree of ischaemia.

**The Pulses.** Skillful examination of the arterial pulses needs practice, but no clinical test is of greater importance. Vasodilatation in the limb should first be promoted by previous exercise and by performing the

examination in a warm room. The patient's muscles must be relaxed. If the examiner has difficulty in differentiating the patient's pulse from his own, he should see if the patient's *radial* pulse rate and rhythm are comparable with the pulse in question. Anomalous arteries must be recognised, e.g. a radial pulse palpable in the "anatomical snuff-box," or a peroneal pulse replacing the *dorsalis pedis* in the foot. In the arm, pulses can usually be felt with ease. In the leg, the femoral pulse is usually felt without difficulty, but the others sometimes prove elusive. Two methods are available in the case of the popliteal pulse: (1) The patient lies supine with the knee flexed and the muscles relaxed. Both the examiner's hands are placed round the knee allowing the middle fingers to sink deeply into the popliteal fossa. (2) The patient lies prone and the foot is supported by the examiner's left hand whilst the tips of the middle fingers or thumb of the right hand palpate the popliteal fossa. In feeling for the posterior tibial pulse, the examiner should stand on the side of the leg being examined. The pulse can usually be felt, but it may be obscured by oedema or marked obesity. The *dorsalis pedis* pulse is best felt on the dorsum of the foot just lateral to the first metatarsal. It is often more laterally placed than expected. The pulse of this artery is absent in nearly 10 per cent. of normal people, but in such cases there is usually a peroneal pulse in the region of the external malleolus. In cases of occlusion with good collateral compensation, pulsating vessels may be felt in unusual situations.

*Auscultation.* Murmurs can sometimes be heard with a stethoscope over sites of arterial narrowing. The blood pressure of the leg may be measured by placing an extra long cuff on the thigh and the stethoscope over the popliteal artery.

*The Claudication Time.* This is estimated by asking the patient to step up and down on a platform, to walk upstairs or to amble up an inclined slope, according to the severity of the disease.

*Investigations.* The clinical history and the performance of the tests described above provide sufficient information in many cases for assessing the site and severity of the occlusion. Rest pain, for example, indicates a severe diminution of blood flow to the skin; the degree of ischaemia in the muscles is suggested by the severity of claudication on exercise. Lesser degrees of arterial insufficiency are indicated by a comparison of the temperature, colour, pulses and the dependent rubor of both limbs. Special tests of importance are the blood haemoglobin level and urine examination for albumen and sugar. Often a blood glucose estimation (to exclude diabetes), or electrocardiography (to detect any coronary arterial disease) are required.

*Plain radiography* may show the calcified rings of medial sclerosis (Mönckeberg's sclerosis); although such vessels may be visualised in their whole length they do not produce symptoms. In atherosclerosis, patchy, sometimes quite localised calcification may be observed. Calcifica-

tion of the walls of an aneurysm and its contained thrombus may also be noted.

*Arteriography* in occlusive disease may show : (1) Irregularities in the arterial lumen ; (2) localised areas of complete occlusion ; (3) the distribution and efficiency of the collateral circulation ; (4) the extent of symptomless disease. It is essential to inject sufficiently high up the arterial tree for the upper level of the disease to be visualised—for example, in patients with claudication of the buttock, injection of the abdominal aorta is required. A team trained in taking serial pictures at intervals of a few seconds is necessary. General anaesthesia is advisable as the injection is painful. *Diodone*, B.P., is the medium most often used ; *Ithorast* has been abandoned owing to its carcinogenic properties in animals. Although a temporary arterial spasm may occur, complications such as hæmorrhage or embolism are very rare. Accidental extravascular injection of diodone gives rise to pain only. Sensitivity to the medium has proved fatal, but is avoided by first giving a test dose of 1 ml. intravenously. High concentrations should not be given to patients with defective renal or hepatic function. The femoral and brachial arteries are injected with 20 ml. of 30 to 50 per cent. solution. In *aortography*, 50 ml. of 70 per cent. diodone are necessary, and a special injection pump is an advantage. The aorta may be injected directly, or in retrograde fashion through a catheter passed along the femoral or brachial artery. Aortography, though less safe than femoral or brachial arteriography, is not often followed by complications. The latter, however, include oliguria from renal damage and paraplegia from damage to the spinal cord. Direct injection of the superior or inferior mesenteric arteries in error has resulted in gangrene of the intestine. Separation of a calcified plaque, splitting of the aortic wall or retroperitoneal hæmatoma has occasionally followed direct injection of a diseased aorta.

*Tests for Ischaemia.* For the following tests to be satisfactory the circulation must be at its maximum and the patient should be in a warm room and recently exercised. *Reflex Hyperæmia.*—The limb is elevated and gently massaged towards the heart until it is pale. A sphygmomanometer cuff is then applied for five to ten minutes. Normally, a flush spreads after its release over the whole limb within five to ten seconds, but in areas of skin with defective circulation no flush is seen. In arterial disease this test is not without risk as the use of a tourniquet is necessary. *Plethysmography.*—Measurement of the blood flow through the hand or foot indicates the state of the peripheral circulation of the skin and subcutaneous tissues. When used on the calf or forearm it demonstrates the amount of local muscle flow. Plethysmography finds its application in specialised clinics ; it is of most value in assessing the response to treatment. *Oscillometry.*—This consists in recording the change of pressure in a limb occurring with the heart beat. As the cuff is moved down the limb, the oscillation may be found to become abruptly reduced. This



instrument is of most value in assessing the level of an arterial block and in comparing the circulation of one limb with another. After *intravenous injection of radioactive isotopes* the circulation in the limbs may be assessed using a Geiger counter.

*Tests of the Capacity to Dilate. Reflex Heating.*—Peripheral vasodilatation occurs in response to a rise of body temperature. Heating part of the body, e.g. by means of an electric cradle over the trunk, or placing one arm in water at 44° C., provides a simple method of testing the degree of possible vasodilatation in the skin of the limbs. The patient should be at physical and mental rest. The room should be warm (15° to 22° C.), and the temperature must not be changed during the examination. Draughts must be prevented. The normal response is variable, but the toe temperature may rise from room temperature to 35° C. in fifteen minutes. In performing this test, a comparison is made between good and bad limbs with regard to both the rate of rise of temperature and the final temperature; the maximum recording is of less significance. *Sympathetic Block.*—Instead of the reflex heating test, the sympathetic chain may be blocked with local anaesthesia; the temperature is recorded as before. *Spinal anaesthesia* is unsatisfactory as the fall in blood pressure that occurs interferes with local blood flow. *Injection of the peripheral nerves with local anaesthetic*, performed at the knee or elbow is a simple method of producing vasodilatation; it is of value when only the foot or hand are to be assessed.

### THE TREATMENT OF CHRONIC OCCLUSIVE DISEASE

**General Measures.** Cold is injurious; therefore residence in a warm climate may be helpful. Tobacco smoking should be discouraged, as nicotine reduces the blood flow in the distal parts of the limbs; in thrombo-angiitis obliterans it should be forbidden. Reduction of weight in the obese reduces the work of the leg muscles. Diabetes must be controlled and anaemia corrected. As the occlusive diseases are only slowly progressive the patient should be reassured.

**Local Care of Ischaemic Limbs.** The limbs should be warmly clad, but direct heat is avoided. Constricting clothing, such as suspenders, must not be worn. The feet should be kept clean, but they should be dried with care and powder dusted over them and into the socks and shoes. Although full dilatation occurs during sleep, elevation of the legs is inadvisable during the day. Injury and infection are serious; therefore patients should not walk with bare feet, and new shoes must be carefully worn in. *Overriding toes should be gently separated with cotton-wool*, and cracks in the skin carefully rubbed with lanoline. The toenails should be cut with care; the edges are kept straight, not curved like fingernails. Horny nails should first be softened with oil before being cut. If necessary, the nails should be attended to by relatives or a chiropodist. Inspection of the soles of the feet should be made daily, particularly when there is

diminished sensation. Elderly patients may be unable to do this without a mirror.

**Attempts to Improve the Blood Supply.** *Buerger's Exercises.*—The legs are elevated and rested on a support for half a minute or until blanching occurs. They are then held dependent for three to four minutes. This procedure is repeated several times for two periods a day. *Intermittent Venous Occlusion.*—This has been recommended in the belief that the vitality of partly ischaemic tissues is improved by intermittently raising the venous pressure. It may be achieved by encasing the leg in a special envelope which alternately compresses and relaxes the part. Some, however, believe this procedure to be dangerous. Placing the leg in an artificial respirator is probably better, for the compressive phase is thereby avoided. *Vasodilator Drugs.*—Ganglion blocking drugs, such as the methonium and pentoline compounds, act predominantly on the splanchnic area; they are therefore of no direct value in this condition. Tolazoline hydrochloride (Priscol) may be of value; it is a direct anti-spasmodic and also has an adrenolytic action. It does not lower the blood pressure and may be given in doses of 25 to 100 mg. three times a day for long periods if side effects (flushing, palpitation and headache) are not troublesome. Intravenous aminophylline prolongs the claudication time, but its effect is only temporary. Intra-arterial injections of various drugs, including papaverine, procaine, Priscol and heparin, have proved disappointing. Long-term anticoagulant therapy in atherosclerosis is still under trial.

**Reduction of the Amount of Work Done by the Muscles.** Walking slowly delays the onset of claudication, and this may be encouraged by making the patient walk with his hands in his pockets. In calf claudication, the heel of the shoe may be raised  $\frac{1}{2}$  inch, or a check iron fitted to prevent ankle flexion. Complete rest leads to atrophy and thus denies the muscles their most potent of vasodilators (i.e. the liberation of metabolites); it is therefore contraindicated.

**Sympathectomy.** This has a beneficial effect on the skin and subcutaneous tissues by releasing vasoconstrictor tone and abolishing sweating. It is of considerable value in patients with impaired skin nutrition in whom direct arterial surgery is not feasible. Following sympathectomy superficial lesions often heal so that amputation is postponed or prevented; furthermore, if gangrene is already present, a lower amputation is often successful. The reflex heating test previously described may be helpful; a rise of skin temperature in the digits of three degrees is sufficient to justify operation. On the whole, however, this test is of less value than might be expected, for operation is often more satisfactory than it suggests. Sympathectomy may also give relief from rest pain and ischaemic neuropathy, but it is useless in diabetics whose peripheral nerves have degenerated, and it is contraindicated in the presence of oedema of the leg. The effect of sympathectomy on

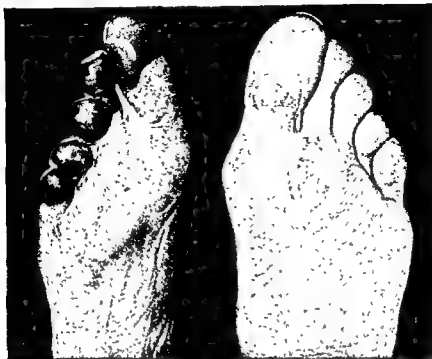
instrument is of most value in assessing the level of an arterial block and in comparing the circulation of one limb with another. After intravenous injection of radioactive isotopes the circulation in the limbs may be assessed using a Geiger counter.

*Tests of the Capacity to Dilate. Reflex Heating.*—Peripheral vasodilatation occurs in response to a rise of body temperature. Heating part of the body, e.g. by means of an electric cradle over the trunk, or placing one arm in water at 44° C., provides a simple method of testing the degree of possible vasodilatation in the skin of the limbs. The patient should be at physical and mental rest. The room should be warm (15° to 22° C.), and the temperature must not be changed during the examination. Draughts must be prevented. The normal response is variable, but the toe temperature may rise from room temperature to 35° C. in fifteen minutes. In performing this test, a comparison is made between good and bad limbs with regard to both the rate of rise of temperature and the final temperature; the maximum recording is of less significance. *Sympathetic Block.*—Instead of the reflex heating test, the sympathetic chain may be blocked with local anaesthesia; the temperature is recorded as before. *Spinal anaesthesia* is unsatisfactory as the fall in blood pressure that occurs interferes with local blood flow. *Injection of the peripheral nerves with local anaesthetic*, performed at the knee or elbow is a simple method of producing vasodilatation; it is of value when only the foot or hand are to be assessed.

### THE TREATMENT OF CHRONIC OCCLUSIVE DISEASE

**General Measures.** Cold is injurious; therefore residence in a warm climate may be helpful. Tobacco smoking should be discouraged, as nicotine reduces the blood flow in the distal parts of the limbs; in thrombo-angiitis obliterans it should be forbidden. Reduction of weight in the obese reduces the work of the leg muscles. Diabetes must be controlled and anaemia corrected. As the occlusive diseases are only slowly progressive the patient should be reassured.

**Local Care of Ischemic Limbs.** The limbs should be warmly clad, but direct heat is avoided. Constricting clothing, such as suspenders, must not be worn. The feet should be kept clean, but they should be dried with care and powder dusted over them and into the socks and shoes. Although full dilatation occurs during sleep, elevation of the legs is inadvisable during the day. Injury and infection are serious; therefore patients should not walk with bare feet, and new shoes must be carefully worn in. Overriding toes should be gently separated with cotton-wool, and cracks in the skin carefully rubbed with lanoline. The toenails should be cut with care; the edges are kept straight, not curved like fingernails. Horny nails should first be softened with oil before being cut. If necessary, the nails should be attended to by relatives or a chiropodist. Inspection of the soles of the feet should be made daily, particularly when there is



(a)



(b)

FIG. 507. (a) Atherosclerotic gangrene. (b) After excision and grafting of the occluded segment of artery. (Courtesy of Professor C. G. Rob.)

muscle is slight ; it is not therefore generally recommended for claudication, although when there is a high arterial block the muscle blood supply may be increased by the improved collateral circulation. The extent of the operation depends on the area to be sympathectomised. In the standard lumbar sympathectomy only the lower part of the leg is denervated. In order to release collateral arteries in the thigh, resection of the first lumbar ganglion is necessary ; for collateral dilatation in the groin and buttock, a low thoracic sympathectomy is required. Periarterial sympathectomy does not lead to peripheral vasodilatation, and is therefore no longer performed. The sympathectomies are described in the chapter on The Autonomic Nervous System, Chap. 60.

**Tenotomy of the Tendo Achilles.** This comparatively simple procedure may be performed subcutaneously under local anaesthesia. Walking is little interfered with, and the claudication time in the calf is considerably prolonged. The tenotomy may have to be repeated if reunion and contraction take place.

### RECONSTRUCTIVE SURGERY

Reconstructive operations may be required for localised occlusions which follow acute or chronic trauma, and for primary thrombosis. They are only of palliative value in atherosclerosis, and are contraindicated in Buerger's disease, for the most distal arteries become affected. In peripheral atherosclerosis, three out of four patients have localised blocks but only a few are found to be suitable for surgical treatment (Rob). Reconstructive surgery may be attempted in those patients whose work is being interfered with by ischaemic symptoms, and whose cardiovascular system is capable of withstanding the expected increase of exercise. The arterial supply of both legs must be assessed since there may be extensive disease on the other side. Reconstructive operations in young patients with gangrene, however, will probably become of increasing importance, for in some cases major amputations can be postponed for a number of years (Fig. 507).

**Endarterectomy (Syn. Arterial Disobliteration).** In atherosclerosis there is a plane of cleavage between the adventitial coat and the diseased inner layers, usually between the external elastic lamina and the tunica media. The diseased inner layers may be removed through a long incision in the artery and the adventitia reconstructed with interrupted stitches. Heparin is given during the operation and afterwards. This procedure may be highly successful in atherosclerotic occlusion of the iliac vessels, for the rapid flow of blood in these vessels tends to prevent post-operative thrombosis.

**Excision and End-to-end Anastomosis.** After resection of short segments of artery (2 to 3 cm.), end-to-end anastomosis may be possible as the vessels in atherosclerosis are often lengthened.

**Arterial Grafting.** Arterial excision and grafting may be performed in

followed by rapid healing, especially if sympathectomy is performed at the same time. In cases with atherosclerosis, a below or above knee operation is necessary if the foot is gangrenous. Although general anaesthesia is preferable, refrigeration anaesthesia, obtained by the application of ice to the limb for several hours, is a safe and satisfactory alternative in senile and ill patients. In young patients, it is justifiable to amputate at a low level, but re-amputation is carried out if the wound does not heal. The aim in senile patients, however, is to achieve a successful primary operation; therefore a mid-thigh amputation is usually performed. Unintelligent and elderly patients do not easily adjust themselves to the use of a complicated artificial limb; for this reason some surgeons claim success with a Stokes-Gritti amputation which produces an end-bearing stump. [The leg is removed through the femoral condyles and the patella is fixed to the under-surface of the femur; weight-bearing on a pylon is begun in six weeks.]

In atherosclerosis, the arteries are brittle and their inner coats easily separate. The use of any form of tourniquet during amputation is therefore inadvisable for fear of precipitating further occlusion. In order that the skin flaps may be viable they are usually made of equal length, but for below-knee amputations some surgeons recommend that the posterior flap should be twice the length of the anterior. Occasionally, in infected cases, it may be advisable to leave the stump unsutured; the skin is prevented from contracting by strapping traction. The stump becomes conical, but is quite satisfactory. Sympathectomy may be undertaken to assist healing; the amount of chain removed depends on the site of amputation (*vide supra*).

### THE VASOMOTOR DISEASES

The arteries and arterioles supplying the skin are normally slightly constricted, but their tone is varied by central and peripheral stimuli. Thus a fall of blood pressure, or emotion, acting through the vasomotor centre and sympathetic nerves may initiate generalised vasospasm. Local stimuli include changes in temperature; cold has a constrictor and heat a dilator effect.

**The Raynaud Phenomenon.** This is characterised by attacks of what appears to be an exaggeration of vasoconstrictor response to either local or central stimuli. The attacks consist of pallor, numbness and mild pain usually affecting the skin of the digits, but occasionally also of the nose and ears, shortly followed by cyanosis and reflex hyperæmia (rubor, warmth and slight swelling). This spastic arterial occlusion may be due to either: (1) An excessive response to average stimuli; or (2) a normal constrictor response in an organically diseased artery (Fig. 509). Thus two varieties of the phenomenon occur: primary and secondary.

**The Primary Raynaud Phenomenon** (*Syn. Raynaud's Disease*). Vasoconstrictor attacks usually involve the hands and feet, although symptoms

primary and post-traumatic arterial occlusion. It is rarely indicated in atherosclerosis, but in suitable cases the insertion of a long "by-pass" graft, with side-to-side anastomosis above and below the block is a procedure of less magnitude giving good results.

### SURGERY OF NECROSIS AND GANGRENE

Every mild inflammation and injury is serious in patients with ischaemia; treatment must therefore not be delayed. Evacuation of pus and excision of sloughs must be undertaken early. Necrotic tendons separate extremely slowly and have to be removed.

Amputation may be necessary in cases of gangrene and in patients with



FIG. 508. Diabetic gangrene. In spite of widespread destruction more distal tissue remains alive. (Courtesy of Professor C. G. Rob.)

severe rest pain despite sympathectomy and reconstructive surgery. Small areas of dry superficial necrosis may be allowed to separate, but amputation is usually necessary in patients with more extensive gangrene. The possible value of direct arterial surgery in the younger patients has already been mentioned. In diabetic patients with gangrene, it is essential to differentiate those with atherosclerotic ischaemia from those with a good circulation in whom the gangrene is predominantly due to sepsis favoured by the diabetes (Fig. 508). In the latter group, the treatment is early drainage of pus, and local amputation, if necessary.

The level of amputation is selected after performing the clinical tests for ischaemia already described. Further help is obtained, when necessary, from a reflex-heating test and arteriography. Although resection must be performed through healthy tissues, every effort should be made to preserve as much of the limb as possible. If the foot is healthy, excision of gangrenous toes (or if necessary a transmetatarsal amputation) is often

successful for Raynaud's disease of the feet, but when performed for the disease in the hands relapse eventually occurs in about a third of the cases. *Secondary Group*.—The primary cause must be recognised and treated, but sympathectomy may also be of value in overcoming vasospasm.

**Acrocyanosis.** In acrocyanosis the distal parts of the limbs are constantly cold and blue, probably due to increased tone in the small arteries producing stagnation in the capillaries and venules. The history usually extends back to early youth. The sexes are affected more or less equally.



FIG. 510. Gangrene of the tips of the fingers in the primary Raynaud phenomenon. (Courtesy of Professor C. G. Rob.)

Raynaud's phenomenon does not occur. *Treatment*.—Sympathectomy is often effective, and is recommended if symptoms are sufficiently severe.

**Erythromelalgia.** This is a rare condition in which attacks of exceedingly painful vasodilatation are initiated by warmth. The patient often sleeps with the feet uncovered for fear of an attack. During attacks the hands or feet are found to be bright red and somewhat swollen. Erythromelalgia may be associated with atherosclerosis or gout. Sometimes it is followed by the onset of polycythaemia rubra vera and hypertension. *Treatment* is unsatisfactory, but aspirin (as required) is of marked benefit. If the disorder is associated with atherosclerosis, sympathectomy may be helpful.

#### THE EFFECTS OF COLD

**Pernio (Chilblain).** Pernio affects people whose skin is cooler than average in cold weather and warms up slowly after exposure. The lesions are most commonly found on the backs of the hands and on the ears, and



are more severe in the hands than the feet. Young girls are most commonly affected ; the disorder is rare in males. The attacks are often more frequent during menstruation, whereas during pregnancy remission may occur. On examination, there is no evidence of arterial disease. The condition is apparently a physiological fault in the arterial wall, but it is not known whether this is in the motor nerves or in the arterial muscle.

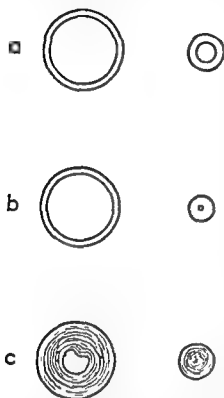


FIG. 500. Spastic occlusion. (a) Normal physiological contraction of a digital artery. (b) Excessive contraction (primary Raynaud phenomenon). (c) Normal contraction of a diseased artery (secondary Raynaud phenomenon). In (b) and (c) the arterial lumen is almost obliterated.

Attacks can often be induced by placing one or more extremities in cold water for a few minutes, especially if the main artery to the limb is occluded with a sphygmomanometer cuff. On restoring the blood flow, a good hyperæmic response occurs. Although gangrene is never extensive, necrosis of the tips of digits is common and sometimes reaches the distal interphalangeal joints (Fig. 510). Raynaud's phenomenon should only be considered a primary manifestation if it is long-standing and if there is no evidence of arterial disease.

#### The Secondary Raynaud Phenomenon.

This occurs in : (a) *Chronic Obliterative Arterial Diseases*.—In these diseases, in which there is already an impaired circulation, normal constrictor responses may result in Raynaud attacks. About one patient in thirty is so affected. Spasms are usually asymmetrical and most severe in the territory of the arteries most affected by the primary disease. The reflex hyperæmia test is found to be abnormal and arteriography shows partial or complete digital artery occlusion. (b) *Chronic Arterial Irritation*.—

Examples include : (1) The use of vibration tools, e.g. the pneumatic drill. The frequency of vibration which induces attacks is in the region of 3,000 per minute. (2) "Collagen" diseases, such as scleroderma, lupus erythematosus and polyarteritis nodosa. (3) Sympathetic nerve irritation, e.g. from a cervical rib. (4) Certain toxic states, e.g. ergot and lead poisoning.

**Treatment.** *Primary Group*.—Raynaud's disease is often controlled by wearing warm clothing or residing in a warm climate. The administration of Priscol for the necessary length of time is rarely tolerated by the patient. In severe progressive cases, sympathectomy should be performed (see under The Autonomic Nervous System). It is nearly always

**Frost-bite.** This condition is produced by temperatures below  $0^{\circ}$  C. It is due to slow-freezing of the tissues, in which the formation of ice crystals and the marked vasospasm lead to gangrene. At first the skin may only be pale or slightly yellow. In severe cases blistering and gangrene follow (Fig. 511). A red wheal borders the damaged zone. The raised epidermis of the blisters is much thicker than in the blisters of burns. Gangrene is often less extensive than the initial appearance suggests. The microscopic changes are similar to those of pernio. Most of the change occurs during the initial freezing, but thrombosis in the smaller arteries adds to the extent of the gangrene.

**Trench Foot.** Trench foot occurs at freezing point or just above ; it is due to prolonged cooling in the presence of moisture and immobility ; most of the damage occurs during thawing when there is a marked transudation of fluid. The legs are swollen and feel cold. At first they are pale and pulseless. On warming they become red and exceedingly painful—a stage which may last for several days. Fluid blebs form in the skin, together with intracutaneous hæmorrhages ; gangrene of the skin may follow. After recovery from the acute stages the patient may be left with a cold cyanotic limb and have symptoms of chronic ischæmia. Raynaud attacks may occur.

**Treatment of Frost-bite and Trench Foot.** *Slow* thawing is essential, and the part must not be rubbed. The limb itself is exposed but warmth is applied to the body. In trench foot, elevation is required to reduce the œdema. Anticoagulant drugs are recommended to prevent spread of the thrombosis. Sympathectomy may be necessary later to improve the circulation. The slow separation of gangrenous digits may be followed by trimming operations or skin grafting.

### ACUTE OCCLUSION OF PERIPHERAL ARTERIES

Acute peripheral arterial occlusion is a rare condition, but a limb may be lost by incorrect treatment in the early stages. The causes are : (1) *Trauma* (discussed later). (2) *Embolism*.—Acute arterial embolism may occur : (a) In certain forms of heart disease, as the result of detachment of an intracardiac thrombus or vegetation ; and (b) in atherosclerosis due to separation of clot or degenerate material. Secondary thrombosis around an embolus increases the extent of the ischæmia. The arteries most commonly involved in systemic arterial embolism are the femoral and the popliteal. Embolism of the upper limb is rare, and seldom causes gangrene owing to the adequate collateral circulation. A large embolus may obstruct the aorta at its bifurcation ; this condition is of great importance as embolectomy may be life-saving. (3) *Thrombosis*.—Acute arterial thrombosis may complicate any of the degenerative arterial diseases already described.

**The Effects of Sudden Occlusion.** The symptoms and signs vary with

are red and burning; they may become swollen and dusky, and may ulcerate. The condition is due to arteriolar spasm produced by repeated cooling; thickening of the intima is seen on microscopy, with infiltration of the vessel with lymphocytes and monocytes. *Erythrocyanosis frigida* is a type of pernio most commonly seen in the lower part of legs and ankles. Ulceration may occur, and a degree of lymphædema is often present. In



(a)

FIG. 511. (a) Frost-bite in an airman. All the fingers of the right hand and the left little finger are affected and there is œdema of the right hand. (b) The same patient after three weeks, showing gangrene of the right little and ring fingers.



(b)

addition to the changes noted in simple pernio, the subcutaneous tissues are also chronically inflamed and giant cells may be present. *Erythema induratum* is a similar condition seen in patients with active tuberculosis although tubercle bacilli have never been identified in the skin lesions. *Treatment*.—This is mainly preventive and warm clothing should always be worn. Vasodilators, such as Priscol, are of benefit in some cases. Sympathectomy is not indicated. In erythema induratum, the associated tuberculosis is appropriately treated.

## THE TREATMENT OF ACUTE OCCLUSION

Early admission to hospital is necessary, but whilst the patient is awaiting transfer it is most important that the limb should be kept cool, and elevation avoided.

**Conservative Measures.** Reduction of the metabolic need may be achieved by keeping the limb cool. The limb should not be completely exposed, however, as this increases any spasm that may be present; it should on the contrary be lightly wrapped in a thin layer of sterile cotton-wool or gauze. The limb should be kept straight; although slight dependency may assist the collateral supply it also tends to increase œdema. The limb may be rested on a soft splint. In order to encourage the collateral arteries to dilate and to reduce venospasm, heat should be applied to the *body* and hypotension corrected by transfusion. Alcohol is both a sedative and a vasodilator and may be freely administered. Intra-arterial papaverine (32 to 65 mg. or  $\frac{1}{2}$  to 1 gr.) and intravenous hypertonic saline (200 ml. of a 2 per cent. solution) have been recommended. Pethidine, being an antispasmodic, is preferable to morphia for the relief of pain. In the acute stage, reflex heating is as effective as sympathetic interruption; furthermore sympathetic block is inadvisable when anticoagulant drugs are being given for there is a risk of hæmorrhage on needling. Synovial adhesions are prevented by putting each joint through its whole range of movement daily. Spread of thrombosis should be discouraged by giving the anticoagulant heparin (Chap. 75). Heparin is also an antispasmodic and assists in relieving ischæmic pain. Even if embolectomy may become necessary later, heparin may safely be given early as its effects can be rapidly neutralised by the intravenous injection of 1 per cent. protamine sulphate. After a few days, anticoagulant therapy may be continued using one of the coumarol derivatives. Systemic antibiotics should be given to reduce the risk of secondary infection of the ischæmic limb.

**Embolectomy.** Popliteal and brachial emboli usually do well when treated conservatively. Operation is advisable for iliac and femoral emboli, for even if the limb survives severe symptoms of ischæmia persist. Aortic emboli are usually fatal unless treated by immediate embolectomy. As a general rule, embolectomy is indicated for all peripheral emboli if there are no signs of recovery within four hours from the onset of symptoms. It often happens that the patient with acute arterial occlusion is not admitted to hospital until signs of recovery are apparent in the limb. If, on the other hand, a limb has been ischæmic for more than ten hours, it is probably better to administer anticoagulant therapy, elevate the part to prevent œdema and encourage movement in the limb.

**The Operation.** General anæsthesia is preferable, but local infiltration or regional block may be satisfactory. The artery is isolated and two

the severity of the occlusion. The following description applies to limbs with complete ischaemia. Pain, although usually severe and early may sometimes be slight, or delayed. The limb is at first white and cold. The arterial pulses are absent below the site of occlusion, but may be increased proximally. The veins are collapsed. The site of occlusion is occasionally recognisable as a palpable swelling in the line of the artery. Nerve conduction ceases after about half an hour; the limb thus becomes paralysed and anaesthetic. Sometimes dying muscle contracts and produces a flexion deformity. The skin soon becomes purple-blue, and white areas form between the coloured zones (Bier's spots). Later collections of fluid may be seen in the skin. The gangrene is almost always moist, but later a well-defined line of demarcation usually develops. Shock may be severe and the resultant fall of blood pressure increases the ischaemia. The site of block may be judged from the peripheral pulses; it is most likely to be at a vessel bifurcation. The temperature gradient of the limb shows a maximum drop just below the level of obstruction. Absence of arterial pulsation at this point may be confirmed by oscillometry. Arteriography is not recommended in acute occlusion in case the limb is jeopardised by further arterial spasm.

**Degree of Recovery.** This depends in the first instance on the collateral response. Later it depends on the degree of recanalisation in the clotted vessel. If the limb survives, chronic arterial insufficiency often remains. The first evidence of recovery is a feeling of "creeping warmth" in the part. With an adequate collateral blood supply the distal pulse in the arm may return in three or four days, but in the foot it may not be felt for as long as three weeks. Residual signs include those already described under chronic ischaemia, but there may also be fibrosis of muscle (Volkmann's ischaemic contracture), or patchy anaesthesia, often of the "glove and stocking" type. As the acute episode is commonly complicated by thrombosis in the deep veins, the limb may remain oedematous.

**Differential Diagnosis.** The differentiation of embolism from thrombosis is not always easy. The presence of heart disease liable to produce emboli, e.g. myocardial infarction, bacterial endocarditis or auricular fibrillation, is in favour of embolism, whereas a previous history of ischaemic symptoms in the limb suggests thrombosis. A dissecting aneurysm of the aorta may simulate aortic embolism.

**Prognosis.** The outlook depends on the patient's general condition, the site of the embolus, and the adequacy of the collaterals circulation. It also depends on the early management, for if the limb is kept cool (i.e. its metabolism is reduced) its chance of survival is improved. In acute occlusion there is little time for the collateral system to expand, and gangrene, usually of the moist type, results in nearly 50 per cent. of cases. The mortality rate is fairly high, partly as a result of associated heart disease.

### THE TREATMENT OF ACUTE OCCLUSION

Early admission to hospital is necessary, but whilst the patient is awaiting transfer it is most important that the limb should be kept cool, and elevation avoided.

**Conservative Measures.** Reduction of the metabolic need may be achieved by keeping the limb cool. The limb should not be completely exposed, however, as this increases any spasm that may be present; it should on the contrary be lightly wrapped in a thin layer of sterile cotton-wool or gauze. The limb should be kept straight; although slight dependency may assist the collateral supply it also tends to increase œdema. The limb may be rested on a soft splint. In order to encourage the collateral arteries to dilate and to reduce venospasm, heat should be applied to the *body* and hypotension corrected by transfusion. Alcohol is both a sedative and a vasodilator and may be freely administered. Intra-arterial papaverine (32 to 65 mg. or  $\frac{1}{2}$  to 1 gr.) and intravenous hypertonic saline (200 ml. of a 2 per cent. solution) have been recommended. Pethidine, being an antispasmodic, is preferable to morphia for the relief of pain. In the acute stage, reflex heating is as effective as sympathetic interruption; furthermore sympathetic block is inadvisable when anticoagulant drugs are being given for there is a risk of hæmorrhage on needling. Synovial adhesions are prevented by putting each joint through its whole range of movement daily. Spread of thrombosis should be discouraged by giving the anticoagulant heparin (Chap. 75). Heparin is also an antispasmodic and assists in relieving ischæmic pain. Even if embolectomy may become necessary later, heparin may safely be given early as its effects can be rapidly neutralised by the intravenous injection of 1 per cent. protamine sulphate. After a few days, anticoagulant therapy may be continued using one of the coumarol derivatives. Systemic antibiotics should be given to reduce the risk of secondary infection of the ischæmic limb.

**Embolectomy.** Popliteal and brachial emboli usually do well when treated conservatively. Operation is advisable for iliac and femoral emboli, for even if the limb survives severe symptoms of ischæmia persist. Aortic emboli are usually fatal unless treated by immediate embolectomy. As a general rule, embolectomy is indicated for all peripheral emboli if there are no signs of recovery within four hours from the onset of symptoms. It often happens that the patient with acute arterial occlusion is not admitted to hospital until signs of recovery are apparent in the limb. If, on the other hand, a limb has been ischæmic for more than ten hours, it is probably better to administer anticoagulant therapy, elevate the part to prevent œdema and encourage movement in the limb.

**The Operation.** General anaesthesia is preferable, but local infiltration or regional block may be satisfactory. The artery is isolated and two

the severity of the occlusion. The following description applies to limbs with complete ischaemia. Pain, although usually severe and early may sometimes be slight, or delayed. The limb is at first white and cold. The arterial pulses are absent below the site of occlusion, but may be increased proximally. The veins are collapsed. The site of occlusion is occasionally recognisable as a palpable swelling in the line of the artery. Nerve conduction ceases after about half an hour; the limb thus becomes paralysed and anæsthetic. Sometimes dying muscle contracts and produces a flexion deformity. The skin soon becomes purple-blue, and white areas form between the coloured zones (Bier's spots). Later collections of fluid may be seen in the skin. The gangrene is almost always moist, but later a well-defined line of demarcation usually develops. Shock may be severe and the resultant fall of blood pressure increases the ischaemia. The site of block may be judged from the peripheral pulses; it is most likely to be at a vessel bifurcation. The temperature gradient of the limb shows a maximum drop just below the level of obstruction. Absence of arterial pulsation at this point may be confirmed by oscillometry. Arteriography is not recommended in acute occlusion in case the limb is jeopardised by further arterial spasm.

**Degree of Recovery.** This depends in the first instance on the collateral response. Later it depends on the degree of recanalisation in the clotted vessel. If the limb survives, chronic arterial insufficiency often remains. The first evidence of recovery is a feeling of "creeping warmth" in the part. With an adequate collateral blood supply the distal pulse in the arm may return in three or four days, but in the foot it may not be felt for as long as three weeks. Residual signs include those already described under chronic ischaemia, but there may also be fibrosis of muscle (Volkmann's ischaemic contracture), or patchy anæsthesia, often of the "glove and stocking" type. As the acute episode is commonly complicated by thrombosis in the deep veins, the limb may remain oedematous.

**Differential Diagnosis.** The differentiation of embolism from thrombosis is not always easy. The presence of heart disease liable to produce emboli, e.g. myocardial infarction, bacterial endocarditis or auricular fibrillation, is in favour of embolism, whereas a previous history of ischaemic symptoms in the limb suggests thrombosis. A dissecting aneurysm of the aorta may simulate aortic embolism.

**Prognosis.** The outlook depends on the patient's general condition, the site of the embolus, and the adequacy of the collaterals circulation. It also depends on the early management, for if the limb is kept cool (i.e. its metabolism is reduced) its chance of survival is improved. In acute occlusion there is little time for the collateral system to expand, and gangrene, usually of the moist type, results in nearly 50 per cent. of cases. The mortality rate is fairly high, partly as a result of associated heart disease.

## THE TREATMENT OF ACUTE OCCLUSION

Early admission to hospital is necessary, but whilst the patient is awaiting transfer it is most important that the limb should be kept cool, and elevation avoided.

**Conservative Measures.** Reduction of the metabolic need may be achieved by keeping the limb cool. The limb should not be completely exposed, however, as this increases any spasm that may be present; it should on the contrary be lightly wrapped in a thin layer of sterile cotton-wool or gauze. The limb should be kept straight; although slight dependency may assist the collateral supply it also tends to increase œdema. The limb may be rested on a soft splint. In order to encourage the collateral arteries to dilate and to reduce venospasm, heat should be applied to the *body* and hypotension corrected by transfusion. Alcohol is both a sedative and a vasodilator and may be freely administered. Intra-arterial papaverine (32 to 65 mg. or  $\frac{1}{4}$  to 1 gr.) and intravenous hypertonic saline (200 ml. of a 2 per cent. solution) have been recommended. Pethidine, being an antispasmodic, is preferable to morphia for the relief of pain. In the acute stage, reflex heating is as effective as sympathetic interruption; furthermore sympathetic block is inadvisable when anticoagulant drugs are being given for there is a risk of hæmorrhage on needling. Synovial adhesions are prevented by putting each joint through its whole range of movement daily. Spread of thrombosis should be discouraged by giving the anticoagulant heparin (Chap. 75). Heparin is also an antispasmodic and assists in relieving ischæmic pain. Even if embolectomy may become necessary later, heparin may safely be given early as its effects can be rapidly neutralised by the intravenous injection of 1 per cent. protamine sulphate. After a few days, anticoagulant therapy may be continued using one of the coumarol derivatives. Systemic antibiotics should be given to reduce the risk of secondary infection of the ischæmic limb.

**Embolectomy.** Popliteal and brachial emboli usually do well when treated conservatively. Operation is advisable for iliac and femoral emboli, for even if the limb survives severe symptoms of ischæmia persist. Aortic emboli are usually fatal unless treated by immediate embolectomy. As a general rule, embolectomy is indicated for all peripheral emboli if there are no signs of recovery within four hours from the onset of symptoms. It often happens that the patient with acute arterial occlusion is not admitted to hospital until signs of recovery are apparent in the limb. If, on the other hand, a limb has been ischæmic for more than ten hours, it is probably better to administer anticoagulant therapy, elevate the part to prevent œdema and encourage movement in the limb.

**The Operation.** General anæsthesia is preferable, but local infiltration or regional block may be satisfactory. The artery is isolated and two



tapes placed around it. If it is in spasm the local application of 2.5 per cent. papaverine sulphate should be tried. The upper tape is drawn upwards until the artery is thereby occluded and a vertical incision is made in the vessel. The clot is sucked away, and a ureteric catheter is passed up and down to make sure that removal is complete; the artery is then closed with silk sutures. Anticoagulant therapy is begun, or re-established, at once; the first injection of heparin is given as soon as the artery is sutured.

### ARTERIAL ANEURYSMS

Arterial aneurysms (Fig. 512) are considerably more common in men than in women. A *true arterial aneurysm* is a localised dilatation of an artery. One or more arterial layers are present in the wall of the aneurysm. A *false arterial aneurysm* is a cavity containing blood or clot in direct communication with the lumen of an artery. The wall of the aneurysm is composed of condensed connective tissue only. There are *fusiform aneurysms* (spindle-shaped) and *saccular aneurysms* (communicating with the sides of arteries). Many aneurysms are a combination of the two types; in the abdomen, for example, there may be fusiform bulging of the aorta in continuity with a saccular aneurysm of the common iliac artery. A *dissecting aneurysm* is due to rupture of the inner coats of the vessel allowing blood to force its way between the

external elastic lamina and the adventitia. This splitting may extend distally throughout the aorta and its branches. Death usually follows within twenty-four hours. Very rarely the aneurysm may reopen into the aorta further down ("double-barrel" aorta), thus allowing some of the blood flow to continue. Such cases occasionally recover. *Cirroid aneurysms* are more properly classified as malformations, for they consist of abnormal formations of blood vessels, often with communications between arteries and veins.

**Aetiology.** Congenital aneurysms are common within the cranial cavity but rare elsewhere (Chap. 56). They are the commonest cause of sub-arachnoid hæmorrhage. A true aneurysm may follow acute contusion of an artery, particularly in the presence of atherosclerosis. False aneurysms may result from partial division of an artery if

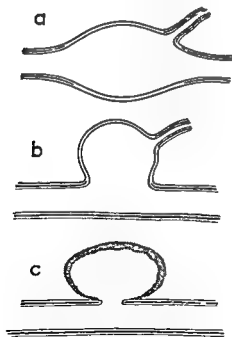


FIG. 512. Arterial aneurysms. (a) Fusiform. (b) Saccular. (c) False; the sac wall consists of condensed fibrous tissue and organised blood clot.

hemorrhage is confined within the tissue planes (Fig. 513). Continuous trauma may lead to aneurysm formation, e.g. a subclavian aneurysm in a patient with a cervical rib. Atherosclerosis, with medial weakening, is in many countries the commonest cause of aneurysms. Some of the patients are diabetic, whereas others are hypertensive, for high blood pressure favours the development of aneurysms. Atherosclerotic aneurysms occur mainly in the aorta (Fig. 514) and larger arteries, especially where a vessel is unsupported by muscle and subject to bending. Dissecting aneurysm

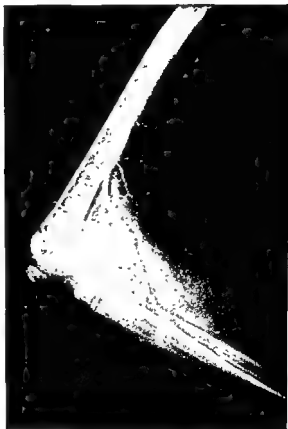


FIG. 513. Traumatic aneurysm of the popliteal artery in a boy of seven. The injury was produced by a piece of wire. (Courtesy of Mr. R. S. Murley.)

of the aorta usually follows mucinoid necrosis of the media, but it may be due to rupture through a plaque of atheroma. Septic emboli, as may occur in bacterial endocarditis, may produce mycotic aneurysms. This type is more common since the introduction of the treatment of endocarditis with antibiotics, for lives are saved or prolonged so that aneurysms have a chance to develop. Small aneurysms occasionally form in the walls of tuberculous cavities. Tertiary syphilis used to be a frequent cause of aneurysm. The thoracic aorta is the usual site, but the medium-sized arteries are sometimes involved (Fig. 515). Polyarteritis nodosa may present as a case of peripheral aneurysm.

**Clinical Features.** An aneurysm lies in the line of an artery and its pulsation is expansile. Unless the sac is consolidated (*vide infra*) the swelling is reducible, and when it is compressed, a temporary *bradycardia* may be noticed. On release, the aneurysm refills with the next few heart beats. An aneurysm expands on compression of the artery distally. A systolic bruit may be heard over an arterial aneurysm on auscultation, but a thrill is rarely felt in the absence of a venous fistula (*vide infra*).



FIG. 514. Aortogram showing an aneurysm of the abdominal aorta. (Courtesy of Professor C. G. Rob. X-ray by Dr. David Sutton.)

The more confined the space in which an aneurysm lies, the greater is the number of extrinsic signs produced. Pressure on nerves may lead to pain and muscular weakness, and venous compression produces oedema. Bone may be eroded when other structures are spared; for instance, radiography of the lumbar spine, in the presence of an abdominal aneurysm may show considerable destruction of the intervertebral bodies, but preservation of the intervertebral discs. Signs of peripheral ischaemia may develop, either from obstruction to collateral vessels or from release of clot from within the aneurysm. The extrinsic effects of an aneurysm are dependent on its relationship to other structures. Such effects can

be inferred from a knowledge of regional anatomy and a description of individual aneurysms of the limbs need not therefore be given. Intra-cranial and intrathoracic aneurysms are considered in their appropriate sections.

**Complications.** *Thrombosis.*—Both stagnation of the blood stream and atheroma, which is common in an aneurysmal sac, contribute to the development of thrombosis. The clot is conspicuously laminated, the older layers being lighter in colour than the younger ones. Parts of the thrombus may become calcified.

Some of it may become detached and lead to embolism.

Thrombosis of an aneurysm may occasionally lead to spontaneous cure ("consolidation").

*Rupture.*—Hæmorrhage may occur into a hollow space or viscus, e.g. the trachea or peritoneal cavity, or may be confined within the tissue planes ("suffusion").

*Infection.*—Mycotic aneurysms have already been described. Secondary infection in an initially sterile aneurysm is very rare. If it occurs, septicæmia, or secondary hæmorrhage from rupture of the aneurysm may result. *Peripheral ischæmia* has already been mentioned.

**Prognosis.** In aortic aneurysms the expectation of life is less than three years. Many peripheral arteriosclerotic

aneurysms become complicated, e.g. 50 per cent. of popliteal aneurysms result in gangrene. Fifty per cent. of patients with dissecting aneurysms of the aorta die within twenty-four hours and very few survive long. Spontaneous cure sometimes occurs with saccular aneurysms.

**Differential Diagnosis.** An aneurysm has sometimes been incised in mistake for an acute abscess. All clinical swellings should be examined for pulsation and a differentiation made between the expansile and transmitted variety. Any swelling overlying an artery pulsates, but the impulse of an aneurysm is always *expansile*. In thin people, the normal abdominal aorta may easily be felt, and patients not infrequently seek advice because of its pulsation. The absence of any marked lateral pulsation usually prevents confusion. Acute leakage from an aortic



FIG. 515. Syphilitic aneurysm of the brachial artery. (Courtesy of Professor C. G. Rob.)

**Clinical Features.** An aneurysm lies in the line of an artery and its pulsation is expansile. Unless the sac is consolidated (*vide infra*) the swelling is reducible, and when it is compressed, a temporary *bradycardia* may be noticed. On release, the aneurysm refills with the next few heart beats. An aneurysm expands on compression of the artery distally. A systolic bruit may be heard over an arterial aneurysm on auscultation, but a thrill is rarely felt in the absence of a venous fistula (*vide infra*).



FIG. 514. Aortogram showing an aneurysm of the abdominal aorta. (Courtesy of Professor C. G. Rob. X-ray by Dr. David Sutton.)

The more confined the space in which an aneurysm lies, the greater is the number of extrinsic signs produced. Pressure on nerves may lead to pain and muscular weakness, and venous compression produces œdema. Bone may be eroded when other structures are spared; for instance, radiography of the lumbar spine, in the presence of an abdominal aneurysm may show considerable destruction of the intervertebral bodies, but preservation of the intervertebral discs. Signs of peripheral ischæmia may develop, either from obstruction to collateral vessels or from release of clot from within the aneurysm. The extrinsic effects of an aneurysm are dependent on its relationship to other structures. Such effects can

by gangrene. Hunter's ligature is the best known, and is placed proximal to the last major branch which the artery gives off before joining the aneurysm. The collateral circulation in the limb allows some blood to reach the aneurysm through this branch. The insertion of lengths of stainless steel wire into the sac, or wrapping layers of cellophane around the aneurysm may induce thrombosis ; these operations are still of use in



FIG. 518. The same case as Fig. 514, after excision of the aneurysm and grafting the aorta. (Courtesy of Professor C. G. Rob. X-ray by Dr. David Sutton.)

frail patients who are unable to withstand more major procedures. The former method is probably more effective and can be carried out through a needle. The latter method is more valuable in preventing rupture because of the resulting peri-arteritis. (2) *Endo-aneurysmorrhaphy*.—An irremovable aneurysm may be obliterated by opening the sac in a bloodless field (i.e. with the use of a tourniquet), turning out the clot, and sewing up the orifices of all communicating vessels from the inside. The sac is left behind. This operation was devised by Matas who also performed a reconstructive operation. In this procedure the sac is opened as before, and the arterial orifices, except for the main arterial openings, are occluded.

aneurysm sometimes presents as a case of "acute abdomen." Dissecting aneurysms may also present in this way. Vascular tumours, e.g. congenital arteriovenous fistulas and bone sarcomas, may show expansile pulsation, but bone sarcomas can usually be diagnosed on their radiological appearances.

**Investigations.** After a full history has been taken and a general medical examination made, the following investigations are carried out :  
**The Wassermann Reaction.** **Plain Radiography.**—This may show the outline of an aneurysm and sometimes calcification of its wall. **Arteriography** is necessary before any surgical procedure is planned. The size of an aneurysm may not be fully revealed, owing to thrombosis in the sac, but an impression of its size and position is obtained, and the extent of the collateral circulation visualised.

**Treatment.** General measures include an attempt to reduce any existing hypertension and to prevent strenuous exertion. Specific therapy may be required for associated or causal diseases, e.g. diabetes mellitus, syphilis and bacterial endocarditis.

**Surgical Treatment.** The present tendency is towards early operation for aneurysms of the main limb vessels. Except in the case of an unimportant artery, excision of an aneurysm must usually be followed by a restorative procedure on the parent vessel. In the past, gangrene commonly followed such treatment, but recent advances in arterial reconstruction have lowered this risk very considerably. Untreated aneurysms of the abdominal aorta have a poor prognosis ; therefore, especially in young patients, excision has to be considered (Fig. 510). Although excision of an aneurysm is the treatment of choice, it is impossible if : (1) The wall of the aneurysm is too adherent to surrounding structures ; (2) vital arterial branches, unsuitable for grafting, must necessarily be divided (e.g. in the upper abdominal aorta) ; or (3) the patient is unfit for such a serious operation. The surgical procedures are divided into two groups :

(a) *When Excision is Possible.* (1) *Excision and Arterial Suture.*—End-to-end suture of the arterial wall is occasionally possible. (2) *Excision and Ligation.*—This is possible in the case of small and unimportant arteries. (3) *Excision and Grafting.*—Arterial continuity may be restored using an arterial graft (*vide infra*). Such grafts continue to function satisfactorily in a large proportion of cases. With regard to the abdominal aorta, the aneurysm must be resectable below the coeliac axis and the renal arteries, although distally a graft can be joined to the common iliac arteries, or their immediate branches, with sacrifice of the inferior mesenteric artery.

(b) *When Excision is Impossible.* (1) *Attempts to Promote Thrombosis in the Sac.*—Reduction in blood flow through an aneurysm encourages spontaneous thrombosis within the sac. A variety of arterial ligatures at different sites in relation to the aneurysm have been used for this purpose in the past, but they often failed to cure the aneurysm or were followed

Arteriovenous fistulas have occasionally followed operations during which large vascular pedicles have been ligated *en masse*.

**Clinical Features of Arteriovenous Fistulas.** An arteriovenous fistula may pass unrecognised for some time, although in the affected part of the body the skin is warm and the veins are distended. In the case of a varicose aneurysm an expansile swelling may be evident. A continuous thrill may be felt and a continuous ("bee-in-a-bag") hum may be heard on auscultation. Slowing of the pulse may follow digital compression of the tissues over the fistula or of the artery immediately proximal to it ("bradycardiac response"). The peripheral pulses are often weak distal to the fistula and sometimes abnormally strong proximal to it. The pulse pressure is high and there may be a "water-hammer" pulse. There is often a tachycardia and the heart may be enlarged. The oxygen content of the venous blood is high and may reach arterial levels. On arteriography, an arteriovenous fistula is rarely visible, but early venous filling is usually noticeable.

**Treatment. Congenital Fistulas.** Disease which is fairly well localised may be left alone unless cardiac complications develop or gangrene is feared. When the fistulas are limited to the more distal parts of a limb amputation may be indicated. More proximal fistulas present many problems. The operations for traumatic fistulas cannot easily be applied; treatment may have to be limited to partial excision of the diseased tissue and tying the enlarged vessels that supply it; each case, however, has to be considered separately. When the problem is simply that of the difference in length between the two legs, the shoe may be raised on the appropriate side.

**Traumatic Fistula.**—Surgery is indicated if there is peripheral ischaemia or increasing strain on the heart. Arterial ligation above a fistula may lead to gangrene, for the limb then "bleeds into the vein." When possible, the fistula is divided and the artery and vein are repaired. Otherwise, if the artery is important a graft may be inserted. Continuity in the vein should also be restored. In other cases, when the vessels can be safely sacrificed, a quadruple ligature (i.e. ligation of both artery and vein above and below the fistula) should be performed. Following successful surgery the cardiac and circulatory abnormalities usually recover.



FIG. 518. Arteriovenous fistula of the right calf, producing considerable swelling. (Courtesy of Professor C. G. Rob.)



Some of the sac is then dissected free and stitched longitudinally thus restoring the arterial channel. The disadvantage of both these methods is their high recurrence rate.

### ARTERIOVENOUS FISTULAS

An arteriovenous fistula (Fig. 517) is an abnormal communication between an artery and its companion vein. The fistula acts as a "short-circuit" through which the vein returns to the general circulation a

proportion of the blood destined for the periphery. This may result in: (1) An increase in the blood volume and cardiac output; and (2) ischæmia at the periphery, sometimes with gangrene.

An *aneurysmal varix* is an arteriovenous fistula in which there is a direct communication between an artery and a vein. It is so named because the vein enlarges to become a thick-walled varix which pulsates. A *varicose aneurysm* is an indirect arteriovenous fistula with an intervening sac (false aneurysm) between the openings of the artery and vein.

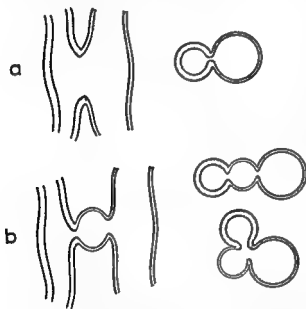


FIG. 517. Arteriovenous fistula. (a) Aneurysmal varix (direct arteriovenous fistula). (b) Varicose aneurysm (indirect arteriovenous fistula).

The term varicose applies to the dilatation of the vein.

**Congenital Arteriovenous Fistulas.** This rare abnormality leads to an increase in the vascularity of a limb and is usually associated with a rise in local temperature. The veins are engorged and may pulsate. Multiple fistulas are usually present. If the disease is in the growing end of a bone, the length of the limb may be altered; growth is usually stimulated, but occasionally it is retarded. The skin and subcutaneous tissues are often spongy, with enlargement of the smaller veins, and the girth of the limb is increased (Fig. 518).

**Traumatic Arteriovenous Fistula.** An arteriovenous fistula may follow injury to an artery and its companion vein. The most common arteries to be involved are the femoral, the popliteal and the internal carotid. The latter, in such cases, communicates in the neck with the internal jugular vein, or in the cranium with the cavernous sinus. A traumatic arteriovenous fistula may result from either a small open wound or a closed injury. The presence of a large hæmatoma, however, acts as a protection, for the increased tension in the tissues obstructs the vein.

A persistent tachycardia is also a sign of importance, for other causes being excluded, it may be due to an unrecognised arteriovenous fistula.

**Treatment.** *Hæmorrhage* should be controlled by local pressure on the wound, pressure on the artery above and below, or the direct application of artery forceps. A tourniquet is difficult to apply sufficiently tightly without causing damage to soft tissues, and when applied too loosely the resulting congestion encourages venous bleeding. Furthermore, it may become loose so that fresh hæmorrhage develops, possibly concealed under the blankets; or it may be left on too long so that amputation becomes necessary. *A tourniquet should only be used to prevent hæmorrhage from a limb that is so badly damaged that amputation is inevitable.* It should only be applied over a bony structure, a minimum pressure is thereby exerted on soft tissues. It must be observed continually and released every fifteen minutes. In cases of severe bleeding, oxygen and morphia help to allay restlessness. The circulatory volume must be adequately restored by transfusion; depletion of blood volume does not help to stop bleeding. The management of an acutely ischæmic limb has already been given.

*Immediate operative treatment* is essential in cases of severe external hæmorrhage or when the blood supply of the limb has been cut off, i.e. it is cold, pale and pulseless. Surgery is otherwise to be avoided, for if reconstructive facilities are not available the main artery may have to be tied, with the risk of gangrene. Suspected cases of arterial injury should be kept under observation in a specialised department in case the circulation of the limb deteriorates. The various types of injury are treated as follows: *Spasm*.—If there is a fracture it is reduced. If ischæmic signs persist, the artery must be explored. Any likely cause of irritation, such as a compressing hæmatoma, or fragment of bone is removed. The local application of 2·5 per cent. papaverine sulphate solution to the wall of an artery for ten minutes usually causes it to relax. Excision and grafting have sometimes been performed as a last resort with success. *Contusion and Partial Division*.—Contusion may lead to delayed rupture of an artery if untreated. In partial division of an artery, suture may be impossible. In both these types of injury, if the artery is vital, a musculofascial barrier may be made over it to encourage the formation of a false aneurysm. If facilities are available, however, it is better to excise the damaged segment of the artery and treat it as a completely divided artery (*vide infra*). *Complete Arterial Division*.—Axial (end-to-end) anastomosis is rarely possible owing to retraction of the ends of the artery. Arterial ligation is performed if the vessel is unimportant. In ischæmia, or when a large artery is damaged, an arterial graft should be inserted if available. Failing this, a vein graft is a good substitute and the external saphenous or external jugular veins make suitable material (*vide infra*). Unfortunately, some patients with divided arteries are suffering from such severe multiple injuries that they are unfit for prolonged operations. In such cases the temporary use of a plastic tube is permissible, but the patient

## ARTERIAL TRAUMA

Arterial trauma is rare in civilian practice, but in the last war approximately 1 per cent. of wounds of the extremities were associated with injury to a major vessel. An artery may be injured in closed or open wounds. Usually there is severe damage to other structures (e.g. the bones). Puncture wounds and incised wounds in the body flexures may, however, result in a predominantly arterial lesion. Butchers, for instance, have on occasion divided the femoral artery whilst cutting hanging meat.

**Types of Injury.** *Arterial Spasm.* Spasm of a major peripheral vessel is rare, but may complicate fractures and high velocity missile wounds. It may last for hours or days. Although it is rarely followed by gangrene it may lead to Volkman's ischaemic contracture. *Contusion* of the wall of an artery may be followed by thrombosis, aneurysm formation or delayed rupture of the artery, especially if it is atherosclerotic, or wound sepsis develops. *Partial division* leads to severe haemorrhage as the arterial muscle cannot retract effectively. Since soft tissues may plug the wound over the injured artery, haemorrhage is often internal and leads to considerable swelling. Although often absent at first, pulsation develops and the swelling is then termed a "pulsating haematoma." Sufficient arterial blood usually reaches the periphery in such circumstances for the limb to retain its vitality. After some weeks, condensation of connective tissue surrounding a pulsating haematoma results in the formation of a false aneurysm. *Complete division* of an artery is followed by shrinkage of the elastic tissue, retraction of the muscle coat and infolding of the intima, so that there may be a spontaneous arrest of haemorrhage. If the artery is anchored by one of its branches this mechanism may be prevented. *Arteriovenous Fistula.*—This condition has already been discussed.

**Effects of Arterial Injury.** *Haemorrhage* may be either internal or external, and has already been discussed. *Ischaemia.*—The development of gangrene after an arterial injury depends on: (1) The particular vessel damaged; (2) whether or not the collateral system is also damaged; (3) the patient's general condition. Lesser degrees of ischaemia lead to muscle degeneration and fibrosis (see under Volkmann's contracture), and symptoms of chronic ischaemia may later develop. Acute necrosis of muscle may be followed by aerobic or anaerobic infections, e.g. gas gangrene.

**Diagnosis.** Arterial injury is diagnosed by: (1) The presence of visible arterial bleeding, or bleeding out of proportion to the size of the wound; and (2) signs of peripheral ischaemia. An arterial lesion should also be suspected when there is undue swelling in a wound; the diagnosis is certain if expansile pulsation is present. Auscultation near a wound is of value, for a systolic murmur can often be heard when an artery is damaged.

*Plastic grafts* may be constructed of cloth, e.g. orlon, nylon or terylene, or be made of polyvinyl alcohol sponge; doubtless other materials will shortly be available. An artery bank is unnecessary and this type of graft is useful in the reconstruction of the aorta or iliac arteries since it can be "made-to-measure."

**Technique.** A graft is chosen slightly narrower in diameter than the recipient vessel, and shorter in length than the gap to be bridged. This is necessary, for the graft becomes stretched when the circulation is restored, and hæmorrhage at the suture lines is easy to control if the graft is taut. If suturing is not to be hampered by the adherence of adventitia, the latter must be removed from the ends of both recipient and donor vessels. The anastomosis is carried out with silk (gauge 00000) lubricated with paraffin and mounted on atraumatic needles; the operation site is frequently moistened with sodium citrate solution. The ends of the vessels are anchored by two sutures, and a continuous everting stitch is used to complete the junction. Whereas anticoagulants are not essential in grafting vessels which are not diseased (e.g. in trauma), most surgeons prefer to give heparin post-operatively until the distal pulses can be felt. When heparin is used the wound is left open so that a deep hæmatoma does not form and compress the graft or the collateral vessels, or cause infection of the wound. Delayed primary suture is performed on the third day and the large blood loss that occurs with the use of anti-coagulants is made good by transfusion.

must be heparinised. After two days' resuscitation the artery can then be grafted. If, owing to adverse circumstances, arterial ligation is unavoidable, neither sympathectomy nor tying the companion vein has been shown to reduce the risk of gangrene.

**Other Arterial Injuries.** *Plaster Casts.*—Little pressure is required to obliterate the small vessels supplying muscles, and even large arteries may go into spasm as a result of swelling in a limb beneath a constricting plaster. Plasters should be removed immediately any signs of vascular impairment occur. The release of hæmatomas and exploration of the main vessels may also be necessary.

The unintentional injection of *Pentothal* into the brachial artery has occasionally led to gangrene of the hand from acute arterial thrombosis. If such an accident is suspected the needle should be left in place and 5 ml. of 2 per cent. procaine injected into the artery, together with 10,000 units of heparin. A brachial plexus block is then performed, after which anticoagulant drugs are given.

### ARTERY GRAFTING

The grafting of arteries has been practised since the first human graft (a vein) was inserted into the popliteal artery in 1912. It has, however, become widely popular only since the last war, and although satisfactory techniques have been evolved a full assessment of results cannot yet be made. A certain number of grafts thrombose. Rupture of a graft is rare. Grafting may be of value in the following conditions: (a) Congenital anomalies, such as coarctation of the aorta; (b) arterial trauma; (c) aneurysms; (d) important vessels adherent to growths; (e) chronic arterial occlusion.

**Materials in Use.** It appears that whether a graft remains patent or not depends more on the condition for which the operation is performed than on the type of graft inserted. *Tissue grafts* are either autogenous, i.e., taken from the same individual, homogenous (taken from another person), or heterogenous (from an animal). They may be arterial or venous. *Artery Grafts.*—These are easier to insert than veins. Homogenous grafts (homografts), which must be obtained aseptically immediately after death, may be stored up to six weeks in saline at 4° C. If a freezing technique is used they may be kept for years. Although homografts do not survive they form a scaffold for the natural rebuilding of the vessel. They are recommended for the replacement of medium-sized arteries such as the popliteal, femoral and subclavian. *Vein Grafts.*—Autografts are always available, e.g. the external jugular and long saphenous veins, and they survive transplantation. They are, however, difficult to suture, and in atherosclerosis are likely to thrombose. They may be remarkably successful in traumatic cases for the replacement of medium-sized arteries. They must be reversed before insertion owing to their valves. Although vein grafts become slightly dilated, aneurysm formation does not occur.

In thrombophlebitis the clotting process is obscured by the inflammatory reaction. Usually the thrombus is firmly adherent, but in fulminating infections it may become softened or liquefied and give rise to septic embolism. Clotted blood is a good culture medium, and thrombosed vessels provide a bridge for the local spread of infection (see *Thrombosis of the Lateral Sinus*, Chap. 51). In phlebothrombosis the first microscopic evidence of intravascular clotting is the deposition of platelets on an irregular patch of tunica intima. As this progresses a "coralline" thrombus forms, made up of finger-like heaps of platelets with fibrin in between. To the naked eye this appears grey. As a result of the impeded blood flow, leucocytes become adherent to the vessel wall. Once the vessel has become completely occluded, the rapid formation of blood clot occurs, extending proximally as well as distally. This thrombus is termed the "propagated" clot, and as it is only loosely attached it may easily become dislodged and form an embolus. A venous thrombus may eventually become organised and the channel permanently blocked. A "phlebolith" is calcification in such a thrombus. When infection is present, an abscess sometimes forms. In most cases of venous thrombosis the clot is absorbed, although the vein rarely returns to normal. Often, destruction of valves leads to venous incompetence.

**Thrombophlebitis.** Veins are less resistant than arteries to inflammatory thrombosis from nearby infection, owing to their thinner walls and slower blood flow. Thrombophlebitis may occur after intravenous injections and continuous drip therapy. Sclerosing injections, used in the treatment of varicose veins, cause obliteration by sterile thrombophlebitis. Thrombophlebitis in Buerger's disease has already been discussed. *Thrombophlebitis migrans* is a rare condition of unknown cause in which episodes of thrombophlebitis occur successively in different parts of the body.

*Superficial thrombophlebitis* is the commonest type of venous thrombosis seen clinically, and is a frequent complication of varicose veins. It can readily be recognised by palpating a firm tender cord surrounded by œdema and redness. The regional lymph nodes may be enlarged, but pyrexia is rarely marked. Abscesses may later develop at the site of thrombosis. As the clot is tethered by the inflammatory process, pulmonary embolism practically never occurs. *Treatment.*—Response to local application of warmth and to systemic chemotherapy is usually rapid; ambulation should be continued (see also under *Thrombophlebitis in Varicose Veins*).

*Deep thrombophlebitis* may result from internal suppuration, e.g. thrombophlebitis of the lateral sinus in mastoiditis. Pylephlebitis of the liver due to abdominal sepsis is described in Chap. 5. Clotting of the iliac or femoral veins may complicate puerperal sepsis and occasionally follows operations on the lower abdomen or pelvis (*vide infra*). The clinical features of deep thrombophlebitis of the limbs include pain, congestion and œdema. The superficial veins may be engorged and there

## CHAPTER 75

### THE VEINS

#### INFLAMMATION AND THROMBOSIS

**Thrombophlebitis and Phlebothrombosis.** Inflammation of a vein, or *phlebitis*, is usually accompanied by intravenous clotting. *Phlebitis* without clotting is comparatively rare, but occasionally occurs near areas of inflammation; it may result from syphilis, and chronic *pernio*s (chilblains). Venous thrombosis is common and its pathology is not entirely understood, but one or more of the factors which predispose to intravascular clotting, i.e. damage to the tunica intima, venous stasis and increased clotting power of the blood, can be incriminated in most cases. Sometimes they seem to be absent. Some of the conditions associated with venous thrombosis are as follows: (1) *Causing Intimal Damage.*—Acute inflammation; non-suppurative inflammation, e.g. Buerger's disease; chemical irritation, i.e. from injections; trauma, including thermal injuries and those due to compression, e.g. by tumours or plaster bandages; invasion by malignant cells. (2) *Causing Venous Stasis.*\*—Cardiac failure; old age; dehydration; immobility and recumbancy; reduced respiratory excursion; arterial insufficiency; varicose veins; venous obstruction of any kind. (3) *Causing Increased Clotting.*—Generalised infections, e.g. typhoid fever; polycythæmia; leukaemia; tissue damage; operations.

Even in the absence of infection, a variable degree of inflammatory reaction is found in the wall of a thrombosed vein. When such inflammatory change is marked, the clinical signs are usually unmistakable and the diagnosis is easy. Pulmonary embolism is rare in such cases owing to anchorage of the clot by the inflammatory process. When the inflammatory signs are slight, the symptoms and signs of thrombosis may be minimal or absent; in these patients pulmonary embolism is a greater risk. It is customary to use the term *thrombophlebitis* when inflammatory changes are marked and *phlebothrombosis* when they are not. It must, however, be remembered that both these conditions are varieties of a single pathological process and the terms cannot be considered watertight. Clotting in the iliac and femoral veins is particularly difficult to classify in this way, for the extensive thrombosis leads to considerable local swelling and constitutional disturbance whether infection is present or not; the term *iliofemoral thrombosis* is therefore used to include all varieties of this condition.

\* Stasis by itself is insufficient, for a vein doubly ligated *in situ* does not clot unless traumatised or otherwise irritated.

To-day, antibiotic therapy renders ligation of the inferior vena cava (in cases of septic embolism) rarely necessary.

**Phlebothrombosis.** The clinical picture of phlebothrombosis is similar to that of thrombophlebitis, but the local signs are less marked. Although there may be a low-grade pyrexia, the constitutional effects are slight. Traumatic and post-operative phlebothrombosis only are considered here. *Trauma.*—Venous thrombosis may be caused by injury of a vein produced : (a) Directly ; (b) by stretching ; or (c) by repeated minor trauma. Air-raid shelterers who slept in deck-chairs sometimes developed swollen legs from a popliteal thrombosis resulting from a combination of pressure and stasis at the back of the knee. Airmen sitting for long periods in aircraft have suffered similarly. The so-called " spontaneous " thrombosis of the axillary vein is almost certainly traumatic in nature as it often occurs after repeated unaccustomed exertion. Spontaneous recovery follows in these cases after a few days or weeks.

**Post-operative Phlebothrombosis.** The incidence of phlebothrombosis amongst patients in both medical and surgical wards is approximately the same, yet the average age of surgical patients is lower and their general condition rather better than those with medical diseases. Of the factors predisposing to thrombosis, recumbency is common to both groups. It is also found in both groups that elderly patients, the obese and those suffering from malignant diseases are more susceptible. Although much remains to be learnt about venous clotting in surgical cases, certain aetiological factors are known. Tissue damage in operations is of importance, since phlebothrombosis is more common when the field of operation is extensive. Increased power of coagulation occurs in the blood after operations. This is particularly marked after splenectomy and there is a correspondingly high incidence of thrombosis after this procedure ; after thyroidectomy for thyrotoxicosis, on the other hand, increased clotting power is slight and post-operative thrombosis is infrequent. Thrombosis in damaged or infected veins is probably the starting point of post-operative thrombosis in some cases, especially after operations on the pelvis where the blood flow is sluggish. Both trauma to the calf veins and venous stasis may result from pressure of the legs on the operating table, or in bed, whilst the patient is unconscious. Immobility of the patient after operation, diminished respiratory excursion, the constricting effect of an abdominal binder, and periods of low blood pressure and dehydration may all be contributory factors.

Post-operative phlebothrombosis most commonly occurs in the veins of the calves, but the iliac or femoral veins may be affected (see under *Iliofemoral Thrombosis*). The thrombosis usually develops during the second week after operation, but may appear any time from the third day up to the third week. A clinical diagnosis of thrombosis can be made in nearly 3 per cent. of patients who have undergone surgery and approximately half of these patients suffer from pulmonary embolism, a small



may be tenderness along the course of the deep veins, which are sometimes palpably thickened. Pyrexia is often considerable and the patient feels ill and disinclined to activity. *Treatment* consists in combating the primary infection. The affected part should be rested and kept warm.

**Iliofemoral Thrombosis.** Iliofemoral thrombosis may be infective or non-infective, i.e. it may be thrombophlebitic or phlebothrombotic. In some cases no cause can be found ("silent" thrombosis). Iliofemoral thrombosis is sometimes a complication of operations on the abdomen, especially in the pelvis. Of considerable interest is its occasional occurrence in ambulant patients with visceral cancer—an unexplained complication first described by Trousseau who himself suffered from the condition.

The onset is usually fairly sudden; in surgical cases it occurs during the second week after operation. It may follow a more distal thrombosis or appear after a pulmonary embolus. Sometimes the disturbance to the limb is relatively mild, but if the clinical manifestations are severe the old term *phlegmasia alba dolens* is appropriate. Pain is often marked, but morphia is seldom necessary. The whole limb may become greatly enlarged, with the swelling tapering off in the thigh. At first brawny, the œdema later pits on pressure. The leg is usually pale from lymphatic stasis ("white" or "milk" leg), but there may be some cyanosis. It is usually warm, but if arteriolar spasm is superadded it is cool. The superficial veins are often distended and on palpation there is tenderness over the whole length of the femoral vein. A moderate degree of fever is usually present, up to 39° C. (102° F.). Shock is occasionally severe and renal failure has been described. Pulmonary embolism is rare, but should the clot become infected septic emboli may be liberated and septicæmia develop. Gangrene of the leg during the acute stage, though described, is most uncommon.

After a few weeks the swelling diminishes, but œdema of the leg and foot usually persists. Recanalisation of the veins is the rule, but further episodes of thrombosis sometimes occur. *Treatment.*—The leg should be rested on pillows and the foot of the bed placed on blocks. The warmth provided by cotton-wool is comforting; the local application of lead and opium lotion or ichthyol ointment may be tried. Anticoagulant therapy need only be prescribed when the signs of inflammation are slight. It must never be given if there is evidence of septicæmia. Antibiotic administration is advisable when symptoms are marked, and essential if there is evidence of bacterial invasion of the blood stream. Sympathetic block is reserved for early cases with cold legs, in which arteriolar spasm may be presumed: it should never be performed when anticoagulant drugs are being given as a hæmatoma may be caused by the injection. Ambulation should be begun as soon as the temperature has dropped and the physical signs have subsided. It is then important to maintain pressure on the leg to keep down œdema if the swelling is not to become permanent.

of chronic œdema and ulceration of the leg. Ligature may be justified : (1) When anticoagulants cannot be given ; (2) if embolism occurs during their administration ; or (3) if they have had to be discontinued owing to bleeding. At operation both femoral veins must be explored, since the condition is frequently bilateral ; the physical signs in one leg may be minimal or absent. Any clots found are removed before the veins are tied. As an alternative some surgeons prefer to ligate the inferior vena cava.

**Sequelæ of Deep Vein Thrombosis.** Superficial veins enlarge and act as collaterals during the acute stage and they often become permanently varicose. Usually the deep venous circulation is partly or completely restored by recanalisation and some patients recover completely. In others, however, destruction of valves and scarring of the vein leads to venous stagnation and incompetence. The communicating veins frequently become incompetent as well, and retrograde flow through these vessels adds to the congestion in the superficial veins which, if not already varicose, soon become so. Both obstruction and incompetence of the deep veins are followed by œdema and by a " tight " feeling in the muscles on exercise. If the deep veins are obstructed, this discomfort is made worse by applying a firm bandage to the limb whereas if they are incompetent a bandage may relieve the discomfort. For effective support, elastic bandages are better than elastic stockings which soon become stretched and cease to fit. The patient should sleep with the foot of the bed raised. At intervals, attacks of cellulitis and acute lymphangitis may occur. Pain is occasionally felt down the course of the veins (post-thrombotic neuralgia). The skin becomes pigmented, indurated and fixed to the deeper structures and chronic ulceration may develop after a trivial injury—one form of " venous " ulcer (*vide infra*).

### VARICOSE VEINS

Veins that have become lengthened, tortuous and dilated are called varicose. Varicose veins occur in the legs, the rectum (hæmorrhoids, Chap. 10), the scrotum (varicocele, Chap. 31), and around the œsophagus in portal hypertension (Chap. 5).

### VARICOSE VEINS OF THE LEGS

**Primary Varicose Veins.** In the legs the superficial veins consist of the long and short saphenous systems. The long saphenous vein extends from the dorsum of the foot and ends in the femoral vein at the saphenous opening approximately 3 cm. below and 3 cm. lateral to the pubic tubercle. The short saphenous runs up the back of the calf to penetrate the deep fascia in the popliteal fossa prior to its termination in the popliteal vein. A considerable proportion of blood normally passes from these superficial veins into the deep system (femoral, popliteal and tibial veins) through

number of which are fatal. The thrombosis is often symptomless and associated with few physical signs. Pulmonary embolism may, in fact, be the first evidence of the disease.

Early diagnosis can only be made if examination of the legs is made daily and if the nursing staff report all complaints of pain in the legs after operation. Pain and stiffness in the calves are usually the only symptoms. The most important physical signs are tenderness of the calf muscles and tenderness over the course of the deep veins in the leg or thigh. Pain on stretching the calf muscles by dorsiflexing the foot (Homan's sign) is a less reliable test. Mild œdema of the ankle is often noted and the leg may feel tense; there is a slight increase in the girth of the limb. There is a rise of temperature between 37° and 38° C. (99° and 100° F.) accompanied by a moderate leucocytosis and increased sedimentation rate. Phlebography is unreliable as an aid to diagnosis.

**Treatment of Post-operative Thrombosis.** This is directed to elimination of known predisposing factors as far as possible, an attempt to prevent pulmonary embolism and encouraging a rapid and complete return of the veins to normal.

*Reduction of Venous Trauma.* Veins should be handled with care during operation, and large vessels ligated flush with the main trunk, so that lengths of vein containing stagnant blood are not left in communication with the circulation. Pressure on the calves of the legs during anaesthesia should be prevented by resting the heels on a sorbo pillow.

*Maintenance of Active Blood Flow.* In shock and hæmorrhage a low circulatory volume should be made good by transfusion. Tight binders are to be avoided and early movements in bed must be encouraged. Early ambulation reduces the incidence of detectable thrombi and the severity of post-thrombotic sequelæ in the limb, but does not decrease the incidence of fatal embolism.

*Administration of Anticoagulants.* Anticoagulant therapy leads to the arrest of thrombosis and considerably reduces the likelihood of further episodes in patients with pulmonary embolism. Anticoagulant drugs also assist in restoring the affected limb to normal. Routine administration of these drugs after major operations carries certain risks and has not been found worthwhile. Anticoagulant therapy should, however, be begun immediately if phlebothrombosis is suspected or if pulmonary embolism has occurred. Details of dosage are discussed later in this chapter. The course of treatment should last at least three weeks, but the patient may get up after four days, for in this time the clot becomes sufficiently anchored to permit ambulation without risk of further embolism. If recumbancy is necessary throughout this period, but the patient is able to get up soon after the normal time for withdrawal of anticoagulants, they should be readministered for the first few days of ambulation.

*Vein Ligation.* Deep vein ligation is sometimes performed in an attempt to prevent pulmonary embolism, but there is a serious risk later

either in the superficial or in the perforating veins, leads to rapid extension of the disease.

**Secondary Varicose Veins.** When superficial varicose veins result from increased pressure in the deep veins, they are termed secondary (Fig. 520). The deep venous congestion is usually due to obstruction of the veins or incompetence of their valves. Other causes of secondary varicosity, such as arteriovenous fistulas, are rare. Deep venous obstruction may be thrombotic or result from pressure, such as that caused by a pelvic tumour. Deep venous incompetence is nearly always post-thrombotic in nature. In deep venous obstruction, the dilated superficial vessels act as a collateral circulation. Such varicosities are compensatory and therefore should not be eradicated. When, however, superficial venous dilation results from deep venous incompetence the varicosities serve no useful function and may therefore be ablated, although this may not relieve existing complications since their primary cause is disease in the deep veins.

**Clinical Features.** Rapidly enlarging veins are often painful and a mild degree of aching and tiredness is a common complaint by patients with long-established disease. The character of the patient appears to be largely responsible for the severity of the symptoms; they are, however, usually slight unless complications ensue. The latter include thrombophlebitis, dermatitis and ulceration; they are discussed, with their treatment, below. Patients easily attribute pain in the leg to any varicosities present and thus the true cause of pain may be obscured. A history of confinement to bed with a white swollen leg, usually after an operation or childbirth, suggests that the varicosities may be secondary to a past deep thrombosis.

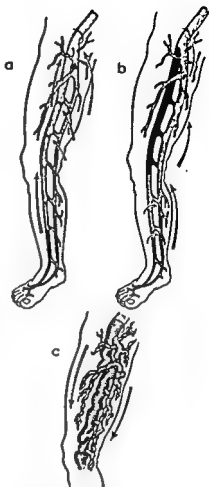


FIG. 520. The effects of deep venous obstruction in the leg. In (a) the venous flow in the leg is normal, a proportion of blood passing from the saphenous system into the deep veins, with valves preventing back-flow. In (b) there is thrombosis of the femoral and popliteal veins. The saphenous system has become dilated and compensates for the deep venous obstruction; the valves in the long saphenous vein have become functionless. (c) Recanalisation has resulted in incompetence of the deep veins. In the erect position the deep venous flow is retrograde and the veins become increasingly varicose.

the many vessels which perforate the deep fascia and muscles, and communicate between the two systems. Although these perforating (or communicating) vessels may occur at any level, one constant communication lies just above the medial malleolus and another at the level of the adductor tubercle. The superficial veins and the extramuscular portions of the perforating vessels, being relatively unsupported by the surrounding tissues, easily become stretched by an increase in venous pressure.



FIG. 519. Incompetence of both long saphenous veins leading to enormous dilatation. This patient had no symptoms.

Varicose veins occur in the legs in approximately 15 per cent. of the population, and advice concerning them is frequently requested. They usually appear between the ages of twenty and forty, and are frequently bilateral (Fig. 519). The vein walls may be thickened, and on division the lumen remains open; later, the walls are thinned and stretched, the valves become inefficient and cyst-like dilatations (varices) appear in the course of the vessels. Periphlebitis leads to adherence of loops of a vein to one another and to the skin. On microscopy the tunica media is most affected—the muscle cells being replaced by fibrous tissue.

In most cases only the superficial veins and their tributaries are varicose. Often there is an hereditary factor. Other predisposing causes are less certain, but work involving standing for long periods,

thus denying the veins the assistance of the muscular “pump,” encourages their development. Tight clothing round the thighs or lower part of the legs is also perhaps an aggravating factor. Varicose veins are more common in women than men. They often become prominent during menstruation and they may appear for the first time in early pregnancy, even before the enlarging uterus is causing mechanical interference with venous return. A certain degree of hormonal influence may therefore be supposed. On the other hand, violent exertion may lead to varicosities, since athletes are often affected. This is presumably either the result of increased blood flow in the veins or their repeated obstruction during muscular contraction. The congenital absence of valves may be a predisposing cause, but dilatation does not always follow this defect. In patients who already have varicose veins, however, loss of valve function,

traverse the groin and extend across the midline over the lower abdomen.

**Tests for Saphenous Incompetence.**—Reflux down the long saphenous vein is normally prevented by competent valves both in the saphenous systems and in the perforating vessels. Incompetence of the valve immediately below the termination of the saphenous vein is demonstrated by the following tests: (1) *The Trendelenberg Test.*—The patient lies supine and the hip is flexed with the knee straight (Fig. 521). The examiner occludes the saphenous opening (a) and asks the patient to stand (b). On releasing the pressure (c) the long saphenous fills from above (Trendelenberg positive). (2) *The Impulse on Coughing.*—Incompetence of the long saphenous vein is accompanied by a palpable impulse on coughing. This can often be felt not only in the groin, but for a variable distance down the leg. This test, though simple, is most useful. In secondary varicose veins, examination may reveal the cause of the obstruction of the deep veins, e.g. an abdominal or pelvic tumour. The degree of ankle oedema is significant; when it is marked, deep venous disease is likely. *Test for Compensatory Veins.*—The patient is made to walk with an elastic bandage applied from the ankle to groin sufficiently tightly to flatten the superficial veins (Fig. 522). When the deep veins are unobstructed the patient can walk without discomfort, but when they are obstructed compression interferes with venous return and a cramping or "bursting" pain results.

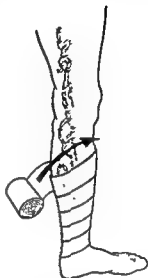


FIG. 522. Test for compensatory varicose veins (see text).

**Differential Diagnosis.** Occasionally a saphenous varix in the groin may be so localised as to be mistaken for a femoral hernia. In the calf a small muscle hernia is sometimes confused with a varicose vein. A muscle hernia, however, changes in size with muscle contraction and not, like a varix, with the position of the limb.

### TREATMENT

**Primary Varicose Veins.** Symptomless veins should be left alone, although in the case of a young person with a positive Trendelenberg test operation may be performed if the disease is progressive. During pregnancy treatment should be conservative as partial regression occurs after childbirth. Varicose veins causing severe symptoms or leading to complications may be treated by injection, operation or elastic stockings. Injection treatment is indicated in cases with competent veins and in order to obliterate any veins remaining after surgery. Operative treatment is required for incompetent veins. Elastic stockings are reserved for

*Physical Signs.* The patient is examined standing with the whole of each leg exposed up to the groin. Veins are usually more easily felt than seen. In obesity, and when the tissues are oedematous, the veins may be

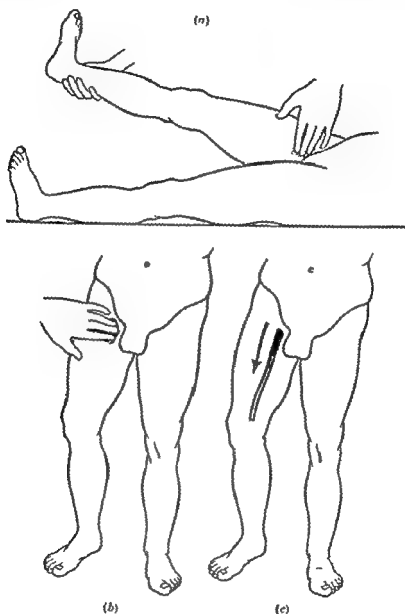


FIG. 521. The Trendelenburg test for incompetence of the saphenous vein in the thigh (see text).

felt as gutters running beneath the skin. Swollen, tortuous veins may be localised to a small area or extend over the whole length of the leg. Varicose veins feel tense, but when there is no proximal obstruction, they collapse when the patient lies down. An unusually large dilatation at one point often (but not necessarily) marks the site of an incompetent perforating vein, or "blow-out." "Compensatory" varicose veins often

**Surgical Treatment.** The essentials are : (1) In the case of varicosity of the long saphenous vein, to divide and tie it in the groin and to divide and tie all its branches at this site. (2) In the case of varicosity of the short saphenous vein, to divide and tie it in the popliteal fossa. (3) In either case to break up the main channel, either by multiple ligation or stripping. In addition to these procedures, local excisions of large groups of baggy veins may be undertaken.

After operation, the patient should be got out of bed at the earliest opportunity, and physical activity must be encouraged throughout convalescence. If this practice is observed, the risk of pulmonary embolism from the detachment of venous clot is almost negligible.

*Division and Ligation of the Long Saphenous Vein.* The surface marking for the termination of the vein is a point where a perpendicular dropped from the pubic tubercle crosses the groin crease. An incision is made in the latter and the long saphenous vein dissected to its junction with the femoral vein. The saphenous is then clamped and divided and all its branches, which may be numerous or restricted to the three named vessels, clamped, divided and tied. Both ends of the saphenous vein are then ligated—the upper end flush with the femoral vein.

*Division and Ligation of the Short Saphenous Vein.* This operation is performed through an incision in the popliteal fossa. The vein passes at a variable level through the fascia which must often be incised before the vein is seen.

*Stripping and Multiple Ligation.* Opinion remains divided concerning the relative merits of these two procedures. When stripping is possible all perforating vessels connecting with the saphenous veins are disrupted ; some perforating vessels, however, connect only with the saphenous tributaries and these are not detached by this method. Bleeding is comparatively slight and complications are no more frequent than with multiple ligation. Previous ligations make stripping impossible ; it is therefore recommended that stripping be attempted in the first instance, combined with ligation of remaining branches. Stripping should not be performed on patients with a past history of deep thrombosis, in case the thrombosis recurs. In these cases ligation is safer, for a collateral circulation in the superficial veins is able to develop after saphenous ligation only.

*Stripping.* General anaesthesia is required. The legs should be slightly raised to reduce bleeding during operation. Groin dissection and ligation is performed as already described. The rounded end of a Myer's type stripper (Fig. 523) is then inserted at the ankle and manipulated until it comes out in the groin. If this manoeuvre fails, an attempt is made to pass the stripper in retrograde fashion down the saphenous vein. The vein is tied to the stripper and pulled out in its whole length. If the stripper cannot be made to traverse the whole length of the vein, a small incision is made over the stripper at the point of arrest and the vein divided at this site ; the stripper is then reinserted. Multiple stripping



elderly patients, pregnant women and those patients in whom other methods have failed or cannot be applied.

**Secondary Varicose Veins.** In the early stages, a light bandage may give symptomatic relief. After recanalisation of the deep veins, superficial varicosities may be treated by surgery or injections. The bandage test described for "compensatory" veins is of value in the selection of such cases, for veins which can be obliterated by pressure without symptoms on exertion may safely be eradicated.

**Injection Treatment.** Sclerosing substances are injected in order deliberately to damage the tunica intima, and produce a chemical thrombophlebitis. The inflammatory reaction must be sufficient to produce a firm clot that will not detach and has little tendency to recanalise. When, however, the saphenous vein is incompetent, the rapid blood flow and high venous pressure make such a result impossible to achieve. Major complications of injections include: (1) Anaphylaxis from sensitivity to the substance used; and (2) sloughing of the skin ("injection ulcer") as a result of spill of the sclerosing substance outside the vein. The first is avoided by giving a test dose (0.5 ml.) on the first occasion, but an injection of adrenaline should always be at hand in a vein clinic. Injection ulcers are avoided if a careful technique is used. Pulmonary embolism is exceedingly rare; one estimate is 1 in 15,000 cases. The incidence is, however, raised if patients do not remain ambulant after injection. Substances in use include: Ethamolin (monoethanolamine oleate 5 per cent. with 2 per cent. benzyl alcohol),  $\frac{1}{2}$  to 2 ml.; sodium morrhuate, 5 per cent., 5 ml.; and quinine, 5 per cent., with urethane, 2.5 per cent., 5 to 15 ml. Ethamolin, although not the strongest sclerosing agent, is probably the safest. Injections (not more than two at a time) should be given at intervals of one to two weeks; longer intervals encourage the development of anaphylaxis. The most satisfactory results are obtained using the empty vein technique. The needle is inserted with the patient erect, or sitting and the leg dependent; the patient is then asked to lie down, or if previously sitting, to raise the leg, before the injection is given. Another method is to use one light tourniquet above and another below the site of injection. The needle is inserted with the patient recumbant, the tourniquets being removed prior to injection. This method (which is not always possible) both prevents shift of the needle during change of the patient's position and discourages fainting attacks. The leg is kept still for five minutes after injection as muscular movement sucks blood from the superficial into the deep veins and allows less time for the sclerosant to produce local irritation of the vein. After this, a temporary dressing is firmly applied. After successful injection the vein becomes red and tender; the patient should be warned of this. In the presence of active thrombophlebitis, injections are followed by an exaggerated reaction, they should, therefore, be withheld until any inflammation has subsided.

by local pressure. *Pigmentation* of the skin is common, especially in areas of old thrombophlebitis and ulceration. *Dermatitis*.—Congestion of the skin as a result of varicose veins appears to increase its sensitivity. Dermatitis may follow scratching or arise in the skin near an ulcer. It may, however, be a form of "contact" dermatitis and the type of stocking or bandage worn may be responsible. Occasionally a localised rash becomes generalised. Treatment includes a search for sensitising agents used either during employment or on the leg in the form of dressings; any found must be eliminated. The skin around ulcers should be protected by an ichthyol varnish if the discharge appears to be irritating. Greasy substances are badly tolerated, but wet applications, e.g. silver nitrate solution or lead lotion, are of benefit. For chronic eczema Lassar's paste with crude coal tar, or Viscopaste and ichthyol, are satisfactory. When itching is troublesome hydrocortisone cream (1 per cent.) may prove effective. *Thrombophlebitis*.—Acute thrombophlebitis is common. It has been described previously. Pulmonary embolism is extremely rare; in such cases the embolus arises from propagated clot lying higher up the vein than the upper limit of swelling and tenderness. Thrombophlebitis responds well to supporting bandages and ambulation should be continued. If, however, the main trunk of the long saphenous vein is involved, immediate saphenous ligation is advocated, not only to forestall embolism, but also to hasten recovery. The application of a firm pad above the upper limit of inflammation, followed by bandaging of the leg, is a less satisfactory alternative. *Oedema* of the ankle is not a very common complication of superficial varicosities, but in the case of secondary varices it is often marked. *Varicose ulceration* is discussed below.

### VENOUS ULCERS

Venous ulcers are common in the lower limb. They occur almost exclusively in the lower third of the leg (the "gaiter" area) and may be single or multiple. Both limbs are frequently affected. The ulcers are painful and tender, and have irregular (sometimes serpiginous) shelving edges, usually with evidence of epithelial proliferation at the margin. A certain amount of pigmentation and induration is found in their vicinity. The cause is increase in the venous pressure either from incompetence or obstruction of the veins.

*Varicose Ulcers*. Primary varicose veins account for rather more than half the cases of venous ulceration in the leg (Figs. 525 and 526). Skin necrosis often follows a mild injury, an attack of thrombophlebitis or the misplaced injection of sclerosing agents. On other occasions these factors are absent. Stasis in the saphenous systems, malnutrition of the skin due to a mass of distended veins beneath it, and induration of the tissues, all contribute to the chronicity. The majority of varicose ulcers remain unhealed for months or years, if untreated, although a few close up and



FIG. 523 A Myer's type stripper, showing the long saphenous vein after extraction in "concertina" fashion.

*Hæmorrhage* is usually due to a technical fault and is avoided if a careful exposure is made, anomalous veins are recognised and care is taken with thin-walled varices, especially in the groin. If bleeding is violent the femoral vein should be compressed above and below, and the bleeding vein sought, for if bleeding is not controlled in this way the femoral vein itself may be torn. If so, it can usually be repaired. Otherwise the use of a temporary pack is permissible; on no account should the femoral vein be tied. *Wound sepsis* is not infrequent, especially after incisions made in areas where the skin is in bad condition.

*Pulmonary embolism* is extremely rare if ambulation is begun after operation, and if the saphenous vein is tied flush with the femoral vein to avoid a pocket in which a thrombus could form. A temporary, mild degree of *œdema* occurs in a few cases. *Recurrence* may take place despite conscientious surgery. Some of the causes of failure are as follows: (a) Insufficiently high ligation of the long saphenous vein; (b) failure to tie the main trunk; (c) failure to tie all the branches in the groin (Fig. 524); (d) failure to divide all incompetent perforating veins; and (e) operating on secondary veins. Provided secondary veins are ruled out, further ligature operations may prove successful.

#### COMPLICATIONS OF VARICOSE VEINS

*Hæmorrhage* may follow trauma. It ceases on elevation of the limb and can be controlled

may in this way be performed. After operation the legs are supported with crêpe bandages.

*Multiple Ligation.* The veins must be tied at many points. As there is a constant communication at the level of the adductor tubercle it is important to tie the vein just below this point. Other sites are chosen over "blow-outs" and over large veins. Five or six ties may be necessary below the knee. The selected sites are marked with indelible ink before operation with the patient standing.

#### Complications of Operation.



FIG. 524. A cause of post-operative recurrence of varicose veins. All the branches of the long saphenous vein were not divided in the groin at the previous operation. Their dilatation has led to further incompetence in the superficial venous system.

by local pressure. *Pigmentation* of the skin is common, especially in areas of old thrombophlebitis and ulceration. *Dermatitis*.—Congestion of the skin as a result of varicose veins appears to increase its sensitivity. Dermatitis may follow scratching or arise in the skin near an ulcer. It may, however, be a form of "contact" dermatitis and the type of stocking or bandage worn may be responsible. Occasionally a localised rash becomes generalised. Treatment includes a search for sensitising agents used either during employment or on the leg in the form of dressings; any found must be eliminated. The skin around ulcers should be protected by an ichthyol varnish if the discharge appears to be irritating. Greasy substances are badly tolerated, but wet applications, e.g. silver nitrate solution or lead lotion, are of benefit. For chronic eczema Lassar's paste with crude coal tar, or Viscopaste and ichthyol, are satisfactory. When itching is troublesome hydrocortisone cream (1 per cent.) may prove effective. *Thrombophlebitis*.—Acute thrombophlebitis is common. It has been described previously. Pulmonary embolism is extremely rare; in such cases the embolus arises from propagated clot lying higher up the vein than the upper limit of swelling and tenderness. Thrombophlebitis responds well to supporting bandages and ambulation should be continued. If, however, the main trunk of the long saphenous vein is involved, immediate saphenous ligation is advocated, not only to forestall embolism, but also to hasten recovery. The application of a firm pad above the upper limit of inflammation, followed by bandaging of the leg, is a less satisfactory alternative. *Oedema* of the ankle is not a very common complication of superficial varicosities, but in the case of secondary varices it is often marked. *Varicose ulceration* is discussed below.

### VENOUS ULCERS

Venous ulcers are common in the lower limb. They occur almost exclusively in the lower third of the leg (the "gaiter" area) and may be single or multiple. Both limbs are frequently affected. The ulcers are painful and tender, and have irregular (sometimes serpiginous) shelving edges, usually with evidence of epithelial proliferation at the margin. A certain amount of pigmentation and induration is found in their vicinity. The cause is increase in the venous pressure either from incompetence or obstruction of the veins.

**Varicose Ulcers.** Primary varicose veins account for rather more than half the cases of venous ulceration in the leg (Figs. 525 and 526). Skin necrosis often follows a mild injury, an attack of thrombophlebitis or the misplaced injection of sclerosing agents. On other occasions these factors are absent. Stasis in the saphenous systems, malnutrition of the skin due to a mass of distended veins beneath it, and induration of the tissues, all contribute to the chronicity. The majority of varicose ulcers remain unhealed for months or years, if untreated, although a few close up and

others heal for a while but break down again. Treatment by compression usually produces healing within three months even though ambulation is continued, and if the varicose veins are operated upon recurrence is unlikely.

**Venous Ulcers due to Disease of the Deep Veins (Syn. "Post-thrombotic" or "Gravitational" Ulcers).** When the deep veins of the leg are incompetent, the weight of the column of blood that the limb has to support is considerable, and the perforating veins become incompetent. Unlike saphenous vein incompetence when the raised pressure can be evenly

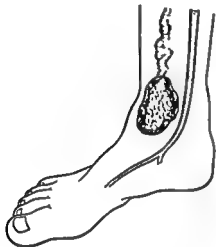


FIG. 525. Varicose ulcer. The deep veins are normal, but the superficial veins are varicose. Oedema of the ankle is only moderate.

FIG. 526. A healing varicose ulcer. In this case varicose veins were only visible below the knee, but extensive varicosities could be felt as gutters running through the indurated subcutaneous tissues of the leg.



FIG. 526.

distributed throughout the limb, incompetence of the perforating veins allows the deep venous pressure to act on a limited area of subcutaneous tissue. Furthermore, such incompetence permits the active ejection of blood into the superficial tissues of the lower part of the leg on muscular contraction of the calf. For these reasons, ulceration is more severe in such patients than in those with varicosity of the saphenous systems alone (Figs. 527 and 528). Varicosity of the superficial veins may be absent or slight. If present, it may result from the same deep venous disorder, but although it is not the primary cause of ulceration it may assist in its development. Sometimes a history of deep thrombosis is not obtained. It is believed that a relatively symptomless thrombosis of the calf veins may account for some of these cases, the popliteal and femoral veins

remaining unaffected. "Post-thrombotic" ulcers are usually situated over the inner side of the lower third of the leg—an incompetent perforating vein being present beneath them. At first similar to a varicose ulcer, a post-thrombotic ulcer becomes larger and deeper, often encircling the limb. There is considerably more ankle œdema, and the ankle joint often becomes stiffened; in time an equinus deformity of the foot develops.



FIG. 527. Venous ulcer due to deep venous disease ("post-thrombotic" or "gravitational" ulcer). The deep veins are dilated and incompetent, and an incompetent perforating vein lies in the floor of the ulcer. The superficial veins are moderately varicose, perhaps also the result of the deep venous disease.



FIG. 528. "Post-thrombotic" or "gravitational" ulcer of fifteen years' duration. Note the encircling nature of the ulcer, and the thickening of the skin of the foot from chronic œdema. None of the superficial veins were varicose in this patient.

Sometimes there is elephantiasis of the lower part of the leg and foot. On X-ray examination, a considerable degree of periostitis may be visible beneath the ulcer. Malignant (epitheliomatous) change may take place after a number of years in neglected cases (*Marjolin's ulcer*, Fig. 529).

**Differential Diagnosis of Chronic Ulcers of the Leg.** Venous ulcers are painful and usually affect the lower part of the leg; any ulcer outside the "gaiter" area is almost certainly not venous. Varicose veins may be present. Ulcers due to *arterial insufficiency* affect especially the heel and

foot. They are accompanied by the signs of arterial ischaemia previously described. *Hypertensive ulcers* may occur in hypertension on the outer side of the calf. They begin as a painful red patch and are due to arterial thrombosis. *Chronic anaemia*, especially of the haemolytic variety, may lead to superficial ulceration. The shin is the usual site. This cause may be excluded by blood examination. *Syphilitic ulcers* are painless; they are commonly multiple and situated in the *upper* third of the leg. They



FIG. 529. Marjolin's ulcer. (Case of Professor J. C. Goligher.)

have serpiginous edges and a "wash-leather" base. The Wassermann reaction is not always positive. *Chronic perniosis* affects the back of the calf and the skin is red or blue, and cold. *Trophic ulceration*, due to nervous disorder, may have to be excluded. Ulcers of this nature affect the pressure areas and are accompanied by abnormal neurological signs. "Tropical" ulcers, common in hot climates, are sometimes seen in travellers. On microscopic examination of scrapings from tropical ulcers Leishmann-Donovan bodies may be seen. Ulceration of the leg may be *self-induced* (ulcer "artefacta"). In cases of difficulty in diagnosis, biopsy of the ulcer margin is often helpful.

**Treatment of Venous Ulcers.** A full investigation is necessary to determine the vascular condition of the leg and to eliminate other possible

causes of ulceration. *Varicose ulcers* respond well to compression. Although infection is relatively unimportant, a few days' cleansing with Eusol before applying occluding bandages may be helpful. Local application of 1 per cent. chloromycetin ointment may be valuable in grossly infected ulcers. The superficial veins must be treated appropriately, but it is advisable to achieve healing of the ulcer first so that accurate surgery on the lower leg is possible; however, in refractory ulcers, operation may be needed earlier. Particular care should be taken to tie any vein immediately above the ulcer. "*Post-thrombotic*" ulcers are usually more refractory to treatment, but often heal provided efficient support is maintained. Failing this, healing can usually be achieved by elevation of the leg in bed. Afterwards, an elastic support must be worn permanently or recurrence is very likely. Carcinomatous ulcers, if small, may be treated by radiotherapy, but if the limb is grossly disorganised, amputation may have to be performed.

*Supporting Bandages.* Bandages extend from the base of the toes to the tibial tubercle. Irritating applications to the ulcer are avoided and bland substances such as zinc cream or arachis oil are used. Unna's bandage consists of zinc oxide, gelatin, glycerin and water; Viscopaste and Dalzoband are proprietary forms of this application. A comfortable bandage is made by placing a few strips of Viscopaste over the ulcer and applying several thicknesses of Tubegauz over the leg, with a final layer of Elastoplast put on with the adhesive side outwards. Viscopaste or Elastoplast alone (provided that the patient is not sensitive to the adhesive) are also satisfactory. Bandages may be left on from two to four weeks before being reapplied. Discharge through the bandage is an inconvenience, but does not delay healing. In order to prevent dermatitis from the discharge, a varnish of 50 per cent. ichthyol in water may be painted around the ulcer and over the whole leg. When the bandage is applied great care is taken to see that it is firm yet not too tight, and without rucks. When healing is beginning, a rubber pad is placed over the ulcer to reduce surrounding oedema. At this stage, co-operative patients may be supplied with a strong elastic (webbed) bandage which they apply themselves before rising; a simple dressing is used at night. Epithelialisation of the ulcer does not signify completion of the healing process. Bandages should be retained for a further month to ensure complete subcutaneous consolidation before gravity is allowed to act on the unsupported leg. Provided the limb is firmly supported, exercise is of value as it encourages venous return through the mechanism of the muscle "pump." Standing for long periods, or sitting with the legs dependent, or crossed, must be avoided. Ankle movements should be practised in order to avoid stiffness and to discourage stasis. Care must be taken to avoid injury to the leg. Weight reduction is often advisable.

*Surgical Treatment of Venous Ulcers.* Apart from the surgical treatment of any associated varicose veins, operation is only indicated if a venous



ulcer remains unhealed for twelve months in spite of carefully applied supporting bandages. *The ulcer should then be excised and a search made for deep perforating veins. The latter are tied and a skin graft is applied to the wound. Deep Vein Ligation.*—In patients with long-standing ulcers and severe "bursting" pain, due to associated deep venous incompetence, ligation of the popliteal (or femoral) vein has been tried with disappointing results. *Lumbar sympathectomy*, which used to be recommended, is now thought only to benefit those cases with an associated degree of ischaemia from arterial degeneration.

### VENOUS TRAUMA

Even if the venous pressure is low, bleeding may be prolonged, for the walls of veins only contract weakly and their fascial attachments may hold them open. At operation, venous bleeding is found to be particularly troublesome from raw surfaces, e.g. the tonsillar fossa. In the limbs, first-aid measures consist of elevation of the part to reduce congestion, and the application of local pressure. *A tourniquet should not be used.* Large veins that have been partially or completely divided should be repaired by suture. Ligation of a single internal jugular vein is permissible, and the brachial and superficial femoral veins may be sacrificed. Subcutaneous haematomas may result from injury to veins. As they may take a long time to absorb and may become secondarily infected, evacuation is usually advisable.

**Air Embolism.** Air embolism is an occasional complication of accidental wounds, e.g. "cut-throat." For embolism to occur venous pressure must be negative and it is therefore unlikely when a cut vein bleeds profusely. Although rare, air embolism may also result in sudden death at operation. Procedures which may be so complicated include: the induction of a pneumothorax or pneumoperitoneum, insufflation of the Fallopian tubes, instrumental abortion, neurosurgical procedures (especially on sitting patients), and radical mastectomy. Air embolism from the last-mentioned operation usually results from attempting to clamp the anterior perforating vessels too deeply, with the result that both a pneumothorax and intrathoracic damage to an intercostal vein occur. Air embolism may also complicate intravenous therapy, especially if fluids are given under pressure. If a clot has formed around the filter, air embolism may occur during blood transfusion even though the bottle is not empty.

**Clinical Features.** Air that has been aspirated becomes churned up with the blood in the heart. This may lead to: (1) Interference with the cardiac output; or (2) cerebral anoxia due to displacement of blood by air in the cerebral vessels. Sometimes a sucking noise may be heard as the air enters the blood stream. Collapse may be immediate. The heart beats rapidly, but the radial pulse is absent. Bubbling noises may be heard on auscultation over the precordium. Death may be almost

instantaneous, but recovery occurs if the amount of air aspirated is small, i.e., less than 50 ml.

**Treatment.** Pressure must immediately be applied to the vein; at operation, a finger should be placed on the vessel, the wound flooded with saline and a ligature applied. The patient's feet should be raised and oxygen given. In desperate cases, aspiration of the heart chambers and of both jugular veins should be attempted.

**Fat Embolism.** This type of embolism occasionally follows severe injuries, especially of the limbs. The fat particles are liberated from adipose tissue or bone marrow and they enter the circulation through a damaged vein. Symptoms appear a few days after the accident, and depend on whether the lungs or the brain are affected. In either case pyrexia occurs, and fat particles may be found in the urine. *Pulmonary* fat embolism leads to patchy consolidation of the lungs, and there is anoxia with marked cyanosis. Fat globules may be detected in the sputum. Death may result from right-sided heart failure, or from bronchopneumonia. In the case of *cerebral* embolism the patient is at first restless, then drowsy. Death may occur in coma, but recovery sometimes takes place.

**Treatment** is almost entirely confined to attempts at prevention, e.g. in fracture cases especial care should be taken to avoid unnecessary movement and manipulation. Any complicating pneumonia is treated by antibiotics. In comatose patients, burr-hole exploration of the skull may be undertaken since other causes of coma, such as intracranial hæmatomas, must be excluded.

## ANTICOAGULANT THERAPY

by

A. A. G. LEWIS

**Normal Coagulation.** When blood comes into contact with a foreign surface or with tissue extracts, the platelets and four other agents (Factors V and VII, Christmas Factor and antihæmophilic globulin) react in the presence of calcium to produce thromboplastin within a few minutes. This rapidly converts prothrombin to thrombin, which in turn forms fibrin from fibrinogen.

The drugs used to prevent intravascular coagulation are : (1) Heparin ; (2) those of the coumarin group; and (3) phenindione derivatives. *Heparin*, though normally present throughout the body, is found mainly in mast cells in the lungs and liver. It inactivates thrombin, preventing the formation of fibrin while inhibiting that of both thrombin and thromboplastin. These effects are immediately annulled by the intravenous injection of 10 ml. of

1 per cent. protamine sulphate. Heparin is available in solutions of 1,000 units/ml., 5,000/ml. and 25,000/ml. (100 units = 1 mg.). The anticoagulant action of 10,000 units, given intravenously, is maintained for a few hours only, so that a prolonged effect has to be achieved by one of three methods: (1) Intravenous injections of 10,000 units every four hours. (2) Intramuscular injections of 12,500 units eight-hourly. These may be painful, and sometimes cause local hæmatoma formation. (3) Intravenous infusion of a solution of 20,000 units in 1 litre of saline at a rate of 20 drops a minute. There may be considerable variations in individual responses to the drug, and large doses may be necessary to control acute thrombophlebitis.

Other drugs are active when given by mouth. Several are available; two of the most useful are *Dindevan* (which depresses the formation of prothrombin by the liver) and *Marcoumar* (which depresses Factor VII production as well). The actions of all these drugs, however, can be checked by the same laboratory test—the estimation of the “prothrombin” time. 0.1 ml. of a suspension of thromboplastin (preferably prepared from fresh human brain) is heated to 37° C. with 0.1 ml. of plasma, 0.1 ml. of M/40 CaCl<sub>2</sub> added and the clotting time noted. With the same thromboplastin and calcium, the test is repeated with normal plasma. In most laboratories, the time for coagulation is twelve to eighteen minutes, though it may be longer. The time for the test plasma should be two and half times as long as the normal. This may be expressed as the “prothrombin index” which is  $\frac{\text{Time for normal}}{\text{Time for test}} \times 100$ . This

should not be more than 40 per cent. for clinical control. If much less than 40 per cent., there is a risk of spontaneous hæmorrhage.

**Technique of Anticoagulant Therapy.** When the need to prevent intravascular clotting is urgent, 10,000 units of heparin should be given intravenously at once and one of the above methods of administration continued for forty-eight hours (though the dosage should be reduced on the second day). At the same time the first dose of *Marcoumar* (21 mg.) or of *Dindevan* (200 mg.) is given. Very often, control can be established more slowly, when heparin can be omitted altogether. On the next day, 9 mg. of *Marcoumar* may be given in the morning, or 50 mg. *Dindevan* morning and evening. On the third day the prothrombin concentration should be within the therapeutic range; heparin is discontinued and *Marcoumar* then given (0.75 to 1 mg. daily) or *Dindevan* (25 to 50 mg. b.d.) to maintain this effect (a few patients are comparatively resistant to *Dindevan* therapy, and young adults and obese patients need a higher dosage). After the first few days, the prothrombin concentration may be estimated twice a week, and later, only once a week. The first indication of overdosage is usually hæmaturia (*Dindevan* may impart an orange-pink colour to normal urine). Bleeding from mucous membranes is less common. The drug should be temporarily discontinued if this occurs.

The prothrombin concentration rises to normal within forty-eight hours of stopping Dindevan therapy, but not for seven to fourteen days after Marcoumar. If more immediate control is necessary, 25 mg. of vitamin K orally or 10 mg. intravenously is effective in a patient taking Dindevan; a blood transfusion is necessary to reverse the effect of Marcoumar immediately.

**Contraindications to Anticoagulant Therapy.** (1) Recent Operations.—At least twenty-four hours should elapse before prophylactic therapy is begun (then the preliminary heparinisation may be omitted). An exception to this rule is made in vascular surgery. (2) Any suspected bleeding tendency—e.g. from a peptic ulcer. (3) Liver and kidney damage.

Marcoumar should not be given in pregnancy. Menstruation is not affected by these drugs. Minor medical procedures, such as paracentesis, are not contraindicated, but intramuscular injections are better avoided during anticoagulant therapy.

Anticoagulant therapy tends to prevent the formation of new thrombus and limit the extension of any already present, so that this may become firmly adherent to the vessel wall and organised, with no risk of embolism. Moreover, it has been suggested that recanalisation may actually be accelerated. With regular laboratory control, therapy can be continued for weeks or months, but it must be terminated gradually.

## CHAPTER 76

### HÆMANGIOMAS AND ALLIED CONDITIONS

by

VALENTINE A. J. SWAIN

SKIN blemishes are frequently found in the new-born. These birth marks, or *nævi*, vary in nature and size, and their subsequent development differs with the particular type of lesion. They may be only skin deep, or may be the outward and visible sign of a deeper angiomatous condition.

The nomenclature of this subject is confusing; the term "*nævus*" literally means a birth mark, mole or wart, but this name is often applied to a hæmangioma of the skin. To avoid confusion, it is advisable to qualify the word "*nævus*" by the descriptive adjective "*vascular*," "*pigmented*" or "*hairy*." It is generally agreed that a hæmangioma is not a true neoplasm, but rather a disturbance of development arising from a mass of vasoformative tissue; it is thus one of the varieties of hamartoma.\* It has no capsule. These lesions consist of vascular tissue composed of endothelial lined spaces filled with blood, capillaries, larger vessels, veins or arteries. There is great variation in the macroscopic and microscopic appearances; the tissue may vary from one part of the lesion to another.

Angiomas may increase or decrease in size, or remain unchanged. Their growth is probably caused by further development of areas of vascular embryonic tissue, or by canalisation of the adjacent structures. Sometimes the vascular channels within these lesions become thrombosed and subsequent fibrosis results in diminution in size. Occasionally phleboliths† develop. Ulceration and infection may occur, especially when the hæmangiomas lie on exposed surfaces and are therefore subject to injury. The severity of bleeding from an ulcerated surface depends on the size of the underlying vessels. Subsequent healing following ulceration leaves some scarring and thickening of the skin. Sometimes areas of cutaneous thickening (hyperkeratosis) arise giving the surface of the angioma a nodular appearance. Malignant change in hæmangiomas is almost unknown, but a few examples of metastasis have been recorded. It is generally agreed that the condition known as angiosarcoma is in fact a very vascular sarcoma rather than an angioma which has become malignant. Hæmangiomas are here classified on clinical rather than pathological appearances after Matthews.

\* Hamartoma = a tumour-like malformation.

† Phlebolith = a venous calculus or concretion.

**Spider Nævus.** This type of nævus may not be present at birth, but may appear in early childhood. It is a superficial lesion with a pink staining centre and lashes of capillary lines radiating outwards. They are flat and fade on compression. This type of capillary nævus tends to disappear spontaneously and therefore often needs no treatment. Otherwise, the centre of the lesion is coagulated with the diathermy needle or carbon dioxide snow. These methods give excellent results.

**Neonatal Staining.** This is evident after delivery as a superficial area of redness, usually over the face and neck. It is probably produced by pressure changes in the skin capillaries during labour. The condition is rather unsightly, but the mother can be reassured that the skin will become normal in a few weeks without treatment.

**Superficial capillary hæmangioma** presents in many forms; Fig. 530 is an example. Other varieties are sufficiently characteristic to warrant separate description, namely: (a) *Salmon Patch*.—This is most commonly seen on the face and neck; it is confined to the surface of the skin and fades on pressure. It has a tendency to decrease as the child grows. Residual discoloration is best treated by radiotherapy or surface diathermy. (b) *Port Wine Stain*.—This deep purple blemish which remains constant in size throughout life may occur on any part of the body (Fig. 531). Its surface is usually flat, but sometimes it is nodular in places. Treatment is often unsuccessful, but if the lesion is unsightly and confined to the surface, the diathermy electrode, radiotherapy or radioactive paint (thorium X) may be tried. Excision and subsequent skin grafting is advisable if the above treatment is ineffective. (c) *Strawberry Mark*.—This lesion is raised above the skin surface and appears as an uneven pink red area on any part of the body; the lesions are usually multiple. Their growth is rather unpredictable for many grow rapidly for a time and then disappear. Occasionally they become ulcerated and bleed. Coagulation with a diathermy needle at several points is effective. If the angioma is ulcerated and exposed to irritation excision is advisable.

**Cavernous Hæmangioma.** This condition which affects the skin and deeper structures is soft and compressible, suggesting larger blood spaces than in the above-mentioned capillary conditions (Fig. 532). Those situated on the face and neck become more swollen if the veins of the neck are digitally compressed or the patient congests the venous outflow by coughing or straining. They are liable to become infected and ulcerated. Thrombosis of their deeper parts may lead to a diminution in size and regression of the swelling. Cavernous nævi in infants seem to increase in size soon after birth, before receding, although their behaviour is unpredictable. Tissues and organs, other than the skin, may be involved, such as muscle, bone, liver, intestine and the kidneys. Small accessible cavernous lesions are best treated with the diathermy needle or radiotherapy. Large lesions are treated by radiotherapy. As irradiation is liable to interfere with the development of growing tissue in children,



FIG. 531. Port-wine stain. (Mr. Anthony Green's patient.)



(a)



(b)

FIG. 530. Superficial capillary nevus: (a) Before superficial X-ray therapy. (b) After treatment. (Dr. Clive Sims' patient.)

especially epiphyseal growth and breast tissue, its application is limited. Excision, with due regard to important surrounding nerves and blood vessels, may be possible in some instances.



FIG. 532. Multiple cavernous naevi showing various stages of regression and scarring. (*Mr. R. Battle's patient.*)

**Arterial Hæmangioma.** The presence of arterial pulsation in a nævus is diagnostic of this type of angioma. Ulceration may occur and lead to severe hæmorrhage. Urgent treatment—blood transfusion and excision—is then required.

**Diffuse Hæmangiomatous Giantism (*Weber-Klippel Disease*).** This rare condition usually affects the limbs, which appear swollen. The skin surface is usually irregular and shows some superficial vessels. There is also an associated increase in growth of the limb. The condition is caused by multiple arteriovenous communications leading to massive swelling of the affected limb. There is enlargement of the heart and increased pulse pressure produced by the shunting of the circulation. Treatment should be carried out before these effects on the cardiovascular system occur. Ligation of the main artery of the limb may be indicated. Other types of localised gigantism occur in hereditary œdema of the leg (*Milroy's disease*) and neurofibromatosis (*von Recklinghausen's disease*).

**Multiple Familial Telangiectasis.** This familial condition is known also as the *Osler-Rendu syndrome*. Angiomatous lesions are found on the face and oral mucous membrane in association with multiple vascular nævi in various viscera; the intestine is most often affected, and the stomach, liver and kidneys may also be involved. The syndrome presents with bleeding from the nose, hæmatemesis or mælæna. It is only after negative investigations for the commoner causes of bleeding that this syndrome is



considered. The cutaneous telangiectases will give a clue to the diagnosis. Other conditions such as hæmophilia and purpura have to be excluded. The treatment consists of cauterisation or excision of the bleeding area. In extensive lesions of the bowel resection may be necessary.

**Miscellaneous Hæmangiomas.** Angioma of the kidney is a rare cause of hæmaturia ; bleeding tends to be severe and may need urgent nephrectomy. Hæmangiomas are not uncommonly seen in the liver, either as multiple or solitary tumours ; rarely they require treatment. Vascular

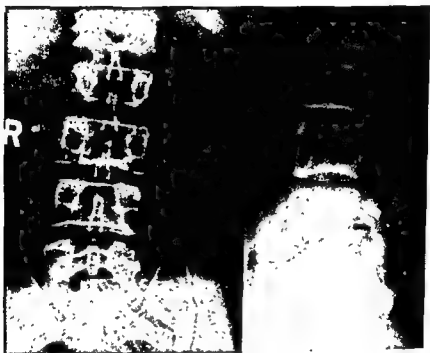


FIG. 533. Radiograph of the spine showing angioma of the body of the third lumbar vertebra. (Courtesy of Dr. J. M. Stewart.)

nævi in the region of the larynx are liable to cause suffocation ; they are best treated by diathermy, preceded by tracheotomy. Any part of the osseous skeleton may be affected by an angioma. Radiologically those in the long bones appear like a coalescent mass of "soap bubbles" and those in the skull present as "sun bursts." A vertebral lesion has a characteristic coarse trabeculated honeycomb appearance (Fig. 533). Bony hæmangiomas may be found accidentally or produce a pathological fracture. Most of the bony lesions respond to radiotherapy. Hæmangioblastomas of the cerebellum are discussed in Chap. 53, and vascular malformations of the cerebrum in Chap. 56.

## CHAPTER 77

### THE LYMPHATICS

THE extensive lymphatic network in the skin and subcutaneous tissues drains into main lymphatic trunks which follow the deep veins. The deep lymphatic vessels are supplied with numerous valves preventing reflux. Lymphatic channels are rarely seen with the naked eye, except for the lacteals, or lymphatic vessels in the mesentery of the small intestine; they are often visible on laparotomy as fine white lines. Lymphatic vessels may be outlined radiographically. Lymphangiography of the leg is performed by first injecting a dye, e.g. Patent Blue V (2 ml. of 11 per cent. solution), into the tissues of the sole of the foot. This stains the lymphatics blue, and on dissection in the region of the ankle deep lymphatic trunks can usually be recognised as blue lines. A cannula is introduced into one of them and an X-ray taken after injecting the vessel with diodone.

### INFLAMMATION

**Acute Lymphangitis.** This is due to hæmolytic streptococci or staphylococci, and may follow superficial sepsis, especially in the hand or foot. The primary infection is usually small and dry, but pus may be present. Superficial lymphatic channels become reddened, giving rise to fine red lines or a diffuse blush in the skin. There is tenderness along the inflamed lymphatics or in the reddened area. Enlargement of the regional lymph glands is usual, and constitutional symptoms, e.g. malaise, rigors and pyrexia are often severe. Response to treatment is usually rapid, but death occasionally results from septicæmia. *Treatment.*—A culture should if possible always be made from the original lesion and appropriate antibiotics given. The limb is rested and kept warm; attempts to produce lymphatic stasis by means of a light tourniquet (Bier's congestion) are no longer made. Incision of the original lesion or of abscesses forming in the lymphatic tracks may be required.

**Chronic Lymphangitis.** Thickened lymphatic channels can occasionally be felt draining the site of a primary syphilitic chancre. They may also be palpable in some forms of tuberculosis, e.g. lupus and tuberculous verruca. Lymphatics may become fibrosed after operations, probably as a result of infection. Fibrosed lymphatics at the elbow are shown in Fig. 534. They became both visible and palpable shortly after radical mastectomy and disappeared in a few weeks.

Oedema may result from obliteration of lymphatic channels by infection, and further attacks of lymphangitis may follow; this alternating infection and oedema leads to one type of chronic lymphœdema. *Erysipe-*

loid of *Rosenbach* is due to the *Erysipelothrix rhusiopathiae* and is a low-grade, localised lymphangitis of the skin, usually of the fingers. The swelling has a well-defined edge, is deep red and slightly indurated, but is neither hot nor tender. Spontaneous cure results in two to three weeks.

### LYMPHADENITIS

**Acute Lymphadenitis.** Palpable enlargement of the regional lymph glands is a common accompaniment of acute inflammation in their catchment area. The glands may be painful and tender. Usually they return to normal when the local condition resolves, but suppuration may occur, especially if there is underlying chronic disease of the glands, e.g. tuberculosis or malignant metastasis. Persistence of glandular enlargement following acute lymphadenitis usually results from continued activity in the source of infection. Acute generalised enlargement of the lymph glands occurs in certain pyrexial conditions, such as the acute exanthemata and glandular fever.

**Treatment.**—The focus of infection when still present is treated, but sometimes a primary infection cannot be found. In cervical adenitis, the search includes inspection of the scalp, mouth, throat, nose and ears; in inguinal adenitis, it includes the genitalia, umbilicus, perianal skin and skin between the toes. A kaolin poultice may relieve the discomfort of swollen glands. Chemotherapy or antibiotics may be necessary. Abscesses at the focus of infection or in the affected lymph glands are evacuated.



FIG. 534. Palpable lymphatics at the elbow after radical mastectomy. (Courtesy of Professor C. G. Rob.)

**Chronic Lymphadenitis.** (1) *Unresolved Acute Lymphadenitis (Syn. Chronic Septic Lymphadenitis).*—Chronically inflamed lymph glands are firm on palpation, but not stony hard. They become fixed to nearby structures at an early stage, and their size often varies with the activity of the primary focus. Abscesses may occur and be followed by the development of sinuses whilst the breaking-down gland itself is either absorbed or discharged. The cause of chronic enlargement of lymph glands is often impossible to determine on clinical examination alone. Biopsy is frequently necessary, otherwise serious disease may be missed.

**Treatment.**—The primary cause should be treated as necessary. Abscesses may need incision. A single persistently enlarged gland may be excised.

(2) *Tuberculous Lymphadenitis*. Tuberculous disease of the lymphatic system is fairly common. Although it may be secondary to an obvious primary focus, tuberculous adenitis may arise without symptoms of a primary lesion. Clinically the *cervical* glands are most commonly affected in this way, but calcified lymph nodes, i.e. healed tuberculous glands, are incidentally found in abdominal or chest X-rays in a large proportion of fit persons. In the *axilla*, tuberculous glands may be due to extension of disease from the cervical glands, from the breast or from the lungs; only very occasionally are other lesions not detectable. The *inguinal* nodes are rarely enlarged in tuberculosis, but when they are affected a primary lesion is usually obvious, e.g. a tuberculous joint or fistula-in-ano. Biopsy of the inguinal glands is of value in the diagnosis of tuberculous arthritis in the lower limb, for even if they are not enlarged the glands nearly always show histological evidence of tuberculosis. Patients complain of a painless swelling or a discharging sinus. On examination, a group of enlarged glands is usually found. The glands become matted together and adherent to the skin. Sometimes there is a diffuse enlargement of nodes in one or more regions. *Cold abscess* formation is frequent. Pyogenic abscesses, however, not uncommonly result from secondary infection of tuberculous glands. In either case, breakdown of the skin with sinus formation often occurs. Calcium is usually deposited in tuberculous glands after months or years. The usual regime for tuberculosis is prescribed. When glands are localised, and persistent, or cause sinus formation, they are best excised. Tuberculous lymphadenitis is more fully discussed in Chap. 67.

(3) *Syphilitic Lymphadenitis*. In primary syphilis the regional glands draining the site of infection become *painlessly* enlarged and feel firm and discrete on palpation. They are frequently termed "rubbery." In secondary syphilis, there is a generalised enlargement of all lymph glands, and at the elbow the epitrochlear is characteristically palpable. Tertiary syphilitic gummas may also cause regional node enlargement (gummatous adenitis).

(4) *Lymphogranuloma Inguinale*. This is a venereal disease caused by a virus, and begins two to three days after contact. It occurs predominantly in the tropics. The primary lesion is a soft ulcer or vesicle on the genitalia. Considerable enlargement of the inguinal glands occurs later, and chronic sinus formation is common. The lower part of the rectum is sometimes affected, either as the result of sodomy, or in the female as the result of pararectal spread of the disease from the vagina. A rectal fistula or stricture may develop later. *Frei's Test*.—This is positive in over 90 per cent. of cases; a papule, surrounded by erythema, results forty-eight hours after intradermal injection of sterilised pus from a proved case. A complement fixation test is also available. *Treatment* used to consist of antimony injections, but cure is now achieved by four weeks' treatment with Aureomycin or Terramycin. Incision of abscesses in this disease is

avoided, for secondary infection usually follows; instead, repeated aspiration should be carried out under strict aseptic conditions. Sinuses in this disease may persist for months or years. In severe rectal fistula and stenosis a colostomy may eventually be necessary.

## TUMOURS OF THE LYMPHATIC SYSTEM

### A. BENIGN TUMOURS

**Lymphangiomas.** *Capillary lymphangiomas* may occur in the tongue or lip as reddish swellings. In the skin they form flat or warty, yellow-



FIG. 535. Cystic hygroma of the neck. (Courtesy of Mr. G. H. Macnab.)

brown swellings and are sometimes associated with the intermittent discharge of lymph (lymphatic fistula, or lymphorrhœa). *Cavernous lymphangiomas* form more bulky tumours, but they are soft and ill-defined. They may appear in almost any region of the body. A *cystic hygroma* consists of a multilocular cystic mass containing moderately clear fluid (Fig. 535). The swelling is usually found in infants, sometimes being noticed at birth, but its occurrence in adult life is known. Cystic hygromas are most commonly situated in the lower part of the neck; it has therefore been suggested that they originate from sequestration of the primitive endothelium of the jugular sac. They may, however, occur in the axilla. On inspection, the tumour is semi-transparent and transilluminates well. It has indefinite borders and infiltrates the tissue planes. Cystic hygromas

are sometimes fatal, for respiratory obstruction may be produced by submucosal cysts in the larynx. *Treatment.*—Lymphangiomas can usually be excised. They do not respond to radiotherapy. Removal of a cystic hygroma may be very difficult owing to its many extensions and adherence to vital structures. A few months' observation is advisable, for spontaneous regression often occurs. Otherwise, removal may be attempted, but this is best avoided until the child has reached its second year. Aspiration followed by injection of sclerosants is rarely of lasting value, but needling of laryngeal cysts may be required for respiratory obstruction. Tracheotomy is called for on rare occasions.

**Lymphoma.** This is a very rare encapsulated tumour occurring in the tonsil or lymphatic glands. It consists of fully developed lymphocytes. It does not recur after excision.

### B. MALIGNANT TUMOURS

In the primitive mesenchyme, the stem cells become differentiated into lymphocytes and reticulo-endothelial cells. Malignant diseases may develop from either.

**Lymphosarcoma.** This tumour usually begins in a group of lymph glands, but it may occur in lymphoid tissue anywhere; its origin in the alimentary tract in Peyer's patches, although rare, is of surgical importance. Lymphosarcoma eventually becomes disseminated throughout the lymphatic system, but whether this distribution is due to a multifocal origin or metastasis is uncertain. Lymphosarcoma attacks predominantly people of middle and old age. Young people may also be affected; the disease then runs a more rapid course. Diagnostic biopsy is necessary, but block removal of glands is not indicated. Deep X-ray therapy to the affected areas causes a remission of some months and cures have been reported. Resection of lymphosarcomatous tumours in the intestine sometimes results in cure.

**Hodgkin's Disease.** Hodgkin's disease, formerly thought to be an atypical form of tuberculosis, is now considered to be a tumour of reticular tissue. It affects males more than females, usually appearing between the ages of thirty and fifty; it is also seen in infancy and old age. The disease first attacks a single group of glands; in 80 per cent. of cases the cervical region is first affected (Fig. 536). Later, however, it becomes generalised; distant lymph nodes become involved and deposits may be found in the spleen, liver, bones and epidural fat. Deposits sometimes occur in the lungs and skin. Histologically, there is loss of normal architecture and collagen fibres are seen dividing the gland into lobes. Fine collagenous fibrils may also be noted. The reticulum cells are greatly increased in number and various forms of giant cell are apparent, including the Sternberg-Reed cells with their characteristic mirror-image nuclei. The number of granulocytes is increased, especially the eosinophils. Fibrosis later occurs at the diseased sites. Blood examination

shows a moderate leucocytosis, together with an eosinophilia in some cases ; often there is also a hypochromic anaemia.

Affected glands may grow to a great size, but they remain discrete and unattached. At first they are rubbery in consistency, but become harder as fibrosis develops. Bouts of pyrexia occur at intervals of a few weeks (Pel-Ebstein curve). On X-ray examination of the chest mediastinal glandular enlargement may be visible, and "punched-out" spaces may be seen in X-rays of the bones. The diagnosis rests on gland biopsy. The

progress of the disease is spasmodic, occurring in bouts as further tissues become involved. Death usually occurs from three to seven years after the onset of the disease.

**Treatment.** Surgical excision, though inviting, is usually without reward. Arsenic used to be given, but it is now hardly ever used. A course of nitrogen mustard sometimes proves temporarily effective. Deep X-ray therapy causes a marked regression of the disease, but it is hardly ever curative and when applied for recurrences it becomes less effective. **Complications.**—Anaemia occurs in most cases and therefore iron therapy or blood transfusion may be required. Pruritus may be troublesome and is somewhat alleviated by a phenol lotion, or cortisone (200 mg. daily). Bronchial obstruction from enlarged media-



FIG. 536. Hodgkin's disease of the neck.  
(Courtesy of Dr. M. H. E. Hulbert.)

stinal glands may lead to bronchopneumonia. Deposits in the extradural region may result in paraplegia.

**Reticulosarcoma.** Diagnosis is made after biopsy of an enlarged lymph gland or following intestinal resection for an obstructing tumour. Although the tumour is radiosensitive the outlook is poor.

**Secondary Tumours.** Malignant disease of the lymphatic system is most commonly due to metastasis from carcinoma. The lymphatic channels may be intact in such cases, although a hard lymphatic cord is occasionally palpable. The glands are stony hard ; at first they are mobile, but later become fixed. As a result of their attachment, vascular or neurological symptoms may be produced. Lymphoedema often results at the periphery. When malignant glands are rapidly growing, softening

may occur, with skin necrosis and sinus formation. In the neck, multiple sinuses of this kind may lead to confusion with actinomycosis. In malignant melanoma, invasion of the lymphatic system is frequently early. Sarcomas attack lymph nodes less commonly, but the possible metastasis of lymphosarcoma and primarily reticular tumours has already been mentioned. *Diagnostic Biopsy.*—When the situation of the primary disease is uncertain or unknown, biopsy of a secondary malignant lymph node may reveal the nature of the growth.

*Treatment.* The block removal of secondary malignant lymph nodes should be performed only if they are still unfixed to the deep structures and if there is no general dissemination of the disease. Block dissection of the cervical glands may be required for cancerous growths of the tongue, pharynx and larynx (see Chap. 67). Lesser bilateral resections above the hyoid bone, for growths of the lip or floor of the mouth, may be performed through a curved incision in this area. Clearance of the axilla is described in connection with carcinoma of the breast; axillary dissection may also be necessary in cases of epitheliomas of the skin. Inguinal block dissection is required for some cases of cancer of the vulva, urethra and anus. Irremovable glands may be treated by deep X-rays.

#### LYMPHŒDEMA

Lymphœdema is an excessive collection of fluid in the subcutaneous tissue spaces as a result of a local disturbance of venous or lymphatic function. Often it is due to a combination of both venous and lymphatic stagnation. Although in the past it has been usual to differentiate venous from lymphatic œdema it is now considered that such a distinction must often be inaccurate and that both types should be considered together. The term lymphœdema does not apply to œdema of cardiac, renal or nutritional origin.

**Acute lymphœdema** follows trauma, infections and operations and usually resolves when inflammation subsides or the wound heals.

**Chronic Lymphœdema.** In the leg, the ankle and calf are usually most severely affected, but the whole limb may be very swollen (Fig. 537). In the upper limb, the swelling usually begins in the humeral region and shoulder. Chronic lymphœdema causes pallor of the limb and there may be subcutaneous blisters of lymph. Ulceration occurs in some cases. At first the œdema is of the pitting type and disappears if the limb is elevated. After months or years, the œdema becomes brawny and ceases to pit, although it may still partly subside on elevation. In the soft tissues there is an increase in lymphocytes, and often lymph follicles may be seen. Fibrin is deposited in the interstitial spaces, and eventually there is marked fibrosis. When fibrosis is established, the limb feels wooden and the skin wrinkles. The term "*elephantiasis*" is applied to this state. In purely lymphatic obstruction any ulceration tends to remain superficial. Occasionally a temporary lymphatic fistula is produced.



**Venous Causes of Lymphœdema.** *Occlusion* of peripheral veins causes increased production of lymph and overloading of the lymphatic circulation. Proximal venous obstruction (e.g. at the thoracic inlet) may in addition cause interference with the drainage of lymph back into the venous circulation. In the leg, *venous incompetence*, with its associated back-pressure, is also a cause of lymphatic stasis which is more serious when deep veins are incompetent than when only the saphenous system is affected. The increased

venous pressure in congenital or traumatic arteriovenous fistula may also result in lymphœdema.

**Lymphatic Causes of Lymphœdema.** A. *In the Lymphatic Vessels.*—Lymphœdema due to disease of lymphatic channels may be congenital or acquired. *Congenital Diseases.*—Milroy's disease is familial. The œdema becomes severe early in life, either in infancy or before puberty. Although its cause is uncertain, malformation of the lymphatics is a possible cause. Any limb may be affected and the swelling has a well-marked upper limit. By means of lymphangiography, lymphangiectasis or lymphatic dilatation has been shown to be the cause of some forms of peripheral œdema. The condition previously termed "spontaneous"



FIG. 537. Chronic lymphœdema of uncertain cause. Bandaging below the knee has reduced the size of the right leg. (Courtesy of Professor C. G. Rob.)

neous" lymphœdema or lymphœdema precox, is probably of this nature, being due to incompetent or valveless lymphatics which may also be abnormally few in number. An excessive number of incompetent lymphatics occurs in congenital macroglossia (hypertrophy of the tongue) and macrocheilia (hypertrophy of the lip). Chylous œdema refers to a condition in which blebs of milky fluid are to be seen under the skin, sometimes with fistula formation. It is due to reflux in the abdominal lymphatics and, though rare, it is of some interest as ligation of lymphatic vessels in the femoral region or pelvis may result in cure. Congenital constriction rings, of unknown cause but previously thought to be due to amniotic bands, produce circular scars with lymphœdema below. In lipœdema there is an overgrowth of adipose tissue in conjunction with the œdema, and this

condition is painful. Neurofibromatosis may be congenital and associated with overgrowth of fibrous tissue in the limbs, together with lymphatic obstruction and elephantiasis. *Acquired Diseases.*—The severe forms of erythrocyanosis frigida may be followed by lymphœdema, and this is probably the commonest cause of moderate swelling. Long-standing œdema may follow fractures and also circumferential wounds, e.g. burns of the fingers. Lymphœdema may follow erysipelas or recurrent attacks of lymphangitis (Fig. 538). It may follow lymphogranuloma inguinale.

Parasitic lymphœdema is due to the *Filaria sanguinis hominis*. In this disease, œdema is due partly to blockage of the lymphatics by the worms, and partly to the inflammatory reaction which obstructs the remaining channels. The most severe elephantiasis follows this disease and natives have used wheelbarrows to transport the scrotum. Lymphœdema may follow operations involving removal of much soft tissue. It may also result after treatment with X-rays or radium. Choking of distal lymphatics with growth may lead to lymphœdema, although in most cases of malignant disease the swelling is due to gland blockage (*vide infra*). Lymphœdema artefacta is commoner than might be thought, patients deliberately producing œdema by wearing tight bands which are removed before seeing the doctor.



FIG. 538. Lymphœdema due to recurrent lymphangitis. (Courtesy of Professor C. G. Rob.)

**B. In the Lymphatic Glands.** Lymphœdema often follows advanced malignant adenopathy. It may also follow operative removal of glands, or their treatment by radiotherapy. The operation of radical mastectomy (which includes removal of the axillary glands) does not by itself lead to lymphœdema of the arm as the lymphatics lateral to the axillary vein are preserved. Gummatous adenitis is a very rare cause of œdema.

**Management of Lymphœdema.** Of first importance is the search for the cause (*vide supra*). Specific treatment is then undertaken when possible. *Corrective Treatment for Oedema.*—Early treatment is essential if œdema is not to become permanent. It consists of antigravitational measures and bandages. In severe œdema of the leg, the patient is first

**Venous Causes of Lymphœdema.** *Occlusion of peripheral veins* causes increased production of lymph and overloading of the lymphatic circulation. Proximal venous obstruction (e.g. at the thoracic inlet) may in addition cause interference with the drainage of lymph back into the venous circulation. In the leg, *venous incompetence*, with its associated back-pressure, is also a cause of lymphatic stasis which is more serious when deep veins are incompetent than when only the saphenous system

is affected. The increased venous pressure in congenital or traumatic arteriovenous fistula may also result in lymphœdema.

**Lymphatic Causes of Lymphœdema.** A. *In the Lymphatic Vessels.*—Lymphœdema due to disease of lymphatic channels may be congenital or acquired. *Congenital Diseases.*—Milroy's disease is familial. The œdema becomes severe early in life, either in infancy or before puberty. Although its cause is uncertain, malformation of the lymphatics is a possible cause. Any limb may be affected and the swelling has a well-marked upper limit. By means of lymphangiography, lymphangiectasis or lymphatic dilatation has been shown to be the cause of some forms of peripheral œdema. The condition previously termed "spontaneous"



FIG. 537. Chronic lymphœdema of uncertain cause. Bandaging below the knee has reduced the size of the right leg. (Courtesy of Professor C. G. Rob.)

lymphœdema or lymphœdema precox, is probably of this nature, being due to incompetent or valveless lymphatics which may also be abnormally few in number. An excessive number of incompetent lymphatics occurs in congenital macroglossia (hypertrophy of the tongue) and macrocheilia (hypertrophy of the lip). Chylous œdema refers to a condition in which blebs of milky fluid are to be seen under the skin, sometimes with fistula formation. It is due to reflux in the abdominal lymphatics and, though rare, it is of some interest as ligation of lymphatic vessels in the femoral region or pelvis may result in cure. Congenital constriction rings, of unknown cause but previously thought to be due to amniotic bands, produce circular scars with lymphœdema below. In lipœdema there is an overgrowth of adipose tissue in conjunction with the œdema, and this

## SECTION XI

### ORTHOPÆDIC SURGERY

by  
J. G. BONNIN

#### CHAPTER 78

#### CONGENITAL DEFORMITIES

CONGENITAL deformities may be due to a variety of causes, some of a specific nature, others of a more general nature. They may be subdivided as follows: (1) Hereditary causes; (2) intrauterine glandular deficiency, e.g. cretinism; (3) embryological maldevelopments, e.g. meningocele; (4) maternal disease during pregnancy, e.g. syphilis and rubella; (5) intrauterine malposition, e.g. club foot; (6) intrauterine trauma.

**Heredity.** Alterations in the developmental pattern are due to mutations of the genes. These may occur spontaneously. Most mutations are recessive, but the longer a mutation lasts in a family tree, the more it tends to become dominant. We may accordingly separate three groups of hereditary diseases: (1) *Recessive*.—The condition appears in one or more siblings, but dies out in their offsprings, e.g. metatarsus primus varus—one cause of hallux valgus, osteopetrosis, diaphysial aclasis, anomalies of hands and feet. (2) *Dominant*.—Transmitted by parents to some of their children and by them to others, e.g. fragilitas ossium, Mongolian idiocy, congenital dislocation of the hip, achondroplasia. (3) *Sex-linked*.—Transmitted by normal females to male offsprings, e.g. hæmophilia. Often the familial incidence of a disease and spontaneous mutation of a gene (giving rise to sporadic cases) produce an almost equal number of cases, e.g. achondroplasia, diaphysial aclasis, cleidocranial dysostosis.

**Achondroplasia.** The individual has an otherwise normal body, but the long bones fail to grow in length. The facial bones are reduced in size and the forehead is consequently disproportionately large and flat, and the nose short and saddle-shaped. The fingers are stumpy. There is no mental defect. Its transmission as a dominant characteristic can be studied in the dachshund, which owes its form to this condition.

**Diaphysial Aclasis.** Failure of normal development of the epiphyseal plate gives rise to three main groups of deformity: (1) Islands of cartilage

kept in bed with the leg elevated and the foot of the bed on blocks. In resistant cases a trial of two weeks should be given. As soon as the swelling has subsided bandages are applied as described for venous ulcers, except that in lymphœdema it is often necessary to extend them up to the groin. Lace-up shoes should be worn and patients should sleep with the foot of the bed raised. Although drainage by Southey's tubes may be of value in cardiac and renal œdema, there is no place for this treatment otherwise.

*Surgical Methods.* Pressure bandages are the mainstay of treatment and resort to surgery should only be made in long-standing cases of œdema of the lower limb in patients where physical activity is restricted. Excision of the hypertrophied and œdematous tissue may, if necessary, be performed throughout the whole of the lower limb and scrotum. Some skin may survive, but extensive skin grafting is usually required. The results are often most gratifying. Attempts have been made to re-establish the lymphatic circulation by encouraging the growth of new lymphatic channels, but the results have not been convincing. Methods that have been tried include the insertion of strands of silk beneath the skin, the excision of sheets of deep fascia (Kondolœon's operation), and the transplantation of flaps of skin and fascia to bridge the groin or axilla. Sympathectomy is of no value except in post-thrombotic œdema when the legs are cold. Amputation may be called for in patients with extremely heavy legs which interfere with activity, and also in cases of persistent ulceration and infection.

*Trauma.* Lymphatic pathways are numerous, and many may be divided without effect, but encircling wounds of the limbs are often followed by œdema. The thoracic duct may be damaged in operations on the neck or the thorax; damage has also been reported in association with fractures of the thoracic vertebræ. In the neck, an external fistula may result; in the chest an effusion of lymph may occur in the pleural cavity (chylothorax) and in the abdomen the lymph may pool in the peritoneal cavity (chylous ascites).

*Lymphatic Fistula.* Traumatic fistulas have already been mentioned. Congenital lymphatic fistulas may occur in the limbs, but the lymphorrhœa is usually intermittent. External spontaneous fistulas occasionally result in cases of chronic lymphœdema. Malignant disease, such as carcinoma of the œsophagus, may result in chylothorax or chylous ascites after the formation of an internal fistula. Lymph has a high protein content and therefore the serum proteins may become deficient, especially with external fistulas. Fistulas due to wounds should be treated by operation. The duct is dissected free and ligated. It is unnecessary to implant the duct into a vein for an adequate collateral lymphatic circulation always exists or develops later. Chylothorax and chylous ascites are best treated by repeated aspiration.

the sclera is thinned or translucent, and the sclerotics appear blue, and there may be accompanying laxity of ligaments. Otosclerosis develops in many patients between twenty and forty years of age. The disease is very variable in time of onset and severity, but may be roughly divided into three: (1) The fetal type in which there are multiple fractures with gross deformities; the infants are often stillborn. (2) The infantile type; fractures may cease to occur after puberty. (3) The late adolescent type in which the fractures do not occur till later in life. In all, there is osteoporosis and lightly built bones usually associated with poor physique. The fractures unite normally, but their multiplicity, and the softening of the bones sometimes produce gross distortion (Fig. 541).

**Neurofibromatosis.** This frequently shows a familial incidence, and is accompanied by bone changes, notably kyphoscoliosis, but changes in the length of limbs may be present, and hyperosteoses may distort the skull or face (see also Chap. 61).

**Embryological Maldevelopments.** These cover a very wide range. The degree of the final development of an affected limb suggests that the abnormality must often be the result of interference with development at an early stage in embryological life. The white race shows double the number of congenital abnormalities compared with the black races. There is an equal incidence of malformations in the first four children of a marriage, and after the fourth child it rises. There is an increased incidence in children born of mothers over forty years or who have passed through a long period of sterility.

Failure of the neurenteric canal to close produces a range of deformities in the lumbar spine (see also Chap. 59). In the most severe, the neural canal lies open on the skin throughout its length. This is inconsistent with life. In the less severe forms, the membranes may protrude alone (meningocele) or may contain part of the cord (myelomeningocele). These deformities may be consistent with life if normal skin can be closed



FIG. 540. Cleidocranial dysostosis. Note squaring of the forehead and the approximation of the shoulders made possible by absence of the clavicles.

become isolated in the shaft of the bone and produce multiple enchondromas. More commonly a single enchondroma is seen, but its origin is similar. (2) Islands of cartilage become isolated on the surface of the bone and give rise to exostoses, covered by cartilaginous caps. These exostoses cease to grow when general growth ceases. They are most

common around the knee, and may be solitary. The bone ends from which they arise preserve their normal shape. (3) Not only do islands of cartilage become separated both in the bone and on the surface, but there is a general failure of modelling of the bone ends and gross deformities develop. The condition may be limited to one joint, affected symmetrically, or many bones and joints may be distorted (diaphysial aklasis—Fig. 539).

The treatment in all cases is one of expediency. Localised enchondromas should be removed, and the cavity carefully curetted, and then packed with cancellous bone. Multiple enchondromas may produce such deformities of the fingers that their preservation is useless, and amputation has to be recommended. Solitary exostoses are removed. This is usually a simple procedure as the play of tissues over exostoses produces a bursa around them inside which they can be readily defined and removed with an osteotome. In diaphysial aklasis, surgery is limited to removing exostoses which affect function, interfere with bone growth, or cause unsightly deformities. Serious interference with joint function and later arthritic changes are inevitable.

**Osteopetrosis (Marble Bones).** The ends of the long bones are chiefly affected. There

are alternate bands of dense and normal bone. The bones are brittle as in Paget's disease, and transverse fractures occur. The severity of the disease is very variable.

**Cleidocranial Dysostosis.** This rare disease produces combined cranial and clavicular defects. The absent clavicles permit approximation of the shoulders, but interfere little with their function (Fig. 540).

**Osteogenesis Imperfecta (Fragilitas Ossium).** This disease may be familial or sporadic. There is some general defect in mesoblastic tissue formation, so that not only bone is malformed, but the fibrous tissue of



FIG. 539. Diaphysial aklasis. A shortened forearm is also seen in achondroplasia, but there is no deformity of the bone. Note the expansion of the ends of the bones and cystic changes at the distal end of the ulna.

is developing rapidly; ocular, cardiac and other defects may occur. Infection later on is not so serious.

**Congenital Syphilis.** Infection of the mother usually results in a series of miscarriages. If the child survives, it may appear normal for the first few months and then show signs of syphilitic marasmus. The condition is increasingly uncommon to-day, and either as the result of treatment, or increasing resistance in the community, the disease may not become apparent till later in life. In the skeletal system it may show itself in the following ways: *Syphilitic Epiphysitis* (Fig. 542).

—During the first year of life, this may lead to epiphyscal separation. There is a line of rarefaction running across the dense metaphyscal end of the shaft. *Syphilitic Periostitis*.—In the skull this is known as Parrot's nodes and produces the "hot cross bun" appearance around the fontanelle. It may also produce distortion of the tibia (sabre tibia). *Syphilitic synovitis* may accompany epiphysitis or occur alone. Chronic *syphilitic synovitis* commonly affects both knees, when it is termed *Clutton's joints*. *Disturbances of Dentition*.—Moon's turreted molars, peg-top teeth and Hutchinson's notched incisors are classical signs of congenital syphilis. As with acquired syphilis, gummas and

parasyphilitic manifestations may appear. [*Acquired Syphilis*.—The bones are not affected till the tertiary stage of the disease. A diffuse periostitis may occur, thickening the shaft of a long bone—most commonly the tibia. Gummas also occur, either as a localised central area of destruction surrounded by a dense zone, or as a diffuse gummatous infiltration, in which there are many coextensive cavities and general rarefaction leading perhaps to pathological fractures.]

The effects of intrauterine malposition are described in the discussion on the origin of club feet. Malposition *in utero* may also play a part in the production of some forms of dislocation of the hip. Maldevelopment itself, however, may lead to malposition *in utero*.

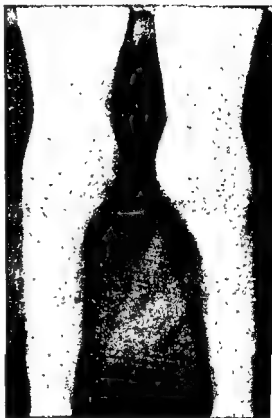


FIG. 542. Syphilitic epiphysitis of the lower end of both femora in a congenital syphilitic boy aged seven. The swelling of the joints suggests effusion into both joints although there was none.



over them to prevent ulceration and consequent meningitis ; they are frequently accompanied by congenital deformities of the feet. In the most innocent form (*spina bifida occulta*) there is failure of the spinous process to develop normally, often indicated by a fatty tumour or excessive hair. There may be a palpable depression which may arouse suspicion of spondylolisthesis, and require radiography to exclude it. The lower lumbar spine is very liable to minor abnormalities in the number of vertebrae, and their relation to the sacrum. Sacralisation of one or both transverse processes of the fifth lumbar vertebra, or absorption of one vertebra into the sacrum or the lumbar spine is common, and is important in the consideration of prolapse of intervertebral discs.



FIG. 541. Severe osteogenesis imperfecta; note the marked osteoporosis and grossly deformed pelvis. The patient's first fracture occurred at the age of eight and the last at the age of twelve. She suffered ten fractures in all, and was a dwarf 4 feet in height.

Some of the abnormalities of the hand and foot are described in other chapters. The most severe defects, sometimes described as amniotic amputations, may result in the loss of the distal half of an extremity, or a limb may fail to develop at all, being represented by a deformed remnant or dimple. Extra digits, or rudimentary limbs, can be readily removed. Defects in limb length and size are common. In congenital coxa vara the neck of the femur may be almost absent, and the condition may be unilateral or bilateral. Failure of development of the tibia may produce bowing, or an osseous defect in its lower third ; such a gap is particularly hard to fill by grafting. These defects in internal limb pattern arise later in development ; it has therefore been suggested that they are due to localised failure of the blood supply to the area involved.

Allied to embryological disturbances is the effect of maternal disease such as rubella during the early part of pregnancy, i.e. when the embryo

is developing rapidly; ocular, cardiac and other defects may occur. Infection later on is not so serious.

**Congenital Syphilis.** Infection of the mother usually results in a series of miscarriages. If the child survives, it may appear normal for the first few months and then show signs of syphilitic marasmus. The condition is increasingly uncommon to-day, and either as the result of treatment, or increasing resistance in the community, the disease may not become apparent till later in life.

In the skeletal system it may show itself in the following ways:

*Syphilitic Epiphysitis* (Fig. 542).

—During the first year of life, this may lead to epiphyseal separation. There is a line of rarefaction running across the dense metaphyseal end of the shaft. *Syphilitic Periostitis*.—

In the skull this is known as Parrot's nodes and produces the "hot cross bun" appearance around the fontanelle. It may also produce distortion of the tibia (*sabre tibia*).

*Syphilitic synovitis* may accompany epiphysitis or occur alone. Chronic syphilitic synovitis commonly affects both knees, when it is termed *Clutton's joints*. *Disturbances of Dentition*.—Moon's turreted molars, peg-top teeth and Hutchinson's notched incisors are classical signs of congenital syphilis. As with acquired syphilis, gummas and

parasyphilitic manifestations may appear. [*Acquired Syphilis*.—The bones are not affected till the tertiary stage of the disease. A diffuse periostitis may occur, thickening the shaft of a long bone—most commonly the tibia. Gummas also occur, either as a localised central area of destruction surrounded by a dense zone, or as a diffuse gummatous infiltration, in which there are many coextensive cavities and general rarefaction leading perhaps to pathological fractures.]

The effects of intrauterine malposition are described in the discussion on the origin of club feet. Malposition *in utero* may also play a part in the production of some forms of dislocation of the hip. Maldevelopment itself, however, may lead to malposition *in utero*.



FIG. 542. Syphilitic epiphysitis of the lower end of both femora in a congenital syphilitic boy aged seven. The swelling of the joints suggests effusion into both joints although there was none.

## CHAPTER 79

### HÆMATOGENOUS OSTEOMYELITIS

This form of osteomyelitis is distinguished only by its origin from that due to direct bone infection such as may follow compound fractures. Hæmatogenous osteomyelitis is usually a disease of the active years of life occurring before the epiphyses have united, though rare cases arise in adults. The bone infection is blood-borne from some pre-existing focus. Common examples of primary foci are impetigo, infected blisters, furunculosis, tonsillitis, and minor infected wounds. The organism encountered most commonly is the *Staphylococcus pyogenes* (85 per cent.). Next in frequency is a streptococcus, and very rarely *B. typhosum*, *pneumococcus*, and *H. influenzae*. The site of commencing infection is usually the metaphysis. It is considered that this part of the bone is most frequently attacked because it is the site of minor injury, and because organisms tend to settle in a focus of diminished resistance aided perhaps by hairpin bends in the growing blood vessels. The influence of trauma is shown by the sex incidence, for the disease is much more common in boys, and is rare in children under the age of two, when trauma is minimal. When it does occur in infants it often pursues a different course, and the infecting organism is more commonly a streptococcus.

**Pathology.** The organisms are filtered from the blood stream, and form an abscess just below the epiphyseal plate, i.e. in the cancellous bone of the metaphysis. From here the infection spreads in various directions largely determined by the anatomy. It spreads under the epiphyseal plate to the surface of the bone. As the periosteum is attached to the epiphyseal plate, it necessarily forms a subperiosteal abscess, and may track widely down the shaft of bone under this membrane. Though to some extent restricted, the escape of pus under the periosteum is a form of decompression, and is often accompanied by a fall in temperature. Pus may spread down the medullary canal of the bone and out through Haversian canals to the subperiosteal space or less commonly in the reverse direction. It is most uncommon for it to spread through the epiphyseal plate to the joint, though when the epiphysis is intracapsular (e.g. the hip) the joint may be involved. Spread to the joint may also occur through the soft tissues.

The special features of osteomyelitis as compared with other inflammatory diseases are due to the solid nature of the tissue involved. Thus compression of the vessels against the bone occurs when there is a rise in pressure in the bone, though vascular occlusion is more commonly due to thrombosis produced by diffusion of toxins. The solid nature of the tissue

makes its absorption slow and difficult, especially if there is devitalisation of large fragments of bone. The destruction of bone leaves a space which cannot fill and forms a rigid-walled abscess cavity from which there is a continuous or intermittent discharge. Although the pathological changes are important, the radiological appearances are even more important, for it is on these that diagnosis and treatment are often based.

### *Pathology*

**Subperiosteal New Bone.** This occurs first at the margins of the subperiosteal abscess where the circulation in the periosteum is least disturbed.

**Osteoporosis.** As a result of microscopic death of bone with absorption, the bone is thinned.

**Sequestration.** Macroscopic death of bone leaves a fragment of dense bone standing out from the surrounding osteoporotic bone, and when separate is surrounded by pus and granulation tissue.

**Sclerosis.** Two types have to be distinguished. The slow thickening of chronically inflamed bone due to new bone deposits, commonly subperiosteal; and the increased density of a sequestrum due to the deposit of calcium salts in it.

### *Radiological Appearances*

A thin triangle of bone becomes apparent on the surface of the bone. This is not usually visible till the seventh to tenth day, and is often the first radiological change seen in long bones.

Thinning of the bone may be the first radiological sign of osteomyelitis of a cancellous bone.

There is patchy osteoporosis, in which lie larger loculi. In these may be seen dense fragments of bone, the sequestra. The whole metaphysis of a bone may form a sequestrum. New bone formation from the periosteum around a sequestrum produces an irregular containing cage, the *involucrum*.

In subperiosteal new bone formation the whole circumference of the bone may become dense. When sequestra incite a foreign body reaction, calcium is deposited and they become very dense, and the walls of the cavity in which they lie are well defined.

Hæmatogenous osteomyelitis may be divided into three clinical types : (1) **Fulminating Osteomyelitis.**—This is due to an overwhelming blood-borne infection in which septicæmia dominates the picture, and death commonly occurs before there is any evident localisation in bones. The blood culture is positive. Administration of large doses of antibiotics is the only hope of saving life. (2) **Acute Osteomyelitis.**—Following a

transitory bacteriemia, the infection is localised in one or more bones. The blood culture may be positive on one or two occasions (Fig. 543). (3) *Subacute Osteomyelitis*.—This is due to infection with attenuated organisms, or to the patient's high resistance. It occurs in typhoid fever, in which the infection of the bone is often mild and subperiosteal. Subacute clinical and radiological features are now seen more frequently in osteomyelitis from other organisms, treated early with adequate doses of antibiotics.



FIG. 543. Osteomyelitis of the humeral metaphysis, resulting in a pathological fracture. Note the drill holes made for decompression of the medullary cavity.

**Clinical Features of Acute Osteomyelitis.** These consist of the general manifestations of a severe infection: raised temperature ( $103^{\circ}$  to  $104^{\circ}$  F.), a rapid pulse and a flushed and ill child. There is a leucocytosis of 12,000 to 30,000. The blood culture may be positive. The local signs which precede the general signs by a variable interval which is related to the severity of the infection, consist of swelling, heat, redness and tenderness around an epiphysis; the epiphyses around the knee are most commonly involved. The nearness of the swelling to the joint may make the distinction from pyogenic arthritis difficult, but in osteomyelitis, if the limb is handled gently, a small range of painless movement of the joint can be determined. This excludes direct involvement of the joint. Oedema may be extensive, and is co-existent with pus formation. Later fluctuation can be demonstrated.

**Differential Diagnosis.** *Acute Arthritis*.—When in doubt between a serous effusion into the joint and frank infection, the joint may be aspirated, but not through an infected area. *Cellulitis*.—This may be difficult to distinguish. The toxæmia is less than in osteomyelitis; the infection is often not related to an epiphysis; the bone is not tender to jarring. No radiological changes appear. On draining such an abscess dead bone cannot be detected. *Note*.—Pyogenic bursitis may secondarily involve underlying bone. *Rheumatic Fever*.—There is involvement of one large joint after another. The child sweats profusely. Confusion arises at the beginning of the illness when a single joint is affected. When there is doubt, a wide spectrum antibiotic should be given and a medical opinion sought. *Poliomyelitis*.—The fever accompanied by pain and tenderness

in the muscles around a joint may arouse the suspicion of osteomyelitis. A pleocytosis in the C.S.F. and the onset of paralysis leave no doubts about the diagnosis. More commonly subacute osteomyelitis, or osteomyelitis in a deep situation, e.g. around the hip, or the spine, is mistaken for poliomyelitis. *Osteogenic Sarcoma*.—This when rapidly growing may arouse suspicion of acute osteomyelitis. Epiphyseal separation in *scurvy* or *syphilis* may be mistaken for osteomyelitis. The differential diagnosis is often rendered more difficult by the reduction in activity of osteomyelitis by antibiotics. In these cases it may be necessary to wait for radiological changes or localisation of pus to confirm the diagnosis. This, however, is no reason for withholding antibiotics till the diagnosis is established, for it is in the early stages of the disease that these are most effective.

**Treatment.** This consists first of all of antibiotics adequate for the age and severity of the infection, and complete local rest to the affected part by means of splinting (e.g. leg traction) and bed rest. Treatment should not await identification of the organism; penicillin is given, for it is effective against the usual causal organisms. A large dose is followed by repeated intramuscular doses at four- or six-hourly intervals (see Antibiotic Therapy, Chap. 110). The response is carefully judged by reference to a four-hourly pulse and temperature chart. Usually there is a rapid improvement in the patient's general condition, and a subsidence of pulse rate and temperature. Failure to respond is due to insensitivity of the organism to penicillin (an event of increasing frequency), or toxic absorption from pus under pressure. The latter is indicated by increasing local swelling and œdema. If this is so, operation is indicated. An incision is made down to the bone; any pus is evacuated, and a few drill holes are made starting close to the epiphysis and working along the shaft till medullary blood without pus is obtained. The wound is loosely sutured to allow escape of serous exudate or pus. A sterile dressing is then applied. Rapid subsidence of the temperature usually follows and the wound heals without secondary infection. If there is no response to penicillin and no evidence of pus formation, the antibiotic is changed, and the effect of the new drug watched. Failure to respond to the new antibiotic in twenty-four hours is an indication for local exploration despite the paucity of local physical signs.

The use of antibiotics has completely altered the outlook in osteomyelitis. The sterilisation of the blood stream and the bony focus concentrates the infection in one place, and sequestra, if treatment has been early, are microscopic rather than macroscopic, and may be absorbed entirely in mild cases without abscess formation. When an abscess is formed its contents are sterile, and simple drainage only is required; the wound is sutured lightly so that serous exudation can continue till the cavity walls are adherent. Continuation of penicillin therapy and a rigid aseptic dressing technique avoid secondary infection; primary healing is

the rule. Antibiotic therapy should continue for twenty-one to twenty-eight days after the onset of the disease.

**Complications of Osteomyelitis.** *Septicæmia* and multiple foci of infection have been mentioned already. Severe toxæmia and the debilitating effects of long-continued sepsis which sometimes lead to amyloid disease have been eliminated by antibiotic therapy. *Purulent Arthritis.*—Infection of the joint may occur: (a) By direct spread (e.g. in the hip in which the metaphysis is partly within the joint capsule); (b) indirectly through soft tissue planes; or (c) as the result of septicæmia. The outcome depends on the severity of the infection, the resistance of the patient and the speedy administration of effective antibiotic therapy. In

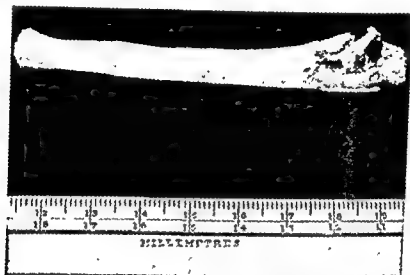


FIG. 544. Massive sequestrum, the whole of the diaphysis of the ulna sequestered in acute infantile osteomyelitis. Note the characteristic pallor of the sequestrum.

infantile osteomyelitis of the upper end of the femur, it is not uncommon for the whole epiphysis of the femur to be lost and the hip to remain permanently subluxated. In other cases, particularly when joint involvement occurs a short time after osteomyelitis is established, it is possible to preserve good joint function, though the damage already done may lead to arthritic change later in life. Treatment consists of immobilisation of the joint, aspiration and the instillation into the joint of a solution of the appropriate antibiotic, repeated daily as long as there are signs of active inflammation, and the maintenance of an effective blood concentration of the antibiotic by systemic administration. Bony involvement of a joint with sequestration and abscess formation demands drainage of the bone as elsewhere. *Sequestration.*—Small sequestra may be absorbed or extruded, but massive sequestra should be removed to hasten healing (Fig. 544). Radiological evidence of separation of the sequestrum, shown by rarefaction around it, should be awaited. The wound can be closed

afterwards. *Cavity Formation*.—Death of bone and the removal of large sequestra may leave a cavity with rigid walls. If the cavity is not obliterated a sinus or recurrent infection occurs. It must therefore be obliterated by one of the methods outlined in the discussion on chronic osteomyelitis. *Pathological Fracture*.—This is now of much rarer occurrence due to the reduction in severity of the disease by the use of antibiotics. It may, however, still occur and demands appropriate splinting and continuation of antibiotic therapy. Healing will occur satisfactorily. *Growth Disturbances*.—These may be due to epiphyseal destruction, or the displacement of an epiphysis. Continued subperiosteal deposit of new bone may thicken a bone considerably. Usually there is shortening, but in a few cases of attenuated infection, stimulation of the epiphysis causes overgrowth of the bone. *Chronic osteomyelitis*, with recurrent acute inflammation, or the persistence of a sinus may follow. This was a common complication before the discovery of penicillin.

**Chronic Osteomyelitis.** (1) Following acute osteomyelitis or the infection of a compound fracture. (2) Chronic localised osteomyelitis (Brodie's abscess). (3) Chronic non-suppurative osteomyelitis (osteomyelitis of Garré). *Chronic osteomyelitis* after acute infection is maintained by persistence of the organisms in bony loculi, which may be microscopic and widespread or macroscopic and localised; or there may be a combination of the two conditions. It is favoured by the occurrence of secondary infection and a mixed bacterial flora, by the presence of sequestra, or the presence of thick-walled cavities which cannot collapse. The condition may present as a long-continued open wound or sinus following acute infection, or as a recurrence of infection, possibly many years after the original acute disease. Sometimes recurrent attacks occur at short intervals accompanied by abscess formation. In a few cases persistent pain of a deep aching character may be the presenting feature, without evidence of pus formation.

The management of chronic osteomyelitis resolves itself into systemic treatment of the infection together with adequate rest, and treatment of the local condition surgically when required. Often the osteomyelitis is widespread, and there is no indication for surgery, but local abscesses must be drained. The conditions which maintain infection and may be relieved by surgery are as follows: (a) Presence of sequestra. These should be removed. (b) Inadequacy of drainage through a long and irregular path. More direct and dependant drainage may clear up the infection, or at least establish a smaller sinus, which, though it may still require regular dressings, reduces the frequency of recrudescence of acute infection. (c) Presence of a cavity with rigid walls. This may lie in bone or be formed entirely by thick fibrous tissue. The cavity must be obliterated and filled with living tissue. This may be done by saucerising the cavity so that surrounding tissues prolapse into it. When this cannot be done without undue weakening of the bone, the cavity may be filled by the partial detachment



of a portion of living muscle, complete with blood supply, and its insertion into the cavity. So effective is antibiotic therapy that in some cases of attenuated infection, such as Brodie's abscess (*vide infra*) and certain cases of chronic osteomyelitis, the cavity can be packed with cancellous bone chips, and these will obliterate the cavity without sequestration. Occasionally it is possible completely to excise an infected bone, such as a



FIG. 545. Brodie's abscess of the upper end of the tibia. This abscess is unusual in having expanded downwards along the medullary cavity.



FIG. 546. Sclerosing osteomyelitis of Garre affecting the shaft of the humerus. There is a diffuse thickening of the humeral shaft, without cavitation or pus formation.

rib or fibula. In very severe long-continued infections of the lower limb amputation may have to be considered.

**Brodie's Abscess.** This consists of a localised abscess in bone seen radiologically as a small cyst, usually in or near the cancellous part of the shafts of long bones, or in cancellous bone (Fig. 545). The X-ray definition of the walls of the cavity is determined by the duration of the disease. In the more recent and active lesions the walls are soft and ill-defined. In the chronic cases, there is sclerosis and a sharply defined wall of compact bone (Fig. 545). The abscesses originally described by Benjamin

Brodie were of tuberculous origin, and although these may still be seen, they are uncommon. In most cases, it is impossible to identify the responsible organism. *Diagnosis.*—Little difficulty arises in most acute cases in which radiological examination shows the presence of the abscess cavity. Difficulty may occasionally arise when the infection is mild and the cavity has not yet appeared. Aching pain, local swelling, and interference with function may arouse suspicion of subacute infective arthritis, rheumatism, or even malingering. The eventual appearance of the cavity in X-rays clinches the diagnosis. The abscess may be discovered by accident in routine radiography. *Treatment.*—This consists of obliteration of the cavity by opening it up and saucerising it, or packing it with bone chips. The contents of the cavity may be purulent, or serous. Culture is usually sterile.

**Chronic Non-suppurative Osteomyelitis (of Garré).** In this disease there is continued subperiosteal deposit of new bone, with resultant thickening of the shaft. Any long bone may be involved. Radiologically, the dense deposit of new bone produces an elliptical enlargement of the bone and prevents any of the interior details of the bone being seen. The density of the radiological shadow is particularly intense (Fig. 546). Patients may become aware of the slow enlargement of the bone and present it as such for diagnosis. More commonly they complain of deep aching pain. This, like all bone pains, is persistent, and worse at night, and in damp weather. It may be so intense as to render the patient's life intolerable. The differential diagnosis includes other causes of subperiosteal new bone formation, e.g. localised Paget's disease and syphilitic periostitis. *Treatment.*—This is always difficult. Massive doses of potassium iodide may relieve the pain in some cases. Short-wave diathermy may relieve or aggravate the condition, but is worth trying. The most certain relief is given by guttering the bone throughout the length involved, so that there is direct access from the remains of the medullary cavity to the overlying soft tissues.

## CHAPTER 80

### TUBERCULOSIS

STREPTOMYCIN and its adjuvants have made a similar revolution in the outlook for tubercular infections as have other antibiotics in osteomyelitis. Recovery from the disease once measured in years is now measured in months. Pasteurisation of milk has altered the relative incidence of infection by human and bovine organisms. Nowadays about 15 per cent. of bone infections are bovine compared with 60 per cent. in the past. Skeletal tuberculosis is always metastatic from a primary tuberculous focus elsewhere, e.g. in the lungs the cervical or the abdominal lymph glands. The secondary lesion may be present with a healed or active primary focus. If active, the primary lesion has also to be treated as indicated in the appropriate chapters. Recrudescence of an old infection always remains a problem; its incidence is increased by the stresses and privations of war. The increasing number of elderly people in the population has contributed to the number of cases of senile skeletal tuberculosis in which the inflammatory reaction is often very sluggish, due partly to acquired resistance.

**Diagnosis.** The history of a primary infection is important, and this may show familial or occupational associations. Skeletal tuberculosis is characteristically slow in onset, with local stiffness, muscular fatigue, and impaired general vigour. Although the disease may be preceded by trauma to the affected bone, the common cause is the showering of the tissues with bacterial emboli due to disruption of a tissue barrier in the primary focus. As the latter is commonly in the lungs they are always X-rayed in search of a pulmonary lesion or enlarged hilar glands. Any sputum is repeatedly examined for tubercle bacilli. Bacteriological proof of the tubercular nature of the disease may be difficult to obtain in some cases. An active chest lesion is not proof that the second lesion is tubercular, though taken in conjunction with local clinical and radiographic signs of tuberculosis, would be strong confirmatory evidence.

The best chances of absolute proof lie in examination and culture of diseased tissue or pus. (1) *Aspiration of a Cold Abscess.*—The risk of secondary infection is avoided by careful aseptic technique and culture through healthy skin. Streptomycin may be instilled into the abscess cavity at the same time, and should be used also to wash out the needle before withdrawal to avoid leaving tubercular debris along the needle track. Tubercular pus has a characteristic thin creamy consistency, and this combined with the absence of heat is almost diagnostic. Systemic antibiotics can, however, sterilise an acute pyogenic abscess so effectively

that in the absence of knowledge of the acute stage confusion is possible. Pus from a *cold abscess* may be sterile. This finding, in the absence of a history of an acute infection treated by antibiotics, is almost as valuable in diagnosis as the discovery of tubercle bacilli or a positive guinea-pig inoculation. (2) *Biopsy*.—This used to be avoided if possible because of the risk of sinus formation or secondary infection. These remain good reasons for avoiding an extensive biopsy, but it may be necessary in a difficult case. Biopsy with a hollow biopsy needle may be used, but is less certain, as infected tissue may not be penetrated. Regional glands are frequently the seat of infection in tubercular infections of the limbs; removal and histological examination of a gland may provide the evidence required.

The *Mantoux test* is sometimes of value. Like other tuberculin tests, a negative result excludes tuberculosis, unless the infection is very advanced; in patients over five years a positive result is of value only when very dilute tuberculin is used. The E.S.R. is raised in tuberculous infections as in many other diseases, but the test is useful in following the course of the disease. The white cell count shows little change, as opposed to more acute infections, unless secondary infection has taken place.

**Tuberculous Osteitis.** The most common site of infection is the spine. Infection of the medullary cavity of the metacarpals or phalanges may occur and is termed *tuberculous dactylitis*. Rarely a local cavity is formed in the bone—a kind of Brodie's abscess. Both pathologically and clinically the progress of the disease may be considered in stages of: (a) Invasion; (b) activity; and (c) healing. In the *invasive stage*, the organisms settle in cancellous bone and form a tubercle, or a series of tubercles. These consist of a central necrotic area, a peripheral zone of giant cells and lymphocytes in an ill-defined granulomatous matrix, and outside this a zone of granulation tissue with a defective blood supply.

In the *stage of activity* these foci spread, becoming confluent and embracing wider portions of cancellous bone, which becomes deprived of its blood supply and so slowly sequesters into the abscess so formed. The abscess tracks along tissue planes to reach the surface. At this stage the radiological appearances are as follows. The bone shows a rounded translucent area, or possibly multiple confluent rounded areas, and in these small sequestra may be seen. In the spine, the sequestra are obscured by the density of overlying tissues, and are often small. The surrounding bone shows no sclerosis, but a radiologically "soft" margin, and there is an absence of any periosteal reaction. There may be a general osteoporosis of the bone in the vicinity of the infection. The extra-osseous abscess is not directly visible, but the distortion of soft tissue shadows gives a clue to its presence, shown in the spine by bulbous enlargement of the paravertebral shadow, or distortion of the line of the psoas. Collapse of bone may occur and obscure the picture. As the disease progresses there is increasing destruction of bone and an increasing amount of fluid

## CHAPTER 80

### TUBERCULOSIS

STREPTOMYCIN and its adjuvants have made a similar revolution in the outlook for tubercular infections as have other antibiotics in osteomyelitis. Recovery from the disease once measured in years is now measured in months. Pasteurisation of milk has altered the relative incidence of infection by human and bovine organisms. Nowadays about 15 per cent. of bone infections are bovine compared with 60 per cent. in the past. Skeletal tuberculosis is always metastatic from a primary tuberculous focus elsewhere, e.g. in the lungs the cervical or the abdominal lymph glands. The secondary lesion may be present with a healed or active primary focus. If active, the primary lesion has also to be treated as indicated in the appropriate chapters. Recrudescence of an old infection always remains a problem; its incidence is increased by the stresses and privations of war. The increasing number of elderly people in the population has contributed to the number of cases of senile skeletal tuberculosis in which the inflammatory reaction is often very sluggish, due partly to acquired resistance.

**Diagnosis.** The history of a primary infection is important, and this may show familial or occupational associations. Skeletal tuberculosis is characteristically slow in onset, with local stiffness, muscular fatigue, and impaired general vigour. Although the disease may be preceded by trauma to the affected bone, the common cause is the showering of the tissues with bacterial emboli due to disruption of a tissue barrier in the primary focus. As the latter is commonly in the lungs they are always X-rayed in search of a pulmonary lesion or enlarged hilar glands. Any sputum is repeatedly examined for tubercle bacilli. Bacteriological proof of the tubercular nature of the disease may be difficult to obtain in some cases. An active chest lesion is not proof that the second lesion is tubercular, though taken in conjunction with local clinical and radiographic signs of tuberculosis, would be strong confirmatory evidence.

The best chances of absolute proof lie in examination and culture of diseased tissue or pus. (1) *Aspiration of a Cold Abscess.*—The risk of secondary infection is avoided by careful aseptic technique and aspirating through healthy skin. Streptomycin may be instilled into the abscess cavity at the same time, and should be used also to wash out the needle before withdrawal to avoid leaving tubercular debris along the needle track. Tubercular pus has a characteristic thin creamy consistency, and this combined with the absence of heat is almost diagnostic. Systemic antibiotics can, however, sterilise an acute pyogenic abscess so effectively

that in the absence of knowledge of the acute stage confusion is possible. Pus from a *cold abscess* may be sterile. This finding, in the absence of a history of an acute infection treated by antibiotics, is almost as valuable in diagnosis as the discovery of tubercle bacilli or a positive guinea-pig inoculation. (2) *Biopsy*.—This used to be avoided if possible because of the risk of sinus formation or secondary infection. These remain good reasons for avoiding an extensive biopsy, but it may be necessary in a difficult case. Biopsy with a hollow biopsy needle may be used, but is less certain, as infected tissue may not be penetrated. Regional glands are frequently the seat of infection in tubercular infections of the limbs; removal and histological examination of a gland may provide the evidence required.

The *Mantoux test* is sometimes of value. Like other tuberculin tests, a negative result excludes tuberculosis, unless the infection is very advanced; in patients over five years a positive result is of value only when very dilute tuberculin is used. The E.S.R. is raised in tuberculous infections as in many other diseases, but the test is useful in following the course of the disease. The white cell count shows little change, as opposed to more acute infections, unless secondary infection has taken place.

**Tuberculous Osteitis.** The most common site of infection is the spine. Infection of the medullary cavity of the metacarpals or phalanges may occur and is termed *tuberculous dactylitis*. Rarely a local cavity is formed in the bone—a kind of Brodie's abscess. Both pathologically and clinically the progress of the disease may be considered in stages of: (a) Invasion; (b) activity; and (c) healing. In the *invasive stage*, the organisms settle in cancellous bone and form a tubercle, or a series of tubercles. These consist of a central necrotic area, a peripheral zone of giant cells and lymphocytes in an ill-defined granulomatous matrix, and outside this a zone of granulation tissue with a defective blood supply.

In the *stage of activity* these foci spread, becoming confluent and embracing wider portions of cancellous bone, which becomes deprived of its blood supply and so slowly sequesters into the abscess so formed. The abscess tracks along tissue planes to reach the surface. At this stage the radiological appearances are as follows. The bone shows a rounded translucent area, or possibly multiple confluent rounded areas, and in these small sequestra may be seen. In the spine, the sequestra are obscured by the density of overlying tissues, and are often small. The surrounding bone shows no sclerosis, but a radiologically "soft" margin, and there is an absence of any periosteal reaction. There may be a general osteoporosis of the bone in the vicinity of the infection. The extra-osseous abscess is not directly visible, but the distortion of soft tissue shadows gives a clue to its presence, shown in the spine by bulbous enlargement of the paravertebral shadow, or distortion of the line of the psoas. Collapse of bone may occur and obscure the picture. As the disease progresses there is increasing destruction of bone and an increasing amount of fluid

in the abscess. It is an interesting feature of the elderly tuberculous patient that the tissue reactions are less acute than in the young, and the disease slower in progress, and abscess formation less common. This is probably due to acquired resistance.

When the disease enters the healing phase there is increasing density of bone, both generally and near to the focus of infection. The abscess cavity begins to diminish in size and may dry up, leaving a mass of fibrous tissue which may eventually calcify. New bone formation begins around the weakened area, in an endeavour to bridge the gap, which, however, may be closed for a long time by fibrous tissue before healing finally occurs. The special consideration of this problem peculiar to the spine is discussed in Chap. 84.

**Tuberculous Arthritis.** The primary site from which a joint is infected is in most instances a focus in bone near the joint. Spread then occurs in the synovial membrane. Synovial infection, cartilage destruction and bone infection are thus present together from an early stage, but vary very much in their relative proportions. It is possible to form a clinical classification of the disease into synovial, cartilaginous and osseous infection. This is, however, of little value in adults in whom the infection is usually destructive and widespread, and in whom the best result to be expected is ankylosis of the joint, but the classification is of value in children who show a greater resistance to the disease than adults. In children, the disease may be limited to the synovium and may heal leaving a mobile joint. If the cartilage is destroyed, perhaps only in part, fibrous ankylosis, leaving perhaps a little movement, may remain. If bone is infected, fibrous or bony ankylosis occurs and *the development of bony ankylosis is the best safeguard against pain, instability and recurrent infection.*

**Diagnosis.** The primary complaint is pain, and secondary to this there is protective muscle spasm fixing the joint. In the early stages, tiredness of the limb may be complained of before the more definite sign of a limp appears. Swelling is slight at first, until there is pus formation, and consists of periarticular infiltration with œdema, rather than an effusion in the joint. In the knee, the thickened synovial membrane may form a roll of tissue above the patella, and at an early stage there may be fluid in the joint. Later the general swelling of the joint with associated vasoconstriction (tumor albus) becomes more obvious as the muscles waste. Deformity depends on the shape of the joint, accentuated by the muscular atrophy around it, and muscle spasm which flexes the joint; it eventually becomes fixed in this position. Abscesses tend to track along tissue planes, and sinuses may form.

Radiologically the initial focus of infection near the joint is not always visible, but zones of rarefaction may be apparent and become better defined later in the disease. There is general decalcification around the joint. The joint space becomes narrow as there is progressive destruction

of cartilage. The joint surfaces become increasingly ill-defined till at last they have a fluffy appearance. Eventually severe bone destruction and collapse of the joint may occur. With healing the bone margins become re-defined. Patchy sclerosis may be present, and there is a return of normal density to juxta-articular bone. If bony ankylosis occurs, trabeculae will be seen crossing the line of the joint. If the ankylosis is fibrous, a softened zone will separate the irregularly defined articular surfaces.

### GENERAL TREATMENT OF TUBERCULOSIS

The object of sanatorium treatment is to place patients under the best conditions for natural resistance to have a full opportunity to act. General rest inevitably necessitates some rest to the diseased region, but more complete rest of the lesion is necessary. This may be obtained in a variety of ways adapted to the site and severity of the lesion, and is discussed in the appropriate chapters. As healing progresses the degree of fixation is slowly relaxed.

**Associated Pulmonary Lesions.** If these develop during the course of treatment of a skeletal lesion, the outlook is seriously affected. It is clear that the patient's resistance even with the assistance of treatment is failing to contain the disease. On the other hand, the discovery of a bone focus in a patient under treatment for pulmonary disease does not affect the outlook as far as survival is concerned, though the patient may lose the function of a joint. The need to allow free access to the chest and to avoid interfering with the chest expansion may modify treatment of bone or joint disease by preventing the application of a heavy plaster, e.g. a thoracobrachial plaster in the treatment of a tuberculous shoulder. The pulmonary lesion also affects the timing of operative treatment. In general it must be healed or healing before operation is undertaken—certainly before a major definitive procedure such as arthrodesis of the knee or hip.

**Specific Drugs.** The discovery of streptomycin has revolutionised the treatment of tuberculosis. This change in outlook would have been short lived, as the tubercle bacillus develops considerable resistance to the drug, had it not been for the discovery of the adjuvant drugs para-aminosalicylic acid (PAS) and isonicotinic acid hydrazide (Isoniazid) which diminish the emergence of resistant strains. It is now clear that in the treatment of tuberculosis, these drugs should be given in combination; streptomycin should never be given alone for tuberculosis. *Dosage.*—For an adult, 1 gm. of streptomycin is given daily by intramuscular injection in two separate doses. With this is given 15 gm. of PAS by mouth divided into five doses, and 150 mg. of Isoniazid in one dose, also by mouth daily. This dosage is continued till 120 gm. of streptomycin have been administered. This ends a course, and a rest must be given if toxic effects are to be avoided. In children, the dosage is proportionately reduced



in the abscess. It is an interesting feature of the elderly tuberculous patient that the tissue reactions are less acute than in the young, and the disease slower in progress, and abscess formation less common. This is probably due to acquired resistance.

When the disease enters the healing phase there is increasing density of bone, both generally and near to the focus of infection. The abscess cavity begins to diminish in size and may dry up, leaving a mass of fibrous tissue which may eventually calcify. New bone formation begins around the weakened area, in an endeavour to bridge the gap, which, however, may be closed for a long time by fibrous tissue before healing finally occurs. The special consideration of this problem peculiar to the spine is discussed in Chap. 84.

**Tuberculous Arthritis.** The primary site from which a joint is infected is in most instances a focus in bone near the joint. Spread then occurs in the synovial membrane. Synovial infection, cartilage destruction and bone infection are thus present together from an early stage, but vary very much in their relative proportions. It is possible to form a clinical classification of the disease into synovial, cartilaginous and osseous infection. This is, however, of little value in adults in whom the infection is usually destructive and widespread, and in whom the best result to be expected is ankylosis of the joint, but the classification is of value in children who show a greater resistance to the disease than adults. In children, the disease may be limited to the synovium and may heal leaving a mobile joint. If the cartilage is destroyed, perhaps only in part, fibrous ankylosis, leaving perhaps a little movement, may remain. If bone is infected, fibrous or bony ankylosis occurs and *the development of bony ankylosis is the best safeguard against pain, instability and recurrent infection.*

**Diagnosis.** The primary complaint is pain, and secondary to this there is protective muscle spasm fixing the joint. In the early stages, tiredness of the limb may be complained of before the more definite sign of a limp appears. Swelling is slight at first, until there is pus formation, and consists of periarticular infiltration with oedema, rather than an effusion in the joint. In the knee, the thickened synovial membrane may form a roll of tissue above the patella, and at an early stage there may be fluid in the joint. Later the general swelling of the joint with associated vasoconstriction (tumor albus) becomes more obvious as the muscles waste. Deformity depends on the shape of the joint, accentuated by the muscular atrophy around it, and muscle spasm which flexes the joint; it eventually becomes fixed in this position. Abscesses tend to track along tissue planes, and sinuses may form.

Radiologically the initial focus of infection near the joint is not always visible, but zones of rarefaction may be apparent and become better defined later in the disease. There is general decalcification around the joint. The joint space becomes narrow as there is progressive destruction

and requires repeated aspiration. Eradication of the diseased bone may be possible.

Tuberculous infection of tendon sheaths is rare, but may still be encountered in the hand—the most common site. The ulnar bursa is the usual sheath involved, and a fluctuant abscess forms extending above and below the transverse carpal ligament (the *compound palmar ganglion*). In this *granulomatous mass* “melon-seed” bodies may develop from condensations of fibrin, and discharge through sinuses in the skin. General and local treatment, including excision of infected granulation tissue, may be required to clear it up.

(under five years  $\frac{1}{2}$  gm. is given daily until a course of 60 gm. is completed, and for a child up to ten years  $\frac{2}{3}$  gm. daily until a course of 90 gm. has been completed ; over this age the adult dosage may be prescribed).

**Operative Treatment.** The choice of the correct moment for operation and its scope are of fundamental importance to success. The use of antibiotics has increased the opportunities for surgery by increasing the patient's resistance and by removing the fear of secondary infection. This, however, does not justify surgery before the patient has had time to build up a general resistance to the disease ; a carefully observed course of treatment with a record of the patient's temperature and E.S.R. forms an essential preliminary, even though it may now be considerably shortened.

The correct line of treatment is governed by the patient's age and resistance, and the condition of the local disease ; it is discussed further in the chapters on joints. A choice is made from the following procedures : (1) *Total Excision of a Focus*.—This is only possible in localised lesions of bone, such as the rare tuberculous Brodie's abscess, or a tubercular osteitis of a rib, or the sternum. (2) *Total Excision of a Joint*.—This is not to be contemplated in children in whom resistance to the disease is high, but may be considered in adults to shorten treatment and as a preliminary to arthrodesis. (3) *Partial Excision of a Focus*.—This may play a part in extensive disease of the spine. In the past, a conservative attitude has prevailed, except when there was spinal cord compression. The beneficial results of decompressing the cord by an anterolateral approach (Chap. 59) on the general progress of the disease has encouraged more active removal of sequestra, granulomatous tissue and drainage of abscesses in the absence of neurological complications. (4) *Arthrodesis*.—In adults, bony fixation of the joint is the only safe end result. This may be accomplished by the disease and its treatment, but to hasten progress supplementary procedures are sometimes used. Care must be taken that the procedure does not fix infected bone surfaces apart. Collapse of cavities is a stage in healing, and the prevention of collapse delays it. Two courses are possible. Either the joint may be opened and the risk of infection flaring up taken, or the joint may be fixed by an extra-articular procedure leaving the focus of infection untouched. Thus in the knee, excision is an almost inevitable prelude to a compression arthrodesis, whereas in the hip the success of extra-articular procedures such as the Brittain operation (Chap. 88) may make it the method of choice. (5) *Synovectomy*.—It is very rarely performed for localised disease. (6) *Amputation*.—This is the last resort in the seriously affected limb with severe secondary infection. It should rarely be necessary nowadays.

**Tuberculous Bursitis and Tenosynovitis.** Embolic infection of a bursa may occur, but more commonly the infection is due to spread from local disease of bone. Infection of the bursa over the great trochanter or of the subacromial bursa may thus occur. An abscess usually forms rapidly

and requires repeated aspiration. . Eradication of the diseased bone may be possible.

Tuberculous infection of tendon sheaths is rare, but may still be encountered in the hand—the most common site. The ulnar bursa is the usual sheath involved, and a fluctuant abscess forms extending above and below the transverse carpal ligament (the *compound palmar ganglion*). In this granulomatous mass “melon-seed” bodies may develop from condensations of fibrin, and discharge through sinuses in the skin. General and local treatment, including excision of infected granulation tissue, may be required to clear it up.

(under five years  $\frac{1}{2}$  gm. is given daily until a course of 60 gm. is completed, and for a child up to ten years  $\frac{3}{4}$  gm. daily until a course of 90 gm. has been completed ; over this age the adult dosage may be prescribed).

**Operative Treatment.** The choice of the correct moment for operation and its scope are of fundamental importance to success. The use of antibiotics has increased the opportunities for surgery by increasing the patient's resistance and by removing the fear of secondary infection. This, however, does not justify surgery before the patient has had time to build up a general resistance to the disease ; a carefully observed course of treatment with a record of the patient's temperature and E.S.R. forms an essential preliminary, even though it may now be considerably shortened.

The correct line of treatment is governed by the patient's age and resistance, and the condition of the local disease ; it is discussed further in the chapters on joints. A choice is made from the following procedures : (1) *Total Excision of a Focus*.—This is only possible in localised lesions of bone, such as the rare tuberculous Brodie's abscess, or a tubercular osteitis of a rib, or the sternum. (2) *Total Excision of a Joint*.—This is not to be contemplated in children in whom resistance to the disease is high, but may be considered in adults to shorten treatment and as a preliminary to arthrodesis. (3) *Partial Excision of a Focus*.—This may play a part in extensive disease of the spine. In the past, a conservative attitude has prevailed, except when there was spinal cord compression. The beneficial results of decompressing the cord by an anterolateral approach (Chap. 59) on the general progress of the disease has encouraged more active removal of sequestra, granulomatous tissue and drainage of abscesses in the absence of neurological complications. (4) *Arthrodesis*.—In adults, bony fixation of the joint is the only safe end result. This may be accomplished by the disease and its treatment, but to hasten progress supplementary procedures are sometimes used. Care must be taken that the procedure does not fix infected bone surfaces apart. Collapse of cavities is a stage in healing, and the prevention of collapse delays it. Two courses are possible. Either the joint may be opened and the risk of infection flaring up taken, or the joint may be fixed by an extra-articular procedure leaving the focus of infection untouched. Thus in the knee, excision is an almost inevitable prelude to a compression arthrodesis, whereas in the hip the success of extra-articular procedures such as the Brittain operation (Chap. 88) may make it the method of choice. (5) *Synovectomy*.—It is very rarely performed for localised disease. (6) *Amputation*.—This is the last resort in the seriously affected limb with severe secondary infection. It should rarely be necessary nowadays.

**Tuberculous Bursitis and Tenosynovitis.** Embolic infection of a bursa may occur, but more commonly the infection is due to spread from local disease of bone. Infection of the bursa over the great trochanter or of the subacromial bursa may thus occur. An abscess usually forms rapidly

greater, as the condition is then purely local, and there is no tendency for other joints to become arthritic. Thus in primary osteo-arthritis, if an affected knee is arthrodesed, the extra strain imposed on the hip or ankle may result in the development of osteo-arthritis of these joints, whereas in secondary osteo-arthritis the chances of the other joints breaking down are much less. The causes of secondary osteo-arthritis include the following: (a) Damage to the joint surfaces. (b) Altered stress and strain on the joint, due to malunion of a fracture, paralysis, occupation, or congenital deformities. (c) The end result of an infective arthritis, or a subacute infective arthritis, or any disease producing recurrent capsular distension and inflammation, such as gout, hæmophilia, villous synovitis. (d) The end result of rheumatic arthritis, after the active disease has ceased.



FIG. 547. Heberden's nodes.

**Primary Osteo-arthritis (Arthrosis Deformans).** This is a disturbance of joint metabolism throughout the body, though its effects may be limited to a few joints only—commonly the larger joints, especially the hip and knee. It is essentially a premature ageing of the joints. It affects both sexes equally, and is most common in old age. The onset is gradual, without any general disturbance or evidence of local inflammation, though the joint may eventually become distorted. People affected are almost always of stocky build, heavy and large jointed. Heredity and occupation play a small role in the ætiology. Heavy outdoor labour predisposes to the disease. Early involvement of the terminal interphalangeal joints of the fingers is the rule; these become enlarged at the shoulders of the phalanges (Fig. 547), producing a readily recognisable deformity (Heberden's nodes). Osteo-arthritis of the metacarpocarpal joint of the thumb also commonly occurs. There is never any

## CHAPTER 81

### CHRONIC ARTHRITIS

IN this distressingly common condition there are two well-defined diseases, namely, osteo-arthritis and rheumatoid arthritis, the contrasting features of which make their diagnosis comparatively simple. There is however, an intermediate group in which the features are mixed; and, furthermore, the two diseases may be present in the same patient. Osteo-arthritis is a "wear and tear" arthritis, and may develop in joints previously the site of acute rheumatism, though the primary infection has ceased to be active. There is also traumatic osteo-arthritis, in which osteo-arthritic changes follow damage to joint surfaces, and osteo-arthritis following uneven distribution of strains placed upon a joint by malalignment of a fractured long bone; these are examples of localised *secondary osteo-arthritis*, but in this chapter discussion is concerned with changes in several joints secondary to general disturbances of bone metabolism, namely, generalised or *primary osteo-arthritis* and *rheumatoid arthritis*.

#### OSTEO-ARTHRITIS (ARTHROSIS DEFORMANS)

**Pathology.** The opportunity to examine an osteo-arthritic joint occurs quite commonly, notably in Keller's operation for hallux rigidus or hallux valgus. The changes are simple and uniform, and characterised by absence of inflammatory reaction in the joint or the ligaments. The joint surfaces show: (1) Patchy erosion of cartilage. (2) Exposure of bone which becomes sclerotic or ivory-like. (3) Areas of erosion of bone communicating with cystic areas under the joint surface. (4) Marginal osteophytes. These consist of outgrowths of mixed bone and cartilage, or cartilage alone. They vary very considerably in size and may block the movement of the joint. (5) The formation of a pannus of granulation tissue, often around the osteophytes and at the margin of the joints; this may appear clinically as thickening of the synovial membrane. The progress of the disease is slow: beginning with softening and erosion of the cartilage, it may progress to complete disorganisation of the joint, though it *never* causes ankylosis, and some movement, however limited, is always preserved. Limitation of movements are always due to capsular contracture, osteophytes, or interlocked degenerated bone surfaces—never to intra-articular adhesions. Loose bodies may form, due to separation of osteophytes or proliferation of cartilage cells. The pathological changes in *primary* and *secondary osteo-arthritis* are the same, but the chances of helping patients with *secondary osteo-arthritis* are much

ulcer, liver disease or hypertension. Treatment begins with 600 mg. daily divided into three doses. This should be reduced at the end of a month to 300 mg. daily. A maintenance dose of 300 mg. may be continued for many months if there are no side-effects. Patients should be warned to stop taking the drug if any unpleasant reactions occur (e.g. gastric disturbances, oedema of the legs or rashes). Complications arise only if



FIG. 548. Advanced osteo-arthritis of the knee joint. There is patchy sclerosis, narrowing of the joint cavity on the medial side and marginal exostosis. There is also considerable subpatellar arthritis.

the drug is continued despite warning signs. Where Butazolidin fails, cortisone in one of its forms, e.g. Prednisone 10 to 25 mg. a day should be tried.

*Relief of Muscle Spasm.* Radiant heat and massage remain the most effective methods, and are often surprisingly useful in joints with marked radiological changes. The use of liniments is chiefly to ensure massage during their application, together with some comforting superficial vasodilatation; they are commonly used in domestic treatment. The trained masseur, however, gets much better results, and combines relaxation of muscle spasm with exercises to maintain the tone of affected muscles. Exercises carried out with the limb suspended in order to relieve joint pressure are valuable in the early treatment of irritable joints. Another and very efficacious method of relieving the joint of limb-weight is the use of the heated pool. This may be combined with massage and exercises. It forms the backbone of spa treatment, though there is nothing to show that the special water used has any particular value. Diathermy, by heating the tissues evenly throughout the limb, produces



fever or other constitutional disturbance and the E.S.R. is within normal limits. The disease may appear after the menopause, especially when this is followed by osteoporosis and increased weight.

Whatever the joint involved, the symptoms are characteristic. At first, the joint is stiff after use. Later it becomes stiff both after use and after rest ; thus getting up in the morning is difficult, but the joint then "works in" and again becomes tired and stiff at the end of the day. Alternatively, the joint stiffens after sitting or resting. There is seldom much swelling of any of the affected joints, except the knee. Muscle wasting slowly occurs, and eventually there is limitation of movement. At intervals there may be phases of increased pain. These may be provoked by minor injury or may come on spontaneously. They may be due to a loose body interfering with the movement of the joint or to periarticular œdema and thickening. Pain is a very variable feature of the disease and may bear no relation to the degree of destruction of the joint, though usually the greater the destruction of the joint the greater is the pain. Sometimes marked destruction causes little pain and sometimes minor destruction may cause severe pain.

The joint has a restricted range of movement. At first pain is felt only at the extremes of movement. As the condition becomes more severe, movements are equally restricted in all directions, and are more painful. Crepitus may be felt, sometimes localised, and sometimes throughout the whole range of joint movement. Effusions are present in acute incidents, but are not common in the ordinary course of the disease. Muscular wasting and contractures with deformity eventually appear. The diagnosis is confirmed by radiological examination (Fig. 548), which shows diminished joint space, or in the later stages irregularity of the joint surfaces and loss of congruity ; patchy sclerosis ; osteophytes, and often marginal erosions near ligamentous insertions ; patchy cystic change in the subchondral bone. There is little general periarticular rarefaction in contrast with rheumatoid arthritis.

**Treatment.** The disease cannot be cured. At the best it can be arrested, and often treatment has to be confined to palliative measures. Diet does not directly influence the course of the disease. Reduction of weight, however, plays a part in the reduction of strain on a joint and should be insisted on in patients who are obese and overweight. Septic foci should be cleared up as part of the measures to improve the general health, but this cannot be expected to exert any special effect on the joints involved.

All measures detailed below play a part in the reduction of pain, but medication may be needed as a supplementary measure. Aspirin in its various forms and combinations is the basic drug, and effective dosage is in the neighbourhood of 2.0 gm. (30 gr.) a day, though it may be more. Butazolidin is also of value though uncertain in its effect, but should be tried if there are no contraindications such as the presence of a gastric

the shoe of the short leg may be of value. Care must always be taken not to over-correct, as this only increases the strains on another set of joints. When joints have adapted themselves to one position it must not be radically changed if pain is to be avoided.

*Surgical Treatment.* Limited treatment may consist of removal of large or painful exostoses, or removal of troublesome loose bodies. In general, limited surgical interference with osteo-arthritic joints is to be avoided and all such procedures should be designed to give minimal interference with joint function for the shortest possible time, so that concentration on active use of the joint can follow shortly afterwards. In the knee joint, where the arthritis may be largely subpatellar and catching



FIG. 549. Arthrodesis of the hip joint by insertion of an iliac graft over the anterolateral portion of the joint. The graft was held in place with screws.

may occur between the rough surfaces, excision of the patella may be worthwhile. In the hip joint it is possible to alter the line of weight bearing through the joint by an osteotomy just above the lesser trochanter (McMurray's osteotomy). The shaft of the femur is displaced medially under the head and union allowed to occur in this position. Metallic fixation of the osteotomy allows earlier use of the limb. This procedure gives great relief from pain, though the arthritis is little changed.

Radical treatment consists of obliteration of the joint and fusion of the bones (arthrodesis) or the formation of a new joint (arthroplasty). The method of arthrodesis is adapted to the particular joint; the hip, the most commonly involved joint, is difficult to fuse (Fig. 549). The procedure may be an intra-articular or an extra-articular one, and

vasodilatation in the involved region. Unfortunately this often increases the pain, probably by increasing the tissue pressure around the joint. It must be used with care and with due warning that it may not be effective and may have to be abandoned. Persistent pain, often worse at night, particularly in the spine, may be relieved by deep X-ray therapy.

*Manipulation.* There have always been those who feel they can achieve by manipulation results which cannot be obtained by other means. Gentle stretching of contracted tissues forms a part of routine treatment along with radiant heat, massage and exercises. Difference of opinion only arises regarding more forcible manipulation, for it is during excessive stretching that harm may occur. However, there are a few cases in which forcible manipulation under general anaesthesia may be of value—notably in arthritic conditions of the foot, such as midtarsal arthritis. Generally manipulations should be gentle and often repeated, rather than single and forcible. Relief of pain and increased mobility can sometimes be obtained in cases resistant to other methods, and thus manipulation has obtained exaggerated importance.

*Intra-articular Injection of Procaine and Hydrocortisone.* Relief from pain was claimed by altering the pH of the joint fluid by injection of lactic acid, but it was soon shown that this exerted no more beneficial effect than injection of 1 per cent. procaine into the joint. Much more satisfactory results are given by the intra-articular injection of hydrocortisone (50 mg. in 2 ml.). These may be combined with manipulation and massage to obtain a greater pain-free range of movement during treatment.

*Alterations in Stresses and Strains on the Joint.* These methods, of which there are many, are adapted to the various joints affected, and depend on variations of three main principles: (1) Restriction of certain joint movements. (2) Fixation of the joint, either partial or complete. (3) Relief of the joint from weight bearing.

A few examples of the application of these principles are given. An acutely painful joint, e.g. after a minor injury, may be rested for a few weeks in plaster. Excessive rest, however, leads to stiffness, and must be avoided if it is wished to retain mobility. A short period of rest for the joint, even rest in bed to avoid weight-bearing, may be of the greatest value as a preliminary to a course of relaxant and mobilising therapy. The use of boots in place of shoes may be of great value in an arthritic ankle by restricting mobility. The light spinal corset may similarly be of value in spinal arthritis. Increasing deformity inevitably imposes strains on sensitive ligaments and therefore has to be controlled. The common abduction deformity at the knee joint may be prevented by a knee cage, though, if severe, this may not be sufficient; then a knee flexion caliper which takes the pressures nearer the foot and the hip may be required. A caliper can be used to relieve the joint (commonly the hip) from weight-bearing and so reduce pain. When arthritic changes have occurred in the spine due to scoliosis from pelvic imbalance, raising

arthritis, and the primary involvement of the joint is periarticular rather than articular. The synovial membrane is, however, swollen and hyperæmic, but there is usually little effusion in the joints, with the exception of the knees, which may be excessively distended. There is hyperplasia of the synovial cells, and increase in the number of synovial villi. The synovial membrane is infiltrated with lymphocytes, plasma cells and fibroblasts, but there are few polymorphonuclear cells. Focal collections of lymphocytes are common. In spite of the absence of poly-



FIG. 551. Rheumatic nodules in the skin over the lower part of the triceps.

morphs in the synovial tissues, they dominate the cells in any effusion. Granulation tissue tends to extend over the margins of the articular cartilage, softening it and destroying it. The bone is rarefied adjacent to it by the hyperæmia around the joint, and eventually the cartilage is lost, exposing bone and granulation tissue. This active surface brought in contact with another similar surface may result in bony fusion of the joint. If the disease is not very active, or if it has died out in a joint, osteo-arthritis supervenes. In all cases, there is widespread involvement of the soft tissues around the joint, including both ligaments and tendons, and these, by contraction and adhesion, may contribute considerably to the deformity. Nodules may appear in the soft tissues elsewhere (cf. the similar rheumatic nodules of rheumatic fever), notably over the ulna, but always related to a subcutaneous bony surface (Fig. 551). They consist of masses of necrotic fibrinoid tissue surrounded by a zone of eosinophil cells, and active granulation tissue. These nodules are painless, and slow to develop or regress.

**Clinical Features.** The disease is more common in women than men, and begins in the active phase of life between twenty and forty years. Heredity may be responsible for a constitutional susceptibility, but not directly for the transmission of the disease. The patient's occupation or cold and damp living conditions may aggravate the disease, but there is no evidence of any causal relationship. The disease usually begins in the smaller joints, although sometimes they may not be involved until late in

fixation may be supplemented by metallic fixation by means of screws or a Smith-Petersen type of pin. The knee lends itself to compression arthrodesis. Arthroplasty is carried out by two main methods, either by excision, e.g. Keller's operation (Chap. 90), or by insertion of a prosthesis such as the metallic cup of the Smith-Petersen arthroplasty of the hip (Chap. 88), or the wider replacement of the head by a prosthesis, as in the Judet operation (Chap. 88). No successful arthroplasty of the knee has yet been designed, but the elbow and the shoulder have been effectively treated by this method. In general, arthroplasties are less efficient than normal joints, and the degree of relief from pain is uncertain. It is for this reason that in osteo-arthritis of the hip, excision of the head and neck of the femur, with removal of part of the acetabulum has been favoured, as it gives a relatively painless joint, though it is less stable than after other operations. A choice between arthroplasty and arthrodesis is made according to the needs of the patient, and particular attention is paid to the patient's age, and the condition of other joints. Arthroplasty is suitable for the light elderly patient, who is not very active and it is necessary also when the disease is bilateral, or when there is osteo-arthritis elsewhere. Arthrodesis is better in young people, as it gives a stronger stable limb, in which further degenerative changes cannot occur.

**Secondary Osteo-arthritis.** The treatment of secondary osteo-arthritis is along similar lines. Because single joints are usually involved and the neighbouring joints are healthy radical surgery is carried out more often.

### RHEUMATOID ARTHRITIS

This is a disease characterised by symmetrical polyarthritis (Fig. 550). The cause of the disease remains unknown, although the discovery of the action of the corticosteroids on the disease has indicated the important role played by the cortex of the adrenal glands in the physiopathology of the condition, but has not revealed the essential cause of the disease.

**Pathology.** The disease is much more acute in onset than osteo-



FIG. 550. The appearance of severe rheumatoid arthritis of the elbow, wrist and finger joints.

arthritis, and the primary involvement of the joint is periarticular rather than articular. The synovial membrane is, however, swollen and hyperæmic, but there is usually little effusion in the joints, with the exception of the knees, which may be excessively distended. There is hyperplasia of the synovial cells, and increase in the number of synovial villi. The synovial membrane is infiltrated with lymphocytes, plasma cells and fibroblasts, but there are few polymorphonuclear cells. Focal collections of lymphocytes are common. In spite of the absence of polymorphs in the synovial tissues, they dominate the cells in any effusion. Granulation tissue tends to extend over the margins of the articular cartilage, softening it and destroying it. The bone is rarefied adjacent to it by the hyperæmia around the joint, and eventually the cartilage is lost, exposing bone and granulation tissue. This active surface brought in contact with another similar surface may result in bony fusion of the joint. If the disease is not very active, or if it has died out in a joint, osteo-arthritis supervenes. In all cases, there is widespread involvement of the soft tissues around the joint, including both ligaments and tendons, and these, by contraction and adhesion, may contribute considerably to the deformity. Nodules may appear in the soft tissues elsewhere (cf. the similar rheumatic nodules of rheumatic fever), notably over the ulna, but always related to a subcutaneous bony surface (Fig. 551). They consist of masses of necrotic fibrinoid tissue surrounded by a zone of eosinophil cells, and active granulation tissue. These nodules are painless, and slow to develop or regress.



FIG. 551. Rheumatic nodules in the skin over the lower part of the triceps.

Clinical Features. The disease is more common in women than men, and begins in the active phase of life between twenty and forty years. Heredity may be responsible for a constitutional susceptibility, but not directly for the transmission of the disease. The patient's occupation or cold and damp living conditions may aggravate the disease, but there is no evidence of any causal relationship. The disease usually begins in the smaller joints, although sometimes they may not be involved until late in

the disease. Characteristically, the metacarpophalangeal joint of the index or middle finger is first involved, and then the proximal interphalangeal joints. Subsequently the larger joints (wrists, shoulders, elbows and knees) may be involved. The pain at first resembles that of osteo-arthritis, being worse after activity, and the joints feel stiff. With the onset of obvious swelling they become more continuously painful and tender. Both hands are usually affected. The terminal interphalangeal joints, in contrast with osteo-arthritis, escape the disease. The swelling involves the periarticular tissues, which are tender; it is regularly fusiform, and made more obvious by the atrophy of surrounding muscles, and is distinctive because of the pale atrophic skin overlying the joint. Except in the knee, there is little fluid in the joints. The onset of the disease is very variable. It may begin gradually or with an acute febrile attack and polyarticular involvement. The E.S.R. is always increased in proportion to the systemic disturbance. The patient is often mildly anæmic, generally pale, and always thin, though usually mentally active and alert. There is no cardiac involvement. Two precipitating endocrine factors have been noted. Thus in pregnancy the increased activity of the adrenals may hold the disease in check until lactation begins, and always produces a remission of established disease; conversely in old age diminished adrenal activity may produce polyarthritides (senile rheumatism). The disease is marked by spontaneous remissions which tend to confuse the assessment of treatment. Generally the disease is steadily progressive, though sometimes no further acute phases appear. There is marked muscular wasting and progressive deformity of the joints. In the hands, the deformity is unmistakable, for there is ulnar deviation of the fingers, often accompanied by subluxations of interphalangeal joints. Flexion deformities of the joints appear and there is increasing disability due to pain and muscular weakness, until the patient is confined to bed as a chronic invalid.

**Radiologically** in the early stages there is a general decalcification of the bones of the hand, thinning of the articular cartilages and consequent narrowing of the joint spaces. Later, there are marginal erosions at the shoulders of the phalanges and punched-out spaces near or just below the joint surface. There are no osteophytic outgrowths in the early stages. The periarticular swelling may be well defined in soft radiographs. With the passage of time, superimposed osteo-arthritic changes appear.

**Treatment.** The general treatment of the disease resembles that for osteo-arthritis, but in the acute phases more intensive therapy with a period of bed rest is required. The systemic manifestations of the disease are treated by measures designed to improve the general health (eradication of septic foci, a full diet with additional iron, general massage and active exercise, and ultra-violet light to improve the appetite and skin tone). *Local Treatment.*—In the acute phase rest is essential. This may be obtained by means of splints or plaster of Paris. When an acute phase has settled down, gentle massage and active exercises are begun. Splints

are discarded, first during physiotherapy only and then during the whole of the day, being retained only at night. It is essential to watch for the beginning of deformities and to prevent their progressing, for late contractures are very difficult to reduce. After cessation of the active episodes of the disease, the management is closely similar to that of osteo-arthritis. Specific remedies are: *Aspirin* in doses of at least 2 gm. (30 gr.) a day and often more. *Butazolidin* 600 mg. a day. *Cortisone* 25 to 100 mg. a day or one of its variants. The response to these drugs is very variable, and they must often all be tried to find the most effective one. None are curative. Cortisone may reduce the E.S.R. and hold the disease in check till a spontaneous remission occurs. It has been aptly said that cortisone does not extinguish the fire of the disease, but merely diminishes the size of the fire which may flare up again on cessation of treatment. Though most effective in active stages of the disease, these drugs are still valuable in relieving pain in the later stages. *Gold Therapy*.—Injection of gold salts has produced many remarkable results, and though there are considerable risks of toxic effects, it should be used when other therapy fails.

**Allied Rheumatic Diseases.** *Still's disease*, which affects children, bears a striking resemblance in its joint manifestations to adult rheumatoid arthritis, but there is always enlargement of the lymph glands and spleen, and there may be some pericardial involvement. Again, aspirin is the most useful drug and the local treatment resembles that of rheumatoid arthritis. *Spondylitis Ankylopoetica* (see also Chap. 84).—This disease is sometimes seen in association with rheumatoid arthritis. It has many points of similarity. Thus there is the occasional acute onset with a raised E.S.R., the loss of weight, the remissions, the eventual fixation of involved joints, the response to cortisone and the similar age of onset; but the localisation of the disease to the spine, its commencement in the sacro-iliac joints and the rare involvement of the hips and shoulders are difficult to explain if the two diseases are to be regarded as being closely related. Furthermore, the response of spondylitis ankylopoetica to deep X-ray therapy is distinctive. *Psoriatic Arthritis*.—Arthritis of the rheumatoid type sometimes occurs in psoriasis, and is remarkable for the rapid recovery which occurs if the psoriasis is cured. It is variable in its onset and severity. *Acute Generalised Osteo-arthritis*. Although it has been emphasised that osteo-arthritis is commonly a disease of gradual onset, occasionally polyarticular osteo-arthritis in middle-aged men affecting the terminal interphalangeal joints and the larger joints is accompanied by a mild general febrile disturbance, and there is a more rapid degeneration of the joints than usual. *Senile Rheumatism*.—This term is applied to the subacute forms of local periarticular swelling and pain occurring in the elderly, particularly around the knees, and in whom there is little evidence of arthritic change. This condition often responds well to Butazolidin.

*Fibrositis*. True fibrositis can be demonstrated in rheumatoid arthritis,



the disease. Characteristically, the metacarpophalangeal joint of the index or middle finger is first involved, and then the proximal interphalangeal joints. Subsequently the larger joints (wrists, shoulders, elbows and knees) may be involved. The pain at first resembles that of osteo-arthritis, being worse after activity, and the joints feel stiff. With the onset of obvious swelling they become more continuously painful and tender. Both hands are usually affected. The terminal interphalangeal joints, in contrast with osteo-arthritis, escape the disease. The swelling involves the periarticular tissues, which are tender; it is regularly fusiform, and made more obvious by the atrophy of surrounding muscles, and is distinctive because of the pale atrophic skin overlying the joint. Except in the knee, there is little fluid in the joints. The onset of the disease is very variable. It may begin gradually or with an acute febrile attack and polyarticular involvement. The E.S.R. is always increased in proportion to the systemic disturbance. The patient is often mildly anaemic, generally pale, and always thin, though usually mentally active and alert. There is no cardiac involvement. Two precipitating endocrine factors have been noted. Thus in pregnancy the increased activity of the adrenals may hold the disease in check until lactation begins, and always produces a remission of established disease; conversely in old age diminished adrenal activity may produce polyarthritis (senile rheumatism). The disease is marked by spontaneous remissions which tend to confuse the assessment of treatment. Generally the disease is steadily progressive, though sometimes no further acute phases appear. There is marked muscular wasting and progressive deformity of the joints. In the hands, the deformity is unmistakable, for there is ulnar deviation of the fingers, often accompanied by subluxations of interphalangeal joints. Flexion deformities of the joints appear and there is increasing disability due to pain and muscular weakness, until the patient is confined to bed as a chronic invalid.

**Radiologically** in the early stages there is a general decalcification of the bones of the hand, thinning of the articular cartilages and consequent narrowing of the joint spaces. Later, there are marginal erosions at the shoulders of the phalanges and punched-out spaces near or just below the joint surface. There are no osteophytic outgrowths in the early stages. The periarticular swelling may be well defined in soft radiographs. With the passage of time, superimposed osteo-arthritic changes appear.

**Treatment.** The general treatment of the disease resembles that for osteo-arthritis, but in the acute phases more intensive therapy with a period of bed rest is required. The systemic manifestations of the disease are treated by measures designed to improve the general health (eradication of septic foci, a full diet with additional iron, general massage and active exercise, and ultra-violet light to improve the appetite and skin tone). *Local Treatment.*—In the acute phase rest is essential. This may be obtained by means of splints or plaster of Paris. When an acute phase has settled down, gentle massage and active exercises are begun. Splints

are those of severe osteo-arthritis, but there is in addition much laxity of the ligaments. It is considered that the degeneration is due to loss of the protective pain sense and the consequent unbalanced muscular activity of the joint. There is interference with the normal processes of repair due to vascular damage so that joints are not greatly improved by splinting and they are very difficult to obliterate by arthrodesis. The diseases which may produce the condition are numerous ; among the commonest are tabes, syringomyelia, and polyneuritis. The arm is relatively infrequently affected. The spine may be involved, and the appearances are then bizarre. Most commonly the large joints of the lower limb are affected. The deformity is progressive and may be marked. If the deformity is great, the arthritis is usually advanced. The striking feature is the complete absence of pain, despite the amount of degeneration of the joint. Marked persistent and recurrent effusions of fluid may occur in the hip and knee. In the ankle, long weight-bearing on the deformed joint may result in open ulceration ; again the joint is painless.

*Treatment.* This depends on the joint involved and the degree of disorganisation. In the upper limb, splinting is all that is usually required. A moulded leather or polythene case is often useful. In the lower limb, a walking caliper may be necessary to relieve the joint from strain. *Arthrodesis is particularly difficult, but may be indicated in the knee or ankle.* Its success depends on the degree of bone sclerosis around the joint ; much sclerosis is inimical to new bone formation. The use of compression in the arthrodesis is valuable.

in gonococcal rheumatism, and in gout. Difficulty arises in establishing its existence apart from such diseases. We know very little about the fluid balance in tissue planes necessary to permit gliding of the scapula or the biceps muscle, or of the physiology of the intramuscular tissue planes. We do know, however, that the fibrositic nodule in muscle described in the past, and given some support for its existence by investigation with electromyography, is produced by stimulation of a nerve root by a prolapsed or degenerate intervertebral disc or osteo-arthritis encoachment on a neural foramen. Thus sciatica was once regarded as referred pain from gluteal fibrositis, but it is now known that the gluteal tenderness and the occasionally palpable nodules are secondary to nerve root irritation. The nodules are small areas of muscle spasm.

There remains a group of wandering muscular pains for which no explanation can be found. These commonly affect the fibro-muscular or fibro-osseous junctions. These are tender to pressure and painful on muscular activity. Common sites of such pain are around the vertebral border of the scapula, and the attachment of the quadratus lumborum to the twelfth rib and the iliac crest.

*Fibrositis of the Trapezius.* Pain around the root of the neck and the shoulder is common. Often it is due to a referred pain from osteo-arthritis in the cervical spine; sometimes it is due to root pressure (disc protrusion or osteophytic occlusion of neural foramina), and there may be a root distribution of pain down the arm. In these conditions there is often a tender thickening in the trapezius where it lies over the supraspinatus, but occasionally there is tender thickening with no evidence of the primary cause. This may be a fibrositis or a fibromyositis, but the evidence for the existence of such a disease is small. Tennis elbow also falls into this obscure category. Here there is pain and tenderness at the fibromuscular attachments of the medial or more commonly the lateral condyle. Lumbago is mentioned elsewhere, as being usually secondary to disc degeneration, with or without root pressure symptoms. Sometimes muscular tenderness, which may be recurrent, occurs in young adults, and it may be relieved by local infiltration with procaine.

Most fibromuscular syndromes show susceptibility to climatic conditions, and a wandering propensity; occasionally they are affected by diet, and have in common an absence of demonstrable cause. They respond rapidly or slowly to heat and massage. They may be precipitated by over-activity or intercurrent infection, and sometimes by emotional stress. As in all conditions in which no satisfactory pathology exists there is much speculation, and ignorance is hidden by the use of such terms as "vagotonic muscular rheumatism," or "stress syndrome."

#### NEUROTROPHIC ARTHRITIS

*Charcot's Joints.* This is a destructive form of chronic arthritis which develops in joints deprived of their sensation. The pathological changes

the characteristic features. Bone pain, aggravated by warmth, may be the first complaint, but attention is more commonly drawn to the condition by the complications, namely, deformity, osteo-arthritis, fracture or sarcoma. The bone is brittle and fractures are therefore transverse. They heal well and in the usual time. Difficulty may sometimes arise in distinguishing between sclerotic Paget's disease of the pelvis and sclerosing secondaries from carcinoma of the prostate. In Paget's disease the alkaline phosphatase is raised during the active phase of the disease, whereas in prostatic carcinoma the acid phosphatase may be raised. The development of sarcoma is an occasional complication. *Localised Paget's disease*, affecting one tibia, or part of the pelvis is more likely to cause confusion in diagnosis. Complete examination of the skull, pelvis and other long bones usually shows a focus of Paget's disease elsewhere. Other diagnoses which may be considered are localised osteitis fibrosa, chronic sclerosing osteomyelitis, syphilitic periostitis, and the periostitis which sometimes accompanies extensive varicose veins.



FIG. 553. Fibrous dysplasia affecting the right clavicle. The cystic changes have the appearance of a tumour, but the outline of the bone has been well preserved in spite of the extent of the disease. Confirmatory changes were found in the skull and other bones.

**Fibrous Dysplasia of Bone.** This condition is bedevilled by a complex terminology, but there are two main types of bone involvement, as in Paget's disease. Firstly, there is a generalised form affecting many bones, sometimes only in one limb or on one side of the body, termed diffuse fibrosis of bone or *polyostotic fibrous dysplasia*, and, secondly, there is the localised form of the disease affecting one bone, and termed local fibrocystic disease, or *monostotic fibrous dysplasia*. The cause is unknown. The blood chemistry is normal. Little change occurs in the condition after growth has ceased. The more severe forms are noted in childhood as the result of deformities or pathological fractures, or because of the occasional association of precocious puberty. Mild cases are often discovered accidentally. The disease is entirely confined to the diaphysis. The bone is thinned and expanded by cyst-like spaces which contain thinned trabeculae, giving removed material a gritty feel (Fig. 553).

## CHAPTER 82

### DISEASES OF BONE METABOLISM

**Paget's Disease (Osteitis Deformans).** Although this disease was first described by Sir James Paget in 1876, its cause still remains unknown. It usually occurs in people over forty years of age, and is often symptomless, until attention is drawn to it by some of its complications. *Pathology.*—Bone is first replaced by connective tissue which then undergoes ossification; cortical bone is absorbed and new subperiosteal bone laid down. This broadens the cortex of the bones, but at the same time the radiological density of the bone is reduced. The trabecular structure of the bones becomes coarsened and emphasised along the lines of weight transmission. The marrow cavity is reduced in size. The skull becomes thickened and in X-rays the tables are blurred, giving a "cotton wool" appearance. Softening of the bones in the stage of connective tissue replacement results in bending and collapse of cancellous bone. Patchy sclerosis may occur, possibly in remissions of the disease, and may be so dense that it causes considerable difficulty in the insertion of wires and screws in fractures of the upper end of the femur. *Diagnosis.*—Enlargement of the skull (Fig. 552), kyphosis, and bowing of the long bones are



FIG. 552. Paget's disease of the skull. Expansion of the two tables of the skull together with patchy sclerosis producing a "cotton wool" effect. (Courtesy of Dr. J. M. Stewart.)

double bend. The costochondral junctions are enlarged, forming the "rickety rosary." The pelvis is compressed and narrowed. The skull tends to become broad and flat, and closure of the fontanelles is delayed. Areas of thinning occur in the skull, especially in the occipital region (*craniotabes*). The dentition is retarded. *Treatment*.—Fresh milk, cod-liver oil, and exposure to sunlight soon arrest the disease. Splinting may be valuable in the early stages while the bones are malleable. Later on, deformities must be corrected by appropriate osteotomies, or wedge resections of bone. Late in life the deformities are liable to produce osteo-arthritis. *Other Forms of Rickets*.—Somewhat similar changes may be produced by other disturbances of calcium absorption. In coeliac rickets the disturbed fat metabolism prevents absorption of vitamins, calcium and phosphorus. In adults, steatorrhœa has the same effect. In renal rickets the disturbed blood chemistry, due to defective renal function, leads to failure of absorption of calcium from the intestine.

*Osteomalacia*. In undernourished communities living on a low vitamin D intake, an increased demand for calcium such as in pregnancy or lactation may produce osteomalacia. The affected mother may give birth to a child with foetal rickets. In time of famine, osteomalacia may make a general appearance irrespective of pregnancy. Bone tenderness and pain in the feet and spine appear, and are added to general kyphosis. Occasionally osteomalacia develops in people, usually women, who are otherwise in good health. They show a disturbed calcium-phosphorus balance and only respond to massive doses of vitamin D. In these cases, transverse bands of rarefaction sometimes appear called "pseudo-fractures."

*Scurvy*. This disease is due to deficiency of vitamin C. Once the scourge of sailors, it is now infrequently encountered in either adults or children. In infants, excessive use of heat in the sterilisation of feeds may produce the condition by destroying the vitamin. The disease is characterised by the occurrence of hæmorrhages. They occur first from the gums and then into the subcutaneous tissues, and subperiosteal hæmorrhages may appear. In infants, there is a disturbance of epiphyseal attachments, and separation of the epiphyses may accompany hæmorrhages around joints (Fig. 554). The hæmorrhages are very painful. In spite of their size and the disturbance of the shape of bones produced by the subperiosteal deposit of new bone, the condition returns to normal with administration of vitamin C, adequate splinting and rest. The radiological appearances without epiphyseal separation are not sharply defined. The metaphyseal surface tends to be dense with a translucent osteoporotic line below it. There is general osteoporosis. The disease is usually seen in the first year of life. It may be confused with other painful conditions around the knee where its effects are most commonly seen (e.g. osteomyelitis, arthritis, hæmorrhage from hæmophilia, traumatic separation of the epiphysis, and syphilitic epiphysitis).

When the disease is localised to part of a bone, it resembles cysts of bone, but when the whole of a bone is involved, the appearances are more distinctive. A search for other sites of involvement including the skull must be made. A common accompanying lesion is increased melanin pigmentation in one or more areas of skin. Pathological fractures may occur and suggest *fragilitas ossium*. The localised form of the disease may be confused with a solitary chondroma, and may only be distinguished by biopsy. The multiple form has to be distinguished from hyperparathyroidism (*vide infra*). In fibrous dysplasia there is no osteoporosis and the blood chemistry is normal.

**Hyperparathyroidism.** The excessive secretion of a parathyroid adenoma causes continuous drainage of calcium from the body. In the skeleton, this is shown by generalised osteoporosis, with collapse of cancellous bone and associated cystic changes. Bone pain is severe, and pathological fractures occur. The disease must be distinguished from other fibrocystic conditions of bone, and from the other causes of osteoporosis: osteomalacia, late or vitamin D resistant rickets, and steatorrhœa. Hyperparathyroidism is further discussed under The Surgery of the Endocrine Glands, Chap. 71.

**Rickets and Osteomalacia.** These are both due to deficiency in vitamin D which is an essential agent in the absorption and utilisation of calcium and phosphorus and therefore in the transformation of osteoid tissue into bone. It is synthesised in the skin by the action of sunlight on ergosterol, and absorbed from animal fats in food. Rickets is due to vitamin D deficiency in infancy and childhood, i.e. when the epiphyses are present and the deformities of growth can occur. Osteomalacia is the term applied to the effects of vitamin D deficiency in adults.

**Rickets.** The disease appears in the second six months of life. The child may be fat, but is pale, flabby and irritable. It is inactive and there is a general loss of muscular tone, and delay in passing the usual milestones of development. Severe deformities are rare nowadays. They are due to lack of treatment and unrestrained activity on the softened bones. If the disease is treated before weight bearing begins, the deformities will be very slight. The normal columnar arrangement of cartilage cells in the epiphyses becomes completely disorganised and irregular. The zone of provisional calcification becomes wide and ill-defined, and the trabecular bone formation loses all systematisation. Most important in diagnosis are the radiological changes. The epiphyses are enlarged and the ends of the bones taper towards the shaft. The epiphyseal plates are widened and they are irregular in outline. They also tend to be cup-shaped with the convexity towards the metaphyses. The chief effects of the softening are found on the weight-bearing bones. Thus the femora are bowed and may be buttressed by hypertrophy of the linea aspera, and coxa vara develops. The tibiae tend to have a

## CHAPTER 83

### TUMOURS OF BONES AND JOINTS

#### TUMOURS OF BONE

ALL primary tumours of bone have a mesoblastic origin. As the cells are capable of differentiation into fibrous tissue, cartilage and bone, it is not surprising that these tissues are not always sharply defined, and that tumours of bone are often mixed. Thus the common osteochondroma arises from a displaced nucleus of an epiphyseal plate, and during the growing period is covered with a cartilaginous cap, although in the adult it is a pure osteoma. In spite of the mixture of cells likely to be encountered, it is convenient to classify tumours according to the predominating tissue.

	<i>Bone</i>	<i>Cartilage</i>	<i>Fibrous Tissue</i>
Benign .	Osteoma. Multiple Exostoses. (Diaphyseal Aclasia.)	Chondroma. Enchondroma { Single. Multiple.	Fibroma.
Malignant .	Osteogenic Sarcoma.	Chondrosarcoma.	Fibrosarcoma.
	<i>Blood Vessels</i>	<i>Blood Cells</i>	<i>Unknown Histogenesis</i>
Benign .	Angioma.	—	Giant-celled tumour.
Malignant .	Angiosarcoma.	Myeloma. Leukæmia. Hodgkin's disease.	Ewing's tumour.

In addition to primary tumours of bone, there are secondary tumours arising from primary growths elsewhere, notably, the breast, prostate, kidney, thyroid, and lung.

#### BENIGN TUMOURS OF BONE

**Osteomas, Osteochondromas and Chondromas.** True *osteomas* are rare tumours of dense lamellar bone. They are sessile, clinging like a limpet to the bones of the skull or one of the skull sinuses (Chap. 58). They are slow growing and often symptomless. *Osteochondromas* are the most common bony tumours encountered. They arise from displaced rests of the epiphyseal plate and are therefore found around the ends of long bones—particularly around the knee. They begin as *enchondromas* and then, forming bone as they grow, become osteochondromas and finally as growth ceases they become osteomas. They are commonly solitary (Fig. 555); sometimes they occur bilaterally, or there may be more than one around a joint.



**Other Disturbances of Bone Metabolism.** *Hyperthyroidism* may cause some general decalcification of bone, but only to a slight extent, and never likely to be confused with that due to hyperparathyroidism. *Hyperpituitarism*.—The eosinophilic anterior lobe cells govern growth, and excessive secretion in an eosinophil adenoma in childhood causes *gigantism* (general overgrowth of the skeleton) and in the adult

*acromegaly* (increased thickness of the bones). This subject is further discussed under *Intracranial Tumours*, Chap. 53). *The Sex Hormones*.—Deficiency does not retard growth, but it does retard skeletal maturation, so that the epiphyses may still be found unclosed over the age of thirty. Deficiency in the adult is one of the factors in senile osteoporosis, and is more obvious in women; post-menopausal osteoporosis is frequently accompanied by obesity and attention is often drawn to the condition by a kyphosis. In spite of undoubted relationship to declining gonadal activity, the administration of the hormones is very uncertain in effect. Testosterone propionate, 50 mg. every other day, may be supplemented with cestradiol 2 mg. every fourth day. If this is ineffective, methyltestosterone may be tried, 15 mg. daily. Adequate vitamin D intake should be assured and a calcium salt may also be given.



FIG. 554. The radiographic appearances of a case of scurvy. There is separation of the lower femoral epiphysis and marked new bone formation under the distended periosteum. In spite of these deformities, a remarkable restoration of the normal appearances of the femoral shaft occurred.

**The Adrenal Cortex.** Excess of adrenal androgens produces early sexual and skeletal maturity, but with eventually diminished height. There is excessive urinary excretion of the 17 ketosteroids. The adrenal corticoids inhibit osteogenesis, and are thus opposed in action to the adrenal androgens. In Addison's disease, in which the secretion of both is impaired, no bone changes occur. Excess of corticoids produces Cushing's syndrome (Chap. 72) in which there is osteoporosis, sometimes leading to collapse of vertebrae.

sharply defined in their cellular characteristics, and may tend to recur locally, or merge slowly into the chondrosarcomas. Recurrence following complete local removal demands radical treatment, i.e. amputation.

**Fibromas.** These are very rare. They may arise from the periosteum or occasionally from the intramedullary connective tissue.

### MALIGNANT TUMOURS OF BONE

Bearing in mind the unsatisfactory shading off into malignancy of some of the benign tumours, we can recognise four main groups of malignant bone tumours: (1) Osteogenic sarcoma. (2) Chondrosarcoma. (3) Fibrosarcoma (spindle-celled sarcoma). (4) Ewing's sarcoma.

**Osteogenic Sarcoma.** This is the commonest variety of malignant bone tumour, and is divided into osteolytic (Fig. 557) and sclerosing types. This differentiation, based on radiological appearances, is of little practical value, for both tumours are very malignant.

most often in the second and third decades of life; it may occur late in life in Paget's disease of bone. The tumour occurs at sites of greatest growth activity, notably the ends of the long bones, and particularly those around the knee. *Symptoms and Signs.*—Pain is the first feature; it becomes severe and is often worse at night. The rate of progress of the pain is related to the rate of growth of the tumour. A diffuse tender swelling attached to the bone then appears. Owing to the vascularity and rapid growth of the tumour it may not be hard. Increased vascularity of the skin over the tumour is common, and the tumour itself may pulsate or have a bruit. In the osteolytic varieties pathological fractures may occur. Rapid metastasis to the lungs is the rule, and death may occur within a few months. In the less malignant tumours, in spite of amputation, death commonly occurs within three years.

*Diagnosis.* The radiological appearances are very variable, and alone do not always allow a diagnosis to be made. They show a variable balance between new bone formation and bone destruction. In sclerosing tumours (20 per cent.), dense subcortical bone forms in the metaphysis.



FIG. 556. Solitary chondroma of a proximal phalanx showing slight calcification. (X-ray by Dr. J. M. Stewart.)

The cartilaginous cap becomes surrounded by a bursa to facilitate the play of tendons and muscles over and around the tumour, and because of the moulding pressures of these structures the osteoma is always directed away from the joint (Fig. 555). Osteomas are composed of cancellous bone; the trabeculae are continuous with those of the cancellous end of the parent bone. They are easily removed, for the bursa provides a ready plane of access when opened; they are cut off with an osteotome.



FIG. 555. A solitary exostosis on the postero-medial aspect of the tibia.

The indications for operation are deformity, interference with joint or muscle function, a snapping tendon which flicks over the exostosis on movement of the joint, or pressure on a nerve or blood vessel. Other osteomas, produced by chronic irritation, are the marginal exostoses of osteo-arthritis, the subungual osteoma, and the traumatic ossification in attachments of tendons, notably the calcaneal spur and "rider's bone" in the adductors.

Multiple osteochondromas are rare. They are a characteristic feature of Ollier's disease (dyschondroplasia) in which the hands are chiefly affected, but also other bones. Multiple osteochondromas occur more often in the hands and feet apart from Ollier's disease, and remain

localised to these regions. More commonly encountered than either of these two conditions is the solitary enchondroma of the phalanges of the hands or feet. Distinct from these again are the enchondromas of the ends of long bones, usually seen around the knee; they also are rare.

Enchondromas of the hands and feet are seen in the adolescent and young adult. Attention may be drawn to them by deep aching pain, particularly after use of the part; by deformity; or by pathological fracture. The tumour, consisting of well-differentiated cartilage, may contain areas of degeneration and calcification. Bone is absorbed, leaving a cystic space (Fig. 556). There is only slight periosteal new bone formation around it; hence there is gradual weakening of the bone leading to fracture. Treatment consists of curettage of the contents of the cavity which is then packed with cancellous bone chips. This type of enchondroma does not recur. Enchondromas in other regions may not be so

sharply defined in their cellular characteristics, and may tend to recur locally, or merge slowly into the chondrosarcomas. Recurrence following complete local removal demands radical treatment, i.e. amputation.

**Fibromas.** These are very rare. They may arise from the periosteum or occasionally from the intramedullary connective tissue.

### MALIGNANT TUMOURS OF BONE

Bearing in mind the unsatisfactory shading off into malignancy of some of the benign tumours, we can recognise four main groups of malignant bone tumours: (1) Osteogenic sarcoma. (2) Chondrosarcoma. (3) Fibrosarcoma (spindle-celled sarcoma). (4) Ewing's sarcoma.

**Osteogenic Sarcoma.** This is the commonest variety of malignant bone tumour, and is divided into osteolytic (Fig. 557) and sclerosing types. This differentiation, based on radiological appearances, is of little practical value, for both tumours are very malignant. Osteogenic sarcoma is found most often in the second and third decades of life; it may occur late in life in Paget's disease of bone. The tumour occurs at sites of greatest growth activity, notably the ends of the long bones, and particularly those around the knee. *Symptoms and Signs.*—Pain is the first feature; it becomes severe and is often worse at night. The rate of progress of the pain is related to the rate of growth of the tumour. A diffuse tender swelling attached to the bone then appears. Owing to the vascularity and rapid growth of the tumour it may not be hard. Increased vascularity of the skin over the tumour is common, and the tumour itself may pulsate or have a bruit. In the osteolytic varieties pathological fractures may occur. Rapid metastasis to the lungs is the rule, and death may occur within a few months. In the less malignant tumours, in spite of amputation, death commonly occurs within three years.

*Diagnosis.* The radiological appearances are very variable, and alone do not always allow a diagnosis to be made. They show a variable balance between new bone formation and bone destruction. In sclerosing tumours (20 per cent.), dense subcortical bone forms in the metaphysis.

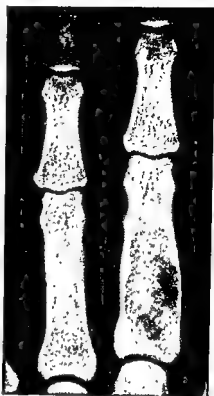


FIG. 556. Solitary chondroma of a proximal phalanx showing slight calcification. (X-ray by Dr. J. M. Stewart.)

Later subperiosteal new bone formation occurs, firstly at the margins of the elevated periosteum (Codman's reactive triangle, cf. new bone formation at the same site in osteomyelitis), and then spreading through the tissue over the tumour in radiating spicules (sun-ray appearance—



FIG. 557. Osteolytic sarcoma of the femur involving the knee joint.

Fig. 558). In osteolytic tumours, there is an area of subcortical bone erosion; this steadily enlarges, without much reactive change in the surrounding bone, though bony remnants may be seen crossing the cavities. This appearance may be produced by erosions from a secondary tumour, or even in osteomyelitis, or a myeloma. The diagnosis is not always easy and the following conditions have to be taken into consideration: (1) Syphilitic osteitis and periostitis. (2) Tuberculosis osteitis. (3) Fatigue fractures, e.g. March fracture. (4) Giant cell tumours. (5) Metastatic tumours with a suppressed primary. (6) Sclerosing osteomyelitis. There is no evidence, despite the vascular nature of the tumour, that biopsy increases the risk of metastasis. This investigation should therefore be performed in all doubtful cases, to determine the nature and degree of malignancy of a tumour, and as a guide to prognosis and treatment, although the microscopic diagnosis of malignancy may sometimes be unreliable.

*Prognosis.* The more anaplastic the tumour the worse is the prognosis. The younger the patient, the worse is the outlook. Tumours of the limbs, being more accessible to radical surgery, have a better prognosis than

tumours of the trunk. The five-year survival rate only amounts to one in ten cases.

**Treatment.** In spite of the grim mortality of the disease, the treatment is amputation for osteogenic sarcomas of the limbs and radical removal of such tumours occurring elsewhere. There is no advantage in amputating high above a tumour; section through clean uninvolved tissue some inches above is all that is needed. No advantage is obtained from removal of associated lymphatic glands. Amputation avoids the pain and discomfort of a rapidly growing tumour and the unpleasantness of possible fungation. It also keeps hope alive, and is better psychologically than the acceptance of the almost inevitable defeat without a fight. Success depends on complete removal of the tumour before metastasis has occurred. It is often impossible to know whether the tumour has metastasised in early cases suitable for operation; therefore the slender chance that it has not must be taken. Radiography of the lungs, the commonest site for secondary deposits is essential. The alternative treatment is radiotherapy. This is indicated for inoperable tumours. In some tumours, notably the spindle-celled sarcomas, preliminary radiotherapy, because of its immediate check to the growth, gives longer time for assessment of a case, and so may enable amputation to be avoided when secondaries are developing.



FIG. 558. Osteogenic sarcoma of the humerus showing the sun-ray spiculation and extensive calcification which may occur.

**Chondrosarcoma.** This is one of the least malignant of bone tumours. It tends to develop in a pre-existing chondroma, and consequently is found at a later age than osteosarcoma (thirty to fifty years). It is slow in growth, and the clinical features, though similar to those of osteogenic sarcoma, are more gradual in appearance. The tumour may reach a large size. It consists of bluish well-differentiated cartilage in which areas of degeneration are common. The medullary type of tumour is cystic in appearance, and radiologically difficult to distinguish from a giant-celled tumour. Erosion of the cortex and extension into the soft tissues occurs. In the more peripheral tumours, the bony enlargement is palpable in the

early stages. This tumour is not radiosensitive, and in the limbs amputation is the only possible treatment. Difficulty may arise in determining the degree of malignancy in certain cases, due to an unrepresentative biopsy or indefinite cellular structure. Some of these tumours are locally recurrent, but show no tendency to metastasis.

**Fibrosarcoma.** This is a rare tumour of adult life. The periosteal type produces a large mass outside the bone, whereas the endosteal type, because of erosion of bone without new bone formation, may produce a cystic cavity resembling that of a secondary carcinoma or osteogenic sarcoma of the osteolytic type. The tumour is slow to metastasise to the lungs. Microscopically it may have a well-differentiated cellular structure, and a small localised tumour may therefore be treated by local excision. A large tumour or a recurrence of a small one calls for amputation when a limb is involved. Some of these tumours have a spindle-cell structure, and are then radiosensitive.

**Ewing's Sarcoma.** This is a rare tumour usually occurring between the ages of ten and twenty years. Its histogenesis is hotly disputed. It produces a general disturbance with fever. There is subperiosteal new bone formation occurring characteristically in layers and likened to an "onion skin." Thus it may be confused with low-grade osteomyelitis, and radical treatment mistakenly postponed. A secondary deposit or an osteogenic sarcoma may also be difficult to differentiate from an Ewing's tumour. The tumour is radiosensitive, and this fact may be used diagnostically though biopsy is wiser. Progressive spread of the tumour is inevitable. The usual treatment is radiotherapy followed by amputation where possible, though the disease is almost inevitably fatal.

**Giant-cell Tumour (Osteoclastoma).** The histogenesis of this tumour is still uncertain. Paget thought it arose from bone marrow, whereas others have thought that it arose from the fibroblasts and that the giant cells are similar to the osteoclasts of normal bone. This view is now being attacked. There is a close similarity between the histology of the giant-cell tumour, the lining of the solitary bone cyst, and generalised fibrocystic disease of bone; although this does not necessarily mean that they are related, such a relationship is an attractive hypothesis. Giant-celled tumours are most common around the knee joint, though they may be found in the epiphyseal region of long bones elsewhere. They are more common in women, and the maximum incidence is between twenty and thirty-five years of age. Pain followed by steady expansion of the bone is the usual presentation, though a pathological fracture may be the first manifestation. The radiological appearances are usually characteristic. A large cavity is visible in the epiphysis. The cortical bone is thinned and expanded, but sharply defined, and there is no new bone formation, unless a pathological fracture has occurred. The cavity is often crossed by coarse trabeculae, giving the appearance of a conglomeration of cysts. The thin subcutaneous cyst walls sometimes crumble, giving the tradi-

tional sign of "egg-shell crackling" on pressure. Macroscopically, the tumour varies greatly in colour according to its vascularity, from yellow through grey to deep red. Microscopically, there is a spindle-cell ground-work in which there is an even distribution of blood vessels and giant cells. Tumours vary in their rate of growth; the more active ones have a very cellular stroma and smaller giant cells. *Treatment.*—The original treatment for this tumour when occurring in the limbs was amputation, but this drastic procedure is now reserved for tumours which have gravely disorganised a joint such as the knee, or for the recurrent and occasional malignant types. When the tumour is accessible and can be removed locally this should be done. The cavity is packed with bone chips, after the walls of the tumour have been removed. Total resection of the affected portion of bone, e.g. the upper end of the fibula is sometimes possible. Because the tumour is radiosensitive, its treatment by deep X-ray therapy is gaining adherents. It may be used preoperatively or as definitive treatment.

**Tumours of the Blood Cells. Multiple Myelomatosis.**—Tumours arise in the bone marrow and the dominant cell is the plasma cell, with an eccentric nucleus and a spoke-like arrangement of chromatin. Occasionally the tumours are composed of myelocytes or erythroblasts (disputed). The tumours are soft, almost fluid and vary in colour with their vascularity. A tumour may appear in one site and remain localised there for a variable time, e.g. one to five years, and may then be called a solitary *plasmocytoma*. Eventually, however, the disease becomes widespread with the clear-cut picture of multiple myelomatosis. The disease is one of adult life occurring between forty and seventy years. The tumours show a predilection for the cancellous bones of the trunk. The limbs are involved in the late stages of the disease. The tumours are small and widely scattered through the ribs, skull, clavicles, pelvis and vertebrae. They can only be palpated in the later stages of the disease, and in half the cases no tumour is ever felt. Bone pain, i.e. deep aching pain, worse with use and at night, is the usual first manifestation of the disease, but, because of its frequent involvement of the vertebral bodies, the presenting symptom may be backache. As the vertebra is destroyed a kyphosis may appear, or the sudden collapse of a vertebra may produce an acute incident, sometimes accompanied by paraplegia.

The radiological features of the disease are diagnostic. Soft punched-out areas, 1 or 2 cm. in diameter, are scattered widely in cancellous bone. There is no bone reaction. Solitary plasmocytomas are more difficult to diagnose as they may show trabeculation resembling a giant-cell tumour, or absorption suggestive of a metastatic tumour.

Confirmation of the diagnosis is best obtained by sternal puncture which usually shows an increase in plasma cells in the marrow. Demonstration of Bence-Jones' protein in the urine is unreliable and it does not occur early in the disease when there is little tumour tissue; it may also



be intermittent. In many cases it is never found at all. Pseudoglobulins, usually in the position of true gamma globulins, can be demonstrated by electrophoresis in 90 per cent. of cases. In doubtful cases biopsy may be needed. *Differential Diagnosis.*—Widespread carcinomatosis may mimic the lesions radiologically, but a primary tumour can usually be found. Hyperparathyroidism may be suspected, but it has its own characteristics

(Chap. 71). *Prognosis.*—

Although the tumours are radio-sensitive, and radiotherapy gives striking relief of pain, particularly in the vertebral tumours, the disease is invariably fatal, and if the condition is widespread to begin with, death occurs within about three years. Longer periods of survival have been reported with solitary plasmocytomas. Pathological fractures may be difficult to treat, but radiotherapy often relieves the pain, though union is by no means certain.

**Leukaemia and Hodgkin's Disease.** Invasion of bone occurs in the later stages of both diseases. Patchy rarefaction and small punched-out areas appear in the bones, sometimes resembling secondary tumours.

### Secondary Tumours of Bone.

Because of the very vascular nature of bone marrow these tumours are common. The characteristic of most secondary deposits is the soft erosion of bone without reactive changes (Fig. 559). This process may go on to pathological fracture, and then some reaction may be seen in an attempt to repair the injury. Cancellous bone, e.g. the ribs, skull, vertebrae, pelvis and ends of the long bones, is usually invaded.

Carcinomas of the thyroid, kidney, breast and prostate have a special propensity to metastasise in bone, but carcinomas of the breast and prostate being so common account for most cases. Tumours of the bronchi, female genitalia, and gastro-intestinal tract account for a small number of secondaries in bone, but almost any malignant tumour, whatever its origin, may metastasise in bone.

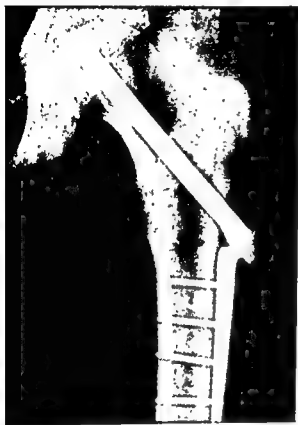


FIG. 559. An osteolytic secondary deposit in the upper end of the femur. Pathological fracture was prevented by insertion of a MacLaughlin nail and plate.

Attention is drawn to a secondary deposit by the occurrence of deep aching pain in the bone, or the appearance of a deformity due to bone collapse, pathological fracture, or soft tissue invasion. Difficulty in diagnosis may arise in the following conditions: (1) *Concealed Primary Tumour*.—After a complete physical and radiological examination for a primary growth, a biopsy of a secondary tumour may be necessary to establish the diagnosis. Even this may not give positive information as to the primary site of the growth, but it usually establishes its innocent or malignant nature. (2) *An Osteopetrotic Deposit*.—Increased density of bone is a characteristic feature of secondary deposits from carcinomas of the prostate, and it occurs occasionally with secondaries from primary carcinomas elsewhere. The condition may be mistaken for Paget's disease, chronic non-suppurative osteomyelitis or sarcoma. Prostatic metastasis is usually confined to the pelvis and lumbar vertebrae; the primary tumour is frequently demonstrable, and the blood acid-phosphatase level is raised. (3) *Collapse of a Vertebral Body*.—Collapse of a single vertebra may obscure the radiological features of the tumour. If a primary tumour cannot be found, it might then be confused with senile osteoporosis, multiple myelomatosis or Cushing's disease. These possibilities must be investigated, and if no conclusion can be arrived at a biopsy of the lesion may be justified. (4) Multiple deposits manifesting themselves by anaemia and an osteoporosis. Examination of the marrow by sternal puncture may show malignant cells.

*Treatment.* The growth of secondary deposits from carcinoma of the prostate is retarded and pain relieved by endocrine therapy (Chap. 29), and temporary regression of secondary deposits from some carcinomas of the breast is achieved by oophorectomy before the menopause and by hypophysectomy or adrenalectomy after the menopause (see also Chap. 73). Deep X-ray therapy may be tried for other types of secondary deposits.

### CYSTIC CONDITIONS OF BONE

There is no sharp line of distinction between a rarefied area in a bone and a cyst radiologically. The radiological appearances of a cyst\* are due to the slow growth of the tumour or disease allowing time for a wall to form, and thus sharply demarcating the cystic area from surrounding bone. The more slowly the disease advances the more sharply defined is the cystic appearance. Cysts may be single or multiple.

*Solitary Cysts. Bone Cyst of Adolescence.*—This is a well-defined cyst occurring in the metaphysis (Fig. 560). It may be a soft chondroma, or it may contain fluid and be lined with a thin fibrous tissue coat containing giant cells. *Chondroma.*—This is most commonly found in the hand, but it may also occur in the metaphysis of long bones (enchondroma). The

\* A cyst, strictly speaking, means any sac containing liquid. Here it is used loosely to describe the sharply defined areas of rarefaction seen radiologically.

bone is expanded. Old chondromas have patchy calcification throughout the stroma. *Solitary Plasmocytoma*.—Myelomatosis develops later. *Brodie's Abscess*.—This is a localised bone infection. In the late stages there is a well-defined sclerotic wall. There is a periosteal reaction if the cyst is near the surface. *Giant-cell Tumour*.—This cyst is multilocular with coarse septa and occurs near the ends of long bones, particularly near the knee. The bone expands, but there is no periosteal reaction. *Gumma*.—This lesion is usually multiple and the walls are ill-defined.

*Tuberculosis*.—Infection of the bone with the tubercle bacillus may produce a variety of Brodie's abscess which may slowly expand bone as in tuberculous dactylitis. *Carpal Cysts*.—Small cysts scattered in the carpal bones may be found on routine radiography. They are harmless, and their significance is unknown. Many varieties of cyst are found in association with the teeth and jaws (Chap. 65).

**Multiple Cysts.** *Multiple Chondromas*.—(a) Affecting the hands alone (rare); (b) associated with dyschondroplasia (Ollier's disease); (c) associated with diaphysial aclasis. *Myelomatosis*.—Usually punched-out but ill-defined walls. *Osteitis Fibrosa Cystica*.—(a) Generalised (*hyperparathyroidism*). Other bones are involved, often including the base of skull. (b) Localised. Many cysts in the bone. Very slow development. Bone shape preserved in spite of extensive change. *Osteo-arthritic Cysts*.—Small cysts are frequently found in the subcartilaginous bone in severe osteo-arthritis.

### TUMOURS OF JOINTS

Tumours of joints are rare. The thickened synovial membrane associated with tuberculous disease and once called "tumor albus" is not a joint tumour in the pathological sense. Joints may become secondarily involved in tumours of bone in their vicinity, but primary tumours are limited to those arising from the synovial membrane. (1) *Synovioma*.—This is most commonly encountered in the hands arising from a tendon sheath. It may arise from joints, and show a variable degree of malignancy. In the more malignant forms there are spindle cells with active nuclei. Foam cells, cartilage cells, and giant cells may be found in both



Fig. 580. An "adolescent" cyst of the femoral neck.

innocent and malignant forms. (2) *Synovial Cysts*.—(a) Ganglia, single and multiple ; (b) cysts of the semilunar cartilages of the knee (Chap. 89); (c) degenerative cysts associated with marked osteo-arthritis. (3) *Synovial Polyposis*.—The formation of multiple villi is accompanied by a persistent or recurrent effusion into the joint. The diagnosis may be difficult without biopsy. (4) *Synovial Chondromatosis*.—In this condition the villi form cartilage and the nodules of cartilage break off into the joint continuing to enlarge in the joint fluid, so that hundreds of rounded loose bodies may be found.

## CHAPTER 84

### THE SPINE

**Clinical Examination.** The complicated structure of the spine and its depth from the surface make clinical examination difficult and radiography all the more important. However, there is much to be learnt both directly and by inference on clinical examination. The three basic manifestations of spinal disorder to be studied are : (1) Deformity, fixed or mobile ; (2) muscle spasm ; and (3) pain, whether local, referred or due to pressure on nerve roots or the spinal cord.

The examination should begin with the patient standing, and the first observation to make is whether the pelvis is level or not, for a spinal curvature may be secondary to alteration in the pelvic level due to a short

leg. The pelvis should be made level by blocks placed beneath the shortened leg (Fig. 561). With the pelvis level, the posture of the spine should be inspected again. Note how patients hold their head, the presence or absence of round shoulders, flattening of the normal lumbar lordosis, and the general bearing—such a valuable clue to muscular tone, physical fitness, and mental make-up.

The combination of rigidity, flexibility and shock absorbing qualities which characterise the normal spine is dependent to a large extent on the intervertebral discs and also on the arrangement of the component sections of the spinal column. The factors adding mobility to strength are the junction of alternate mobile and fixed sections of the spine, and the presence of alternating curves. The mobile cervical spine is curved forwards and rests on the comparatively fixed thoracic spine which is bowed backwards. This in turn rests on the forward curving lumbar spine, which makes a sharp angular junction, the



FIG. 561. The first step in examining the spine in a standing patient is to level the pelvis by balancing any inequality in length of the legs.

lumbosacral joint, with the sacrum. Because of its shape and position, this joint is particularly liable to stresses and strains, and this may account in large measure for its frequent degeneration.

Any increase in the forward bowing of the lumbar spine (*lordosis*) throws the shoulders back to balance the body. *Lordosis* is the inevitable accompaniment of increased backward bowing of the thoracic spine (*kyphosis*). A *kyphosis* may also occur in the lumbar or cervical spine, where greater changes are, however, necessary to reverse the normal curves. Deflection of the spine to the side, i.e. a lateral bend, is known as *scoliosis*. In the thoracic region, it is impossible to have a lateral bend of the spine without rotation, owing to the resistance offered by the ribs. Therefore a thoracic *scoliosis* is always accompanied by a rib deformity; the ribs crowded on the inner side of the curve tend to swing forward, whereas those opened up on the outer side of the curve swing backward and become prominent. Spinal deformity is either widespread or localised. If widespread, it is made up of multiple small deformities of many vertebrae, producing a long curvature, e.g. as in senile osteoporosis, adolescent *kyphosis*, or paralytic deformities. A localised or angular deformity (*gibbus*) is due to local disease with collapse or deformity of one or two vertebrae, e.g. as seen in tuberculosis of the spine (Fig. 562), congenital deformities, or crush fractures of vertebrae. A deformity may alter with changes of posture; thus in *scoliosis* due to muscle spasm from a disc lesion, the deformity may increase as the patient bends forward, or an apparently straight spine may deviate to one side.



FIG. 562. An angular *kyphosis* (*gibbus*) of the thoracic spine, a characteristic sign of tuberculous disease of the spine.

Spinal movements are now tested. The spine is hyperextended by drawing the patient's shoulders backwards. If the spine is fixed by disease or muscle spasm, it is impossible to increase the normal *lordosis*. Forward flexion is now observed. Note whether the patient tends to flex the knee and hip on one side on bending forward. This is characteristic

## CHAPTER 84

### THE SPINE

**Clinical Examination.** The complicated structure of the spine and its depth from the surface make clinical examination difficult and radiography all the more important. However, there is much to be learnt both directly and by inference on clinical examination. The three basic manifestations of spinal disorder to be studied are : (1) Deformity, fixed or mobile ; (2) muscle spasm ; and (3) pain, whether local, referred or due to pressure on nerve roots or the spinal cord.

The examination should begin with the patient standing, and the first observation to make is whether the pelvis is level or not, for a spinal curvature may be secondary to alteration in the pelvic level due to a short

leg. The pelvis should be made level by blocks placed beneath the shortened leg (Fig. 561). With the pelvis level, the posture of the spine should be inspected again. Note how patients hold their head, the presence or absence of round shoulders, flattening of the normal lumbar lordosis, and the general bearing—such a valuable clue to muscular tone, physical fitness, and mental make-up.

The combination of rigidity, flexibility and shock absorbing qualities which characterise the normal spine is dependent to a large extent on the intervertebral discs and also on the arrangement of the component sections of the spinal column. The factors adding mobility to strength are the junction of alternate mobile and fixed sections of the spine, and the presence of alternating curves. The mobile cervical spine is curved forwards and rests on the comparatively fixed thoracic spine which is bowed backwards. This in turn rests on the forward curving lumbar spine, which makes a sharp angular junction, the



FIG. 561. The first step in examining the spine in a standing patient is to level the pelvis by balancing any inequality in length of the legs.

and is influenced by heredity and development. The influence of congenital deformities and disease is excluded from the group of deformities termed postural. Postural deformities can be corrected in the earlier stages by muscular action, but with the passage of time they become habitual, and part of the patient's permanent muscle tone state. The round shoulders of the student are a classical example, in which occupation, muscular relaxation with mental effort, and bad working conditions all play a part in the postural alterations. With age, decrease in muscular tone results in a stoop affecting the thoracic spine; a corresponding hyperextension of the neck produces the "dowagers' hump." The differences in posture between the sexes and the temporary influence of pregnancy are well known.

The mental state of a person is reflected in the physique and posture. Contrast the straight firm spine of the stocky short individual with the drooping lax spine of the tall thin adolescent. It is during adolescence that postural deformities become established, and this is therefore the time to treat them. In mental disease the outlook on life affects the general muscular tone, and this is reflected in changed posture. A stoop, drooping shoulders, increased lordosis, and listless movements characterise the exhaustion of the neurasthenic, or any other emotionally worn out patient.

Effective treatment in many cases is difficult or impossible. It consists of general exercises as well as spinal exercises, and attention to the psychological factors. The younger the patient the greater is the hope of success.

**Defects of Spinal Muscle Tone.** Spinal posture is not only dependent on the spinal column, but also on the bracing effect of the spinal muscles. Thus muscular paresis, dystonia or incoordination cause spinal deformities. *Poliomyelitis* may paralyse the *erector spinæ* unilaterally with its associated muscles, but even if both sides are affected, the paralysis is uneven, and therefore there is not only a forward stoop, but also a lateral bend towards the more paralysed side; this is accompanied by rotation. Sometimes the deformity is severe. Patients tend to escape from surgical appliances when paralysis is marked and operative fusion of the spine may therefore be necessary.

Other neurological conditions, such as neurofibromatosis, syringomyelia and cerebral spastic paralysis, may cause scoliosis, and there are the rare muscular dystrophies which may also cause deformities. However, the commonest deformity is so ill-understood that it still masquerades under the name of "*idiopathic*" scoliosis (Fig. 563); no causal neurological disturbance is found, but there is considerable interference with epiphyseal development which may be secondary to heavy compression stresses on the concave side of the spinal curve. This type of scoliosis develops during the growing period, and often progresses very rapidly in early adolescence. The sites and varieties of the curves are complex. Of



of sciatica associated with a prolapsed lumbar disc pressing on a nerve root. Lateral flexion is now tested, by getting the patient to run the extended fingers down the outer side of the thigh without bending forward. In disc lesions, this movement is often relatively free whereas flexion and extension are greatly limited. When the whole spine is stiff, as in generalised osteo-arthritis, or spondylitis ankylopoetica, movements in all directions are equally restricted. Rotation of the spine is now tested by steadying the pelvis with the hands, and getting the patient to attempt to look over the right and then left shoulder.

The patient is now asked to lie on a couch. Additional information about the patient's pain and muscle spasm is obtained by watching the way in which the patient gets on the couch. Begin with the patient prone and well relaxed. Note the position of the spine and compare it with that of the erect spine. If the deformity is fixed, it remains unchanged, whereas if due to muscle spasm it partly or completely disappears. Run over the spinous process with the fingers, for very small deformities may be readily appreciated by touch. At the lumbosacral level, note any depression of the spinous processes. This may be due to spina bifida occulta in which there is a congenital defect of the laminae, or to spondylolisthesis, in which one vertebra has become displaced forward on the one below. Test the spinous processes and interspinous ligaments for tenderness. In localised disc lesions they are stretched and tender. Test the sacro-iliac joints for tenderness, and then for pain on movement; this is done by hyperextension of the straight leg with the patient prone; this movement first strains the hip and then, by rotating the pelvis, raises the homolateral sacro-iliac joint. Test the other sacro-iliac joint likewise. It is impossible during this test not to strain the lumbosacral joint; therefore pain may be produced at this site, but if both legs are raised, then the lumbar spine may be hyperextended without the sacro-iliac joints being stretched so forcibly and the pain may be localised to the lumbar region or the lumbosacral joint.

Look for the presence of any localised swelling, caused by muscle spasm, or disease, e.g. lumbar cold abscess or a tumour. Now test the muscles for tenderness. There may be tenderness in the trapezius muscles or around the vertebral borders of the scapula. The attachments of the quadratus lumborum to the iliac crest and to the last two ribs may be tender. Tenderness in the buttock and leg is associated with root pressure from a prolapsed disc.

The patient is now asked to turn over on the back, to facilitate palpation of the abdomen and the loins, and to complete the examination of the legs. Radiological examination of the affected region of the spine may now be required.

**Postural Defects.** The posture of the spine reflects the psychophysical make-up of the patient, equally obvious in other activities, such as walking, dancing or sport. It is a reflection of the patient's personality,

particularly in the lumbosacral region. Here failure of fusion of the laminae and spinous processes may produce *spina bifida occulta*—a less advanced degree of the developmental failure which gives rise to other types of spina bifida. *Spina bifida occulta* may be indicated by a depression, a tuft of hair, or merely a palpable irregularity of the spinous processes (see also Chap. 59). It may be mistaken on clinical examination for spondylolisthesis. *Spondylolisthesis*, meaning slipping of one vertebra backward or forward on the vertebra below, is a term which has become attached to a specific congenital deformity, in which there is a defect in the attachment of the laminae and lower articular processes to the pedicles of the

affected vertebra (Fig. 565), with the result that the vertebral body has no posterior support to prevent its slipping forward.

The condition may be encountered in the unslipped stage (spondylolysis). If slow slipping has occurred it is impossible to reduce it. Therefore if operation is carried out, the vertebra has to be fused in the position found. The condition most commonly affects the fifth lumbar vertebra, but may affect any lumbar vertebrae. It may occasionally be found to affect two or three vertebrae together; the resultant large gap to be bridged by grafting, decreases the chances of successful operation. If symptoms arise for the first time after middle age they are probably not due to further slipping, but to degenerative changes in the affected disc or discs, or to pressure on nerve roots of the cauda equina.

Spondylolisthesis is one of the causes of backache in young people. Attention may be drawn to it as the result of a minor accident, and it is often difficult to convince the patient that the accident was not responsible

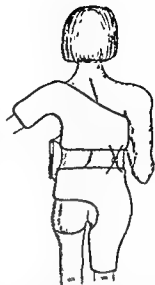


FIG. 564. The method of correction of a spinal deformity by use of Risser's jacket. The jacket, which includes one leg and one arm, is hinged on the right-hand side and is expanded by the use of the turn-buckle on the left.



FIG. 565. Spondylolisthesis. Marked slipping producing the characteristic sharp angular deformity at the level of the lumbosacral joint.

practical importance are the age of onset and the rate of progression of the condition. Severe and progressive deformity calls for operative fixation of the spine, but before this stage is reached treatment is based on the following principles. Spinal exercises tend to develop the stronger muscles more than the weak, and so may aggravate the deformity,



FIG. 563. Idiopathic scoliosis. Note the prominence of the ribs on the convex side of the thoracic curvature.

whereas general exercises, by maintaining a flexible spine and letting the curves "settle," are of some value. Recumbency (twenty-three hours out of the twenty-four) is of value in rapidly growing children, but because of its undesirable side-effects on schooling and personality is usually abandoned for more active measures. *Corrective jackets* may be merely supportive, or may have an adjustable device to produce pressure in the direction likely to correct the curvature. Supportive jackets have an occasional place in the treatment of established cases of slight or moderate severity or to correct muscular weakness, but have little place in the treatment of the more

severe cases. The corrective jacket of the Risser type is usually used as a preliminary to operation (Fig. 564). The deformity is slowly corrected and the spine is then fused in the corrected position by bone grafting.

**Operative Fusion.** Slivers and fragments of cancellous bone are packed in a prepared bed on the laminae and spines. The quantity of bone used is large and best provided by a bone bank. The operation may be staged. Continuous support first by the original plaster and then by a fitted cast or jacket is required for some eighteen months after operation. Other methods of operative correction using metallic struts are also used.

**Congenital Defects.** The spine is a frequent site of congenital defects,

particularly in the lumbosacral region. Here failure of fusion of the laminae and spinous processes may produce *spina bifida occulta*—a less advanced degree of the developmental failure which gives rise to other types of spina bifida. Spina bifida occulta may be indicated by a depression, a tuft of hair, or merely a palpable irregularity of the spinous processes (see also Chap. 59). It may be mistaken on clinical examination for spondylolisthesis. Spondylolisthesis, meaning slipping of one vertebra backward or forward on the vertebra below, is a term which has become attached to a specific congenital deformity, in which there is a defect in the attachment of the laminae and lower articular processes to the pedicles of the



FIG. 365. Spondylolisthesis. Marked slipping producing the characteristic sharp angular deformity at the level of the lumbosacral joint.

affected vertebra (Fig. 365), with the result that the vertebral body has no posterior support to prevent its slipping forward.

The condition may be encountered in the unslipped stage (spondylolysis). If slow slipping has occurred it is impossible to reduce it. Therefore if operation is carried out, the vertebra has to be fused in the position found. The condition most commonly affects the fifth lumbar vertebra, but may affect any lumbar vertebrae. It may occasionally be found to affect two or three vertebrae together; the resultant large gap to be bridged by grafting, decreases the chances of successful operation. If symptoms arise for the first time after middle age they are probably not due to further slipping, but to degenerative changes in the affected disc or discs, or to pressure on nerve roots of the cauda equina.

Spondylolisthesis is one of the causes of backache in young people. Attention may be drawn to it as the result of a minor accident, and it is often difficult to convince the patient that the accident was not responsible



FIG. 364. The method of correction of a spinal deformity by use of Risser's jacket. The jacket, which includes one leg and one arm, is hinged on the right-hand side and is expanded by the use of the turn-buckle on the left.

for the spinal defect. On examination, there may be an obvious deformity (Fig. 565) or merely a depression in the level of a spinous process, or an apparently normal spine. The defect is most easily recognised in lateral radiographs, in which the dog collar appearance is seen (Fig. 566). Antero-posterior views reveal the defect particularly if the displacement is marked; the fifth lumbar vertebra is seen almost end on, producing a typical bowed line across the sacrum.

The symptoms of spondylolisthesis are backache due to spinal weakness, osteo-arthritis, or root pressure (see Chap. 59). In the early stages backache is present only after exertion; it may be relieved by a firm corset, but should the pain persist, or get worse, operative fusion is indicated. This can be done through an anterior approach to the lumbosacral junction, although fusion from behind by means of an H graft is more usual. When the graft only needs to run from the sacrum to the fourth lumbar vertebra, the chances of success are good. In late cases with root pain, the nerve roots may need decompression as well.

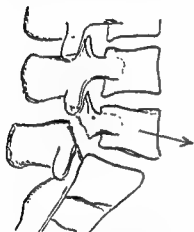


FIG. 566. The dog-collar effect seen in oblique and semi-oblique views of the lumbar spine in spondylolisthesis.

*Sacralisation and Lumbarisation.* The last lumbar and the first sacral vertebra tend to take over each other's roles. Thus the transverse processes of the fifth lumbar vertebra

may closely resemble the lateral masses of the sacrum, or be entirely fused to the sacrum (sacralisation). This may be accompanied by alterations in the levels of the issuing nerve roots and the site of maximum strain on the lumbar spine; the lumbosacral junction may be the joint between the fourth and fifth lumbar vertebrae. Usually, the deformity gives rise to no clinical effects, but if there is any irregularity the upset in spinal balance may lead to local arthritic changes. Occasionally painful arthritis of an abnormal joint occurs. Sometimes the first sacral vertebra resembles a fifth lumbar vertebra (lumbarisation).

*Adolescent Kyphosis.* This condition appears during adolescence and is characterised by the gradual development of a thoracic kyphosis, with the curve centered on the seventh thoracic vertebra. Attention is drawn to the condition by the appearance of the deformity, or by the occurrence of vague backache. It is met with equally in both sexes, and is found in some 2 per cent. of the population. The curve is a gradual one due to failure of development of the epiphyses of the vertebral bodies which thus become wedge shaped. The summation of the wedges produces the curve. Marginal sclerosis develops at the anterior margins of the vertebral bodies, giving rise to osteo-arthritis. Progression of the wedging ceases at the end of growth, but arthritic changes may continue. The condition is

similar to *osteocondritis juvenilis* affecting other epiphyses. Rest may be needed in the acute stages, though generally the discomfort is slight. Little can be done to alter the course of the disease, though the postural defect may be minimised by a course of spinal exercises.

**Schmorl's Nodes.** Central prolapse of the nucleus pulposus of intervertebral discs may occur into the vertebral bodies through defects in the centre of the epiphyseal plates. The corresponding defects in the vertebral bodies are seen in lateral radiographs of the spine. This type of disc prolapse has no apparent clinical significance.

**Senile Osteoporosis.** Loss of muscle tone and decalcification of the vertebræ in old age weaken the spine which gradually yields, and increases the upper thoracic kyphosis. Similar changes occur in the lumbar region. The intervertebral discs degenerate and prolapse into the vertebral bodies, or become extruded between the vertebræ and become surrounded by osteophytes (osteo-arthritis). Slow collapse of a vertebra may occur, or, as the result of a trivial fall, collapse may be acute. In acute cases bed rest may be required, but active treatment is carried out if possible. The support of a corset, or light plaster jacket may help, but exercises if the patient can co-operate are more valuable. As in other osteoporotic conditions general hormone treatment is most uncertain in action although implantations with testosterone may help in elderly women. Butazolidin is a valuable aid in relieving pain, starting with 200 mg. t.d.s. and reducing this after a month to 100 mg. t.d.s.

**Spondylitis Ankylopoetica.** This disease is allied to rheumatoid arthritis, but the reason for its localisation mainly to the spine is unknown. Rarely the two conditions are seen together. It responds, but less dramatically than rheumatoid arthritis, to cortisone and Butazolidin. Males are more commonly affected than females, and the greatest incidence of symptoms is in the third and fourth decades. The disease produces fixation of joints in a peculiar sequence; beginning in the sacro-iliac joints, it spreads to the diarthrodial joints of the lumbar spine and then further and further up the spine. The lumbar intervertebral discs are next involved progressively up the spine. They are destroyed and fused and the spinal ligaments become ossified. Eventually the whole spine may become rigid from the sacrum to the second cervical vertebra (Fig. 567). Movements between the occiput and the atlas and between the atlas and axis always remain. The costo-transverse joints become involved, and rib movements are consequently restricted. The serious effect on respiration is increased by the development of a marked thoracic kyphosis, so that eventually only diaphragmatic respiration remains. The hip joints may be involved, and very rarely the shoulder joints. The intervertebral discs are bridged by osteophytic outgrowths from the vertebral bodies along the longitudinal ligaments of the spine. In osteo-arthritis, the osteophytic growths begin at the edges of the vertebral bodies whereas in ankylosing spondylitis they begin on the side of the vertebral body above and below the discs.

Eventually the whole spine is turned into a solid rod (poker back) which because of the bulges opposite the discs resembles bamboo radiographically (Fig. 568).

*Clinical Features.* The disease is very variable in onset and progression, and may burn itself out leaving little change. Gradual development of



FIG. 567. Characteristic appearance in severe spondylitis ankylopoetica. The knees are flexed in order to carry the spine as upright as possible. The head is fixed in marked forward flexion. The patient's vision is grossly restricted and he cannot see sufficiently far ahead to walk comfortably.



FIG. 568. "Bamboo spine" (spondylitis ankylopoetica). The intervertebral discs are partly calcified and their natural margins are bridged over by calcification in the longitudinal ligaments.

deformity with mild pain is the usual course although deformity alone may be the presenting symptom (Fig. 567). Sometimes pain in the lumbar region radiating to the buttocks and thighs is the main complaint. In young people, there may be an acute onset with severe pain, fever and raised E.S.R. In a few cases, obliteration of the sacro-iliac joints is seen, but no further evidence of progress of the disease follows. The diagnosis is based on the fixity of the spine in all directions, and the radiological evidence of bilateral sacro-iliac sclerosis. The activity of the disease is

determined by the duration and severity of the symptoms. A flare up in an established case may occur. The E.S.R. is a good guide to the activity of the disease.

*Treatment.* The more active the disease the better is the response to treatment. Both Butazolidin and cortisone may control the disease, but the most effective remedy for reducing pain and aborting an acute phase is still deep X-ray therapy, but the late inactive phase does not respond and the response to repeated radiation is less and less satisfactory. Osteo-arthritic changes may follow the abatement of the active phase, and require similar treatment to osteo-arthritis due to other causes. Care must be taken, however, not to reduce air entry to the chest still further by restrictive corsets. Arthritis of the hips in this condition responds unsatisfactorily to arthroplasty because of the formation of new bone around the hip. Respiratory exercises may help to minimise contraction of the thorax. The body and head may become so bent forward that the patient cannot see the ground sufficiently far ahead to walk comfortably. In these circumstances, a spinal osteotomy to correct the angulation of the spine is valuable, as it not only increases the range of vision, but frees the abdominal wall and diaphragm for better respiratory excursion.

*Torticollis. Congenital Torticollis.*—In this condition the baby develops a combination of flexion of the head to one side and rotation of the chin to the opposite side, produced by contraction of one sternomastoid muscle. Two very distinct entities may be found: (1) Sternomastoid contracture, perhaps accompanied by a "tumour" in the upper part of the muscle, but without any facial hemiatrophy. (2) Sternomastoid contracture, with or without a "tumour" (and perhaps contracture of other tissues) together with facial hemiatrophy. In (2) there is a congenital malformation affecting the head and cervical muscles; the resistance offered to delivery by the deformity may rupture a few fibres of the sternomastoid with the production of a hæmatoma and consequent "tumour." No matter what is done, the facial hemiatrophy remains and often becomes more obvious as the child grows. Attempts should be made to maintain the normal posture of the head as outlined below. In (1), the more usual type of torticollis, there has been trauma to the sternomastoid during delivery and a hæmorrhagic "tumour" has developed. Such tumours are common, but they are seldom followed by any contracture, and merely require supervision and gentle stretching from time to time. The unjustified assumption that these tumours will lead to contracture is due to their almost invariable association with already established contractures (*vide* type 2 above). It is difficult to recognise early mild degrees of facial hemiatrophy—hence the need for supervision and a guarded prognosis in all cases.

Established torticollis is treated according to severity along the following lines: (1) Manipulation and application of a plaster thoraco-cervical collar in the over-corrected position. (2) Division of the sterno-



mastoid, followed by corrective exercises or application of a plaster collar. (3) Wider division of the soft cervical tissues, all of which are contracted in severe cases associated with facial hemiatrophy, and fixation in plaster in the over-corrected position. A congenital defect of a cervical vertebra is a very rare cause of congenital torticollis.

*Acquired Torticollis.* The term torticollis is applied to any spasmodic or persistent deformity of the neck, and the causes when catalogued resemble those of deformities in other parts of the spine. *Reflex Causes.*—

(1) Habit; (2) ocular disturbance; (3) irritation of cervical nerves (e.g. in cervical adenitis, otitis media or parotitis). *Neurological Causes.*—

(1) Muscular paresis from poliomyelitis; (2) spasmodic torticollis (cause unknown). *Cervical Disease.*—(1) Scar tissue contracture, e.g. after excision of cervical glands; (2) tuberculosis of the cervical spine; (3) prolapse of a cervical disc; (4) osteo-arthritis of the cervical spine; (5) muscle spasm due to *myofibrositis*, always difficult to distinguish from protective muscle spasm from some underlying disc or bone disease—the common stiff-neck.



FIG. 569. Early tuberculous disease of the spine showing narrowing of the intervertebral disc between the twelfth thoracic and the first lumbar vertebrae.

### TUBERCULOUS DISEASE OF THE SPINE

#### (Pott's Disease)

For the general consideration of tuberculous osteitis the reader is referred to Chap. 80. The maximum incidence of tuberculosis of the spine is in the third decade of life, but it is almost equally common in the first decade, and may be met with at all times of life, even in old age. The infection of the spine is usually blood-borne, although it is occasionally transmitted by lymphatics; the embolic origin of the disease inevitably means that there are often several closely associated foci of infection which eventually become fused. The disease commonly begins in a vertebral body adjacent to an intervertebral disc and spreads in the vertebral body partly destroying it, and then having undermined and destroyed the disc, spreads to the vertebral body on

the other side of it (Fig. 569). Occasionally the disease spreads under the anterior longitudinal ligament and so involves several vertebral bodies (Fig. 570). Destruction of the bone is followed by collapse of the bodies of the affected vertebrae. Angulation of the spine is always sharp and may be very marked if several vertebrae are involved. Necrosis of the bone is followed by liquefaction and invasion with leucocytes to form extensive "cold" abscesses. The form and direction in which an abscess points is determined by the site of infection in the spine and the arrangement of the surrounding tissue planes. In the upper cervical spine, a retropharyngeal abscess may form, or in the lower cervical spine, an abscess is directed toward the root of the neck. In the thoracic spine, an abscess may remain localised with consequent risk of compression of the spinal cord, or it may track along an intercostal space. In the lower thoracic and lumbar region, the sheath of the psoas muscle guides the pus into the iliac fossa, or below Poupart's ligament into the thigh.



FIG. 570. Severe angular deformity of the thoracic spine due to tuberculous destruction of several thoracic vertebral bodies. The condition was accompanied by a paraplegia which was relieved by anterolateral decompression.

Tuberculous toxins inhibit local reparative processes, and therefore the living bone becomes separated by inactive granulation tissue, the extent of which depends on the number of vertebrae destroyed. Unless the vertebral bodies on the two sides of the dead space come into contact by collapse, very considerable time will be required for healing. It is therefore often wise to permit some deformity to hasten healing, rather than to insist on complete reduction of the deformity. Slow invasion of fibrous tissue by bone occurs in the healing stage, and abscesses dry up and may become calcified.

**Diagnosis.** Attention may be drawn to the disease by pain in the back.

This is often unobtrusive and the appearance of a kyphosis or occasionally a cold abscess in the groin, mimicking a hernia, may be the first thing to alarm the patient. Nocturnal restlessness and night starts may be seen in children, who also limit their stooping and walk stiffly. Jarring always makes the pain worse. On examination, the first thing to be seen is often the angular deformity (Fig. 562). If the disease has not advanced to this stage, there is still marked protective muscle spasm, unrelieved by recumbency. The night-starts are probably due to intermittent pain produced by muscle spasm relaxing during sleep. The affected area of the spine is tender on pressure or percussion over the spinous processes. Examination is made for a cold abscess in the appropriate position. Thus abdominal palpation is essential in lumbar lesions. With suspicions aroused by the deformity, local pain and muscle spasm, radiographic examination is ordered. In children, the destruction is usually easily visible. In adults, it may not be so easy to detect in the early stages, and in the elderly the appearances of a secondary neoplasm may, especially if it permits local disc prolapse, resemble tuberculosis of the spine. In the active stage of the disease the vertebral body has eroded softened edges, and the intervertebral disc is usually completely destroyed. Sequestra are occasionally seen. The paravertebral tissues may be bulged by an abscess, or the psoas shadow may be distorted.

*Investigations.* The lungs are always X-rayed for evidence of a primary focus of infection. The E.S.R. is increased in active tuberculous disease. It is not a specific test for tuberculosis, however, but is a good guide to the progress of the disease. The tuberculin test is discussed in Chap. 80. When the diagnosis is in doubt, patients should be admitted to hospital for observation of temperature, repeated E.S.R. and effect of rest on any muscle spasm.

*Differential Diagnosis.* (1) *Developmental Abnormality.*—Recognised radiologically: there is no erosion of bone. (2) *Osteochondritis Juvenilis* of the thoracic spine.—Many vertebral bodies involved each slightly wedged. No general disturbance. (3) *Idiopathic Scoliosis.*—No vertebral collapse or erosion, no general disturbance. (4) *Still's Disease.*—Spinal involvement may be marked in rheumatoid disease of childhood, but is accompanied by a raised temperature, involvement of other joints and enlargement of the lymphatic glands and spleen. (5) *Spondylitis Ankylopoetica.*—This disease may cause confusion in the acute stage. Characteristically the sacro-iliac joints are blurred or fused. (6) *Non-tuberculous Osteitis.*—This is rare in the spine, but infection of a disc may follow lumbar puncture. There is a greater general disturbance and leucocytosis. A primary focus of infection may exist elsewhere. (7) *Neoplasm.*—Primary sarcoma of the spine is rare, but retroperitoneal sarcoma may cause confusion. A secondary carcinoma of the spine is much more common and much more likely to be mistaken for tuberculosis. Vertebral collapse may obscure the radiological details, but the discs tend

to remain intact. In multiple myeloma the appearances are similar to secondary carcinoma. They both tend to affect widely separated vertebrae. Other characteristics of myeloma are discussed in Chap. 83. (8) *Senile osteoporosis* with collapse of a vertebral body may raise suspicion of senile tuberculosis. Osteoporosis also occurs in *Cushing's disease* and may cause collapse of vertebrae. In both conditions the general decalcification, the collapse of other vertebral bodies and the escape from destruction of the discs, help in the diagnosis. (9) *Paget's Disease*.—There are usually characteristic changes in other bones.

**Treatment** (see also under Tuberculosis, Chap. 80). The policy of complete sanatorium rest and minimal interference has been slowly changing since the discovery of antibiotics. These not only have speeded the recovery of the well-established disease and healed minimal lesions, but, by greatly diminishing the risk of secondary infection, have made more extensive surgery possible. Operation was formerly carried out for advanced disease with much bone destruction, for large abscesses or for paraplegia, but nowadays earlier interference is the rule in other cases to cut short the healing time.

*The Acute Phase.* Complete rest under a sanatorium regime and administration of antibiotics constitute the corner stone of treatment in the active phase, or when there is suspicion of reactivity of the disease. This not only provides the best conditions for overcoming the disease, but also provides for systematic observation, which is often necessary to determine the degree of activity of the disease. The patient's weight, general condition, temperature and E.S.R. are continuously charted. *Rest* is provided by recumbency supplemented by spinal fixation appropriate to the age and condition of the patient. In children, the Bradford frame provides a convenient and adjustable support. In adults, a plaster bed may be more satisfactory. Turning the patient is essential to avoid renal stasis and to vary skin pressure areas. A turning-case must therefore be provided. During the later stages of treatment, patients (particularly children) may be kept in the prone position in the case for most of the day. *Antibiotics*.—Streptomycin is administered according to the principles outlined under general treatment (Chap. 80). It may also be injected into joints or into abscess cavities to control the tubercular infection and reduce the risk of secondary infection. Improvement is shown by a drop in temperature and E.S.R., and the better general condition of the patient. Radiologically, the "soft" extending outlines of the lesion are replaced by better defined bone and sclerosis. Osteoporosis diminishes and abscess cavities shrink. New bone may be seen bridging the gap.

*Cold Abscesses.* The treatment is aspiration under aseptic conditions, and instillation of streptomycin and penicillin (0.25 gm. streptomycin, 100,000 units penicillin in 5 ml. distilled water). Calcified abscesses, those with sequestra, and those inaccessible to aspiration (e.g. a paravertebral

This is often unobtrusive and the appearance of a kyphosis or occasionally a cold abscess in the groin, mimicking a hernia, may be the first thing to alarm the patient. Nocturnal restlessness and night starts may be seen in children, who also limit their stooping and walk stiffly. Jarring always makes the pain worse. On examination, the first thing to be seen is often the angular deformity (Fig. 562). If the disease has not advanced to this stage, there is still marked protective muscle spasm, unrelieved by recumbency. The night-starts are probably due to intermittent pain produced by muscle spasm relaxing during sleep. The affected area of the spine is tender on pressure or percussion over the spinous processes. Examination is made for a cold abscess in the appropriate position. Thus abdominal palpation is essential in lumbar lesions. With suspicions aroused by the deformity, local pain and muscle spasm, radiographic examination is ordered. In children, the destruction is usually easily visible. In adults, it may not be so easy to detect in the early stages, and in the elderly the appearances of a secondary neoplasm may, especially if it permits local disc prolapse, resemble tuberculosis of the spine. In the active stage of the disease the vertebral body has eroded softened edges, and the intervertebral disc is usually completely destroyed. Sequestra are occasionally seen. The paravertebral tissues may be bulged by an abscess, or the psoas shadow may be distorted.

*Investigations.* The lungs are always X-rayed for evidence of a primary focus of infection. The E.S.R. is increased in active tuberculous disease. It is not a specific test for tuberculosis, however, but is a good guide to the progress of the disease. The tuberculin test is discussed in Chap. 80. When the diagnosis is in doubt, patients should be admitted to hospital for observation of temperature, repeated E.S.R. and effect of rest on any muscle spasm.

*Differential Diagnosis.* (1) *Developmental Abnormality.*—Recognised radiologically: there is no erosion of bone. (2) *Osteochondritis Juvenilis* of the thoracic spine.—Many vertebral bodies involved each slightly wedged. No general disturbance. (3) *Idiopathic Scoliosis.*—No vertebral collapse or erosion, no general disturbance. (4) *Still's Disease.*—Spinal involvement may be marked in rheumatoid disease of childhood, but is accompanied by a raised temperature, involvement of other joints and enlargement of the lymphatic glands and spleen. (5) *Spondylitis Ankylopatica.*—This disease may cause confusion in the acute stage. Characteristically the sacro-iliac joints are blurred or fused. (6) *Non-tuberculous Osteitis.*—This is rare in the spine, but infection of a disc may follow lumbar puncture. There is a greater general disturbance and leucocytosis. A primary focus of infection may exist elsewhere. (7) *Neoplasm.*—Primary sarcoma of the spine is rare, but retroperitoneal sarcoma may cause confusion. A secondary carcinoma of the spine is much more common and much more likely to be mistaken for tuberculosis. Vertebral collapse may obscure the radiological details, but the discs tend

narrowing of the intervertebral discs. Patchy sclerosis may be seen and narrowing and degeneration of the diarthrodial joints. Accompanying senile osteoporosis may lead to collapse of discs and bodies with deformity, but generally the normal posture of the spine is preserved in primary osteo-arthritis. In secondary osteo-arthritis the deformity produces the abnormal strains which cause the osteo-arthritis. Treatment consists of rest and warmth in the acute stages, radiant heat, massage and exercises in the stage of "grumbling" discomfort, and the provision of a lumbo-sacral corset for recurrent acute attacks. A spinal support is most likely to be effective in localised arthritis.

thoracic abscess) are treated by open operation. Curettage and removal of thickened walls, sequestra and debris are required (cf. treatment of persistent cavities in osteomyelitis, Chap. 79).

*The Healing Phase.* When the patient's general condition is satisfactory, the question of taking further surgical steps to heal the disease may be considered. Children do not suffer seriously from a long period of rest, whereas elderly patients tolerate it badly; therefore activity, even with a "grumbling" lesion, may have to be permitted. The natural processes of repair may be hastened by operative treatment which is designed to eradicate dead tissue and fill the resultant space with bone grafts or soft tissue, or to supplement natural fixation with external grafts. The grafts are applied to the laminae and spinous processes. They consist of either rigid compact bone lengths which are fixed to the spines, or the more satisfactory bone slivers and cancellous bone chips, which are packed over the rawed laminae and spines and closed in by suture of the erector spinæ muscles. Immobilisation in recumbency is then necessary for three months or more.

*The Healed Phase.* Graduated activity is begun, but the patient must wear a spinal brace to maintain fixation of the spine. Watch is kept for recurrence of activity of the disease; the patient is therefore examined at regular intervals. Eventually, if the patient's general condition is satisfactory and there is radiological evidence of sound healing of the bone, the brace may be discarded. Yearly examinations is then all that is required.

*Paraplegia.* The treatment of paraplegia in tuberculosis of the spine is discussed in Chap. 59.

## OSTEO-ARTHRITIS OF THE SPINE

### (Spondylosis)

The disease may be met with as part of a generalised osteo-arthritis (primary osteo-arthritis) (see Chap. 81), or as a localised condition following trauma, prolapse of a disc, congenital deformities and healed tuberculosis (secondary osteo-arthritis). The restriction of movement of the spine is in proportion to the length of spine involved. The symptoms are stiffness of the spine on getting up in the morning, or after any lengthy rest. This works off gradually with activity, but the spine tends to stiffen and become painful at the end of the day. Attacks of acute lumbago may occur at irregular intervals. The disability often varies with the weather. Pain is very variable and intermittent. Advanced changes are frequently found in patients who have never complained of pain. With the passage of time pain tends to diminish and stiffness to increase. The correction of associated defects, such as a stiff arthritic hip, or balancing a short leg may be of value in reducing the strains on the spine. Radiography shows osteophytic outgrowths from the vertebral bodies. There may be

infraspinatus, subscapularis and teres minor around the anatomical neck of the humerus, and forms the floor of the subacromial bursa. It may be partially or completely ruptured.

The clinical features of subacromial bursitis are pain on movement of the shoulder, pain referred to the deltoid insertion and the upper part of the arm, a catch on movement of the shoulder particularly on adduction from the vertical, a visible swelling under the upper part of the deltoid, local tenderness on pressure around the margins of the acromion, and limitation of shoulder movement due to muscle spasm or the causative lesion.

*Treatment* depends on the cause of the bursitis, but in acute bursitis aspiration of the bursa may be of therapeutic as well as of diagnostic value when inflammation is present. In calcifying tendinitis, the bursa may be washed out with saline under local anaesthesia. This is very effective if the calcified material has burst into the bursa. In chronic cases the calcific deposit may have to be removed by operation. In sub-acute and more persistent varieties the injection of 1-2 ml. of hydrocortisone, under local anaesthesia, into the bursa sometimes produces dramatic relief within a few days. If this fails physiotherapy is indicated. Short-wave diathermy is usually the most effective form of treatment. Occasionally when pain is intractable, the acromion has to be removed to decompress the tissues.

**Degenerative Tendinitis of the Short Rotator Cuff.** Postmortem examination in the later decades of life has often shown degenerative changes of the short rotator cuff and the tissues around it. These changes are probably related to old age and a consequent deficiency of blood supply. This spontaneous degeneration, which itself may or may not cause symptoms, increases the susceptibility of the rotator cuff to minor injury and increases the liability to pain and stiffness of the joint after immobilisation. The disease may develop insidiously, beginning with night pain in the shoulder and inability to lie on it. Loss of mobility, particularly internal and external rotation, and later abduction, is then noted. The onset may be precipitated by minor trauma, or follow immobilisation of the shoulder for an injury of the arm. Minor ruptures of the rotator cuff may occur. Examination shows a varying degree of



FIG. 571. Calcifying tendinitis in the short rotator cuff.



## CHAPTER 85

### THE SHOULDER

THE shoulder, though particularly free from the arthritic diseases affecting other joints, is often the seat of persistent and vague pains which may be difficult to elucidate. Painful disorders of the shoulder are divided into two groups: (a) Those caused by nerve-root compression, e.g. in cervical spondylosis, brachial plexus injuries, and the cervical rib syndrome. Pain radiates as far as the fingers and is accompanied by localising neurological signs. (b) Those in which there is local tenderness around the shoulder joint, and usually some interference with the function of the joint, e.g. degenerative tendinitis, subacromial bursitis, osteoarthritis of the acromioclavicular joint, and local diseases of the shoulder joint or surrounding bones.

In young women (e.g. typists) due to over-exertion, and sometimes in older women with failing muscle tone, neuralgic pains of a vague root nature occur as the result of drooping of the shoulder, aggravated as a rule by their particular employment. The drooping shoulders, the physical and mental tone of the patient, and the fact that the condition is relieved by a course of shoulder girdle exercises are characteristic.

Arthritis of the acromioclavicular joint may follow injury to the joint. As the joint is palpable and deformity visible the diagnosis is easily made. Pain occurs only with scapular rotation and not with shoulder movement. Excision of the outer end of the clavicle usually cures the pain.

The *sterno-clavicular joint* may slowly subluxate, making the medial end of the clavicle prominent. The complaint is usually one of altered appearance and not of dysfunction. There is no effective treatment.

**Subacromial Bursitis.** The subacromial bursa has little space to expand between the acromion and the head of the humerus. Any effusion into the bursa or swelling in its floor is thus subject to pressure, and produces pain; this pain may be of dramatic onset, and "freeze" the shoulder. The causes of subacromial pain are: (1) *Acute Bursitis*.—This may be traumatic or rarely inflammatory. (2) *Calcifying Tendinitis*.—This is a variety of degenerative tendinitis found in patients usually under forty-five years. Calcification in the tendons in the floor of the bursa causes chronic pain (Fig. 571), and when the material ruptures into the bursa an acute "frozen" shoulder is produced. (3) *Degenerative Tendinitis*.—This is a common lesion in older patients developing spontaneously or as the result of minor trauma. (4) *Rupture of the Short Rotator Cuff*.—This cuff is formed by the insertions of supraspinatus,

infraspinatus, subscapularis and teres minor around the anatomical neck of the humerus, and forms the floor of the subacromial bursa. It may be partially or completely ruptured.

The clinical features of subacromial bursitis are pain on movement of the shoulder, pain referred to the deltoid insertion and the upper part of the arm, a catch on movement of the shoulder particularly on adduction from the vertical, a visible swelling under the upper part of the deltoid, local tenderness on pressure around the margins of the acromion, and limitation of shoulder movement due to muscle spasm or the causative lesion.

*Treatment* depends on the cause of the bursitis, but in acute bursitis aspiration of the bursa may be of therapeutic as well as of diagnostic value when inflammation is present. In calcifying tendinitis, the bursa may be washed out with saline under local anaesthesia. This is very effective if the calcified material has burst into the bursa. In chronic cases the calcific deposit may have to be removed by operation. In sub-acute and more persistent varieties the injection of 1-2 ml. of hydrocortisone, under local anaesthesia, into the bursa sometimes produces dramatic relief within a few days. If this fails physiotherapy is indicated. Short-wave diathermy is usually the most effective form of treatment. Occasionally when pain is intractable, the acromion has to be removed to decompress the tissues.



FIG. 571. Calcifying tendinitis in the short rotator cuff.

**Degenerative Tendinitis of the Short Rotator Cuff.** Postmortem examination in the later decades of life has often shown degenerative changes of the short rotator cuff and the tissues around it. These changes are probably related to old age and a consequent deficiency of blood supply. This spontaneous degeneration, which itself may or may not cause symptoms, increases the susceptibility of the rotator cuff to minor injury and increases the liability to pain and stiffness of the joint after immobilisation. The disease may develop insidiously, beginning with night pain in the shoulder and inability to lie on it. Loss of mobility, particularly internal and external rotation, and later abduction, is then noted. The onset may be precipitated by minor trauma, or follow immobilisation of the shoulder for an injury of the arm. Minor ruptures of the rotator cuff may occur. Examination shows a varying degree of

fixation of the shoulder. Firstly, there is fixation produced by muscle spasm due to pain from the associated subacromial bursitis, and, secondly, there is limitation of movement due to capsular contracture. Both the active range and the passive range of movement are decreased, and the decrease in internal and external rotation is proportional to the decrease in abduction, suggesting a uniform contracture of the rotator cuff. Most of the remaining movement may be scapulothoracic, for the glenohumeral joint is "frozen" in advanced cases. There is wasting of the short rotator muscles and of the deltoid. Radiological examination often shows little change, although sometimes there is some sclerosis or haziness around the rotator cuff attachments. Osteo-arthritic changes in the shoulder are conspicuously rare. The condition has to be distinguished from injuries of the short rotator cuff. In these cases, there is a full range of passive movement, and in the lesser injuries, though active abduction cannot be initiated, it may be maintained when full abduction is carried out passively. In subacromial bursitis, there is again usually a full range of passive movement though it may be painful, and there is the characteristic catch on active adduction.

*Treatment.* This is unsatisfactory. Pain is the principal complaint, and is not always proportional to the limitation of movement. Aspirin and codeine are of value, and in a few cases Butazolidin is effective. Long-continued physiotherapy consisting of short-wave diathermy, massage and exercises is required. Slow decrease in pain takes place, but there is not always a corresponding improvement in shoulder movements; some patients are left with a very stiff shoulder. Because of the general contraction of the whole rotator cuff, manipulation is usually ineffective, and forcible manipulation is dangerous. If manipulation is employed, it should consist of repeated gentle stretching.

*Bicipital Tendinitis.* The tendon is involved in the degenerative changes affecting the short rotator cuff in the elderly. The tendon sheath may be tender or swollen below the level of the head of the humerus. Rupture of the tendon may occur with a resultant alteration in the shape of the biceps on contraction. In the elderly there is little disturbance of function. Occasionally the rupture occurs in a young adult. Open suture of the tendon to the capsule of the shoulder joint, without opening the joint, is then indicated. Following injury a subacute form of tendinitis may occur in the young.

*Tuberculosis of the Shoulder Joint.* The shoulder, in spite of its proximity to the lungs, is rarely the seat of tuberculosis. Two forms of infection are recognised: (a) A process of slow caseation without pus formation, confined chiefly to the head of the humerus (*caries sicca*). (b) The more usual or florid form, as found in other joints; the disease begins in the metaphysis close to the epiphyseal plate and spreads into the joint. Cold abscesses develop and sinuses form around the lower margins of the deltoid muscle. *Treatment* consists of: (a) Sanatorium

regime ; (b) antibiotics and immobilisation of the arm in a thoraco-brachial plaster. Bony ankylosis of the shoulder is aimed at, but is particularly difficult to achieve. Excision of the joint may be required as a preliminary step, and this is usually followed by fibrous ankylosis. To obtain bony ankylosis extra-articular fixation with grafts is required.

**Arthrodesis of the Shoulder Joint.** Due to the small area of bony contact, and the free mobility of the scapula, the shoulder is one of the most difficult joints to fuse. Arthrodesis may be required for pain in an arthritic shoulder, or for the completion of healing in a tuberculous joint. It is more often required to correct an adducted position of the joint produced by disease, or to stabilise the joint following paralysis of the muscles around (e.g. in poliomyelitis or as the result of brachial plexus injury). The procedure adopted depends on the availability or otherwise of the acromion and glenoid. If these can be used, the head of the humerus is fixed to the glenoid by a long screw, and the osteotomised acromion bent down and fixed to the humeral head. If these structures are not available a subglenoid strut of bone may have to be inserted between the scapular margin and the humerus.

**Recurrent and Habitual Dislocation of the Shoulder Joint.** These conditions are discussed in Chap. 91.

## CHAPTER 86

### THE ELBOW

**Osteo-arthritis.** The elbow is frequently the seat of osteo-arthritis which is usually secondary to an old injury (e.g. dislocation, fractures of the head of the radius, fractures of the olecranon, malunion of fractures of both bones of the forearm, supracondylar fractures, and T-shaped fractures into the joint). The symptoms are those of osteo-arthritis elsewhere, namely, stiffness after rest or excessive use, recurrent effusion, and limitation of movement. The development of loose bodies may produce acute incidents of locking. With involvement of the head of the radius pronation and supination are limited.

**Treatment.** In subacute osteo-arthritis, physiotherapy may give the patient sufficient relief. Loose bodies may have to be removed. Easy access to the joint may be obtained on the lateral side by detaching the extensor muscle origins from the lateral epicondyle and then incising the muscle group for 1 inch in the line of the epicondyle down the head and neck of the radius. With the elbow flexed, the anterior compartment of the joint can be inspected, and with the joint extended the posterior compartment can be inspected. The same incision may be employed for excision of the head of the radius; this procedure may be of value to restore pronation and supination when the head of the radius is the chief seat of arthritic changes. When pain on movement is the chief symptom, relief may be obtained by fixing the elbow at a right angle by means of a moulded leather or polythene splint. Occasionally fusion of the elbow has to be considered. Humero-ulnar fusion is difficult, as the head of the radius has to be left free to rotate if pronation is to be preserved. Arthroplasty of the elbow joint by excision has also to be considered. Movement can usually be preserved at the cost of some loss of strength and stability. The occupation of the patient and whether or not the disease is bilateral influence the choice of treatment.

**Osteochondritis Dissecans.** This disease is discussed in more detail in relation to the knee joint (Chap. 89). In the elbow the capitellum is the source of the loose body. It may be detached from here as the result of direct injury, or it may become detached months or years after as the result of cartilage erosion. The fragment arises most commonly from the inferior aspect of the capitellum, where the head of the radius would damage it most forcibly if dislocated behind it. Occasionally the site of the detachment is not easily located radiologically. Usually there is a single loose body, but it may become fragmented (Fig. 572). Most commonly it lies in the anterior compartment of the joint in the coronoid fossa. When the fragment migrates into the posterior part of the joint it

lies in the olecranon fossa, where it may be more difficult to see. The loose body should be removed, and the site of origin inspected and saucerised to prevent separation of further fragments.

**Traumatic Ulnar "Neuritis"** (see Chap. 61).

**Tennis Elbow.** The pathological basis of this common condition still remains obscure. The patient complains of pain over the lateral epicondyle, aggravated by use of the extensor muscles. A tender spot is found above or in front of the epicondyle, and extension of the elbow with firm flexion of the wrist produces pain over the head of the radius. Occasionally pain is more widespread in the extensor group of muscles. Aching pain radiates both up and down the arm and may be referred to the ulnar border of the hand. Occasionally a small cystic swelling may be found in the extensor origin. A similar condition occurs less commonly around the medial epicondyle, and then affects the use of the flexor muscles. The condition appears to be allied to degenerative tendinitis in other situations.



FIG. 572. Osteochondritis dissecans of the elbow. The bony flake with some calcification of the surrounding cartilage is well shown.

**Treatment.** The effectiveness of physiotherapeutic treatment is proportional to the length of time the condition has existed. If the complaint has been present for months, or there is a palpable thickening or visible swelling, physiotherapy is unlikely to succeed. Treatment consists of radiant heat, massage and exercises, perhaps supplemented by short-wave diathermy, or faradism to the extensor muscles. If this fails, local infiltration of the area with procaine should be tried. When this has abolished the local pain, the extensors should be stretched, by manipulation of the elbow and wrist. The injection may need to be repeated. Hydrocortisone (25 mg. in 2 ml.) may be injected in place of procaine. If pain persists, a cure can usually be effected by division of the extensor origins from the epicondyle. This is a simple procedure which can be carried out through an incision only an inch long. When a small cyst or bursa exists it should be removed.

**Olecranon Bursitis.** This is often the result of chronic occupational irritation of the bursa, but acute inflammation may develop from infection on the hand or forearm. The condition is analogous to prepatellar bursitis and is treated similarly (Chap. 89).

## CHAPTER 86

### THE ELBOW

**Osteo-arthritis.** The elbow is frequently the seat of osteo-arthritis which is usually secondary to an old injury (e.g. dislocation, fractures of the head of the radius, fractures of the olecranon, malunion of fractures of both bones of the forearm, supracondylar fractures, and T-shaped fractures into the joint). The symptoms are those of osteo-arthritis elsewhere, namely, stiffness after rest or excessive use, recurrent effusion, and limitation of movement. The development of loose bodies may produce acute incidents of locking. With involvement of the head of the radius pronation and supination are limited.

**Treatment.** In subacute osteo-arthritis, physiotherapy may give the patient sufficient relief. Loose bodies may have to be removed. Easy access to the joint may be obtained on the lateral side by detaching the extensor muscle origins from the lateral epicondyle and then incising the muscle group for 1 inch in the line of the epicondyle down the head and neck of the radius. With the elbow flexed, the anterior compartment of the joint can be inspected, and with the joint extended the posterior compartment can be inspected. The same incision may be employed for excision of the head of the radius; this procedure may be of value to restore pronation and supination when the head of the radius is the chief seat of arthritic changes. When pain on movement is the chief symptom, relief may be obtained by fixing the elbow at a right angle by means of a moulded leather or polythene splint. Occasionally fusion of the elbow has to be considered. Humero-ulnar fusion is difficult, as the head of the radius has to be left free to rotate if pronation is to be preserved. Arthroplasty of the elbow joint by excision has also to be considered. Movement can usually be preserved at the cost of some loss of strength and stability. The occupation of the patient and whether or not the disease is bilateral influence the choice of treatment.

**Osteochondritis Dissecans.** This disease is discussed in more detail in relation to the knee joint (Chap. 89). In the elbow the capitellum is the source of the loose body. It may be detached from here as the result of direct injury, or it may become detached months or years after as the result of cartilage erosion. The fragment arises most commonly from the inferior aspect of the capitellum, where the head of the radius would damage it most forcibly if dislocated behind it. Occasionally the site of the detachment is not easily located radiologically. Usually there is a single loose body, but it may become fragmented (Fig. 572). Most commonly it lies in the anterior compartment of the joint in the coronoid fossa. When the fragment migrates into the posterior part of the joint it

lies in the olecranon fossa, where it may be more difficult to see. The loose body should be removed, and the site of origin inspected and saucerised to prevent separation of further fragments.

**Traumatic Ulnar "Neuritis"** (see Chap. 61).

**Tennis Elbow.** The pathological basis of this common condition still remains obscure. The patient complains of pain over the lateral epicondyle, aggravated by use of the extensor muscles. A tender spot is found above or in front of the epicondyle, and extension of the elbow with firm flexion of the wrist produces pain over the head of the radius. Occasionally pain is more widespread in the extensor group of muscles. Aching pain radiates both up and down the arm and may be referred to the ulnar border of the hand. Occasionally a small cystic swelling may be found in the extensor origin. A similar condition occurs less commonly around the medial epicondyle, and then affects the use of the flexor muscles. The condition appears to be allied to degenerative tendinitis in other situations.



FIG. 572. Osteochondritis dissecans of the elbow. The bony flake with some calcification of the surrounding cartilage is well shown.

**Treatment.** The effectiveness of physiotherapeutic treatment is proportional to the length of time the condition has existed. If the complaint has been present for months, or there is a palpable thickening or visible swelling, physiotherapy is unlikely to succeed. Treatment consists of radiant heat, massage and exercises, perhaps supplemented by short-wave diathermy, or faradism to the extensor muscles. If this fails, local infiltration of the area with procaine should be tried. When this has abolished the local pain, the extensors should be stretched, by manipulation of the elbow and wrist. The injection may need to be repeated. Hydrocortisone (25 mg. in 2 ml.) may be injected in place of procaine. If pain persists, a cure can usually be effected by division of the extensor origins from the epicondyle. This is a simple procedure which can be carried out through an incision only an inch long. When a small cyst or bursa exists it should be removed.

**Olecranon Bursitis.** This is often the result of chronic occupational irritation of the bursa, but acute inflammation may develop from infection on the hand or forearm. The condition is analogous to prepatellar bursitis and is treated similarly (Chap. 89).



## CHAPTER 87

### THE WRIST AND HAND

**Tumours of the Hand.** The majority of tumours of the hand belong to three main groups: (a) Ganglia, accounting for half the cases; (b) epidermoid cysts; and (c) benign synoviomias or giant-cell tumours of tendon sheaths. Other less common tumours are enchondromas and carcinomas of the skin; the latter may arise after excessive exposure to sunlight, burns or other injuries. Hæmangiomas, glomangiomas, melanomas, fibromas, and lipomas are rare. Sebaceous cysts are only found on the dorsum of the hand and fingers, for there are no hair follicles in the volar skin.

**Ganglia.** These are usually a single cyst but may be surrounded by smaller ones. Their origin is uncertain, but their walls are derived from fibrous tissue, and they contain spheroidal cells which by secretion produce the translucent mucoid contents of the cysts. Recurrence after excision is rare in the hand, but common in the foot, and invariable in the case of similar cysts of the cartilages of the knee which are only locally removed (Chap. 89). Ganglia of the wrist and hand occur in two common situations: (a) Around the wrist, chiefly on the dorsum of the capitate and navicular bones; (b) on the flexor aspect of the fingers, where they are small and tense and often diagnosed as "sesamoids." They may also occur on the dorsum of the fingers in relation to the interphalangeal joints. Patients complain of a smooth, usually painless swelling which, however, as it becomes larger, may cause dull aching after use of the part. The swelling is rounded and fluctuant if not too tense. It is not adherent to the skin, but adherent to deeper structures. Its size may vary, and, if in the carpal region, may become tense with over use of the wrist.

As a ganglion sometimes disappears, immediate surgery is not indicated if it is small or of recent origin, but if a ganglion has been present for four to six months it is unlikely that it will subside spontaneously; it should therefore be removed, particularly if it is unsightly or interferes with function. The operation, though simple, should be carried out under good conditions through a transverse incision. A sphygmomanometer cuff is used as a tourniquet. It is important that all the cyst tissue and any prolongation of the cyst should be removed if recurrence is to be prevented. A few recur, but they are usually finally eradicated by a second operation. Methods of temporary relief consist of puncture with a tenotome, rupture (traditionally by a blow with the family Bible), or aspiration, but when the rent is sealed the cyst distends again. Injection of sclerosing fluids is often patchy in effect, and so recurrence is common.

**Epidermoid Cysts.** These occur most commonly on the volar aspect of the fingers, for they are produced by traumatic implantation of the growing basal layer of the dermis into deeper tissues. They are painless, though they may render the skin over them sensitive to pressure. They are spherical or oval, and always appear very white when the skin is stretched over them (Fig. 573). They are composed of very white fibrous tissue with excessive keratin in its deep layers. The cysts contain material

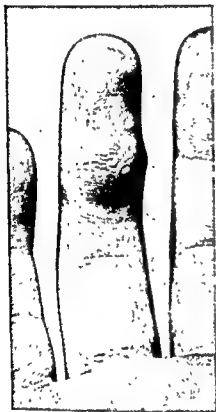


FIG. 573. An epidermoid cyst of the ring finger.



FIG. 574. A giant-cell tumour (fibrous synovioma) of the middle finger.

composed of a large proportion of cholesterol. Excision must be complete to avoid recurrence.

**Benign Synovioma** (*Giant-cell Tumour of the Tendon Sheaths*). This tumour occurs on the dorsal surface of the fingers in the neighbourhood of the joints, or on the volar surface attached to a tendon sheath. It is a lobulated, firm or rubbery tumour, and may cross a finger beneath a tendon (Fig. 574). It varies greatly in its rate of growth. The variety of cells seen microscopically accounts for the many colours of the cut surface of the tumour. There are fibroblasts, spheroidal cells, synovial clefts, giant cells, foam cells, and sometimes hæmosiderosis is present. The tumour is innocent and therefore distinct from the malignant synovioma. However, it should be carefully dissected out, for otherwise recurrences are common,

## CHAPTER 87

### THE WRIST AND HAND

**Tumours of the Hand.** The majority of tumours of the hand belong to three main groups: (a) Ganglia, accounting for half the cases; (b) epidermoid cysts; and (c) benign synoviomas or giant-cell tumours of tendon sheaths. Other less common tumours are enchondromas and carcinomas of the skin; the latter may arise after excessive exposure to sunlight, burns or other injuries. Haemangiomas, glomangiomas, melanomas, fibromas, and lipomas are rare. Sebaceous cysts are only found on the dorsum of the hand and fingers, for there are no hair follicles in the volar skin.

**Ganglia.** These are usually a single cyst but may be surrounded by smaller ones. Their origin is uncertain, but their walls are derived from fibrous tissue, and they contain spheroidal cells which by secretion produce the translucent mucoid contents of the cysts. Recurrence after excision is rare in the hand, but common in the foot, and invariable in the case of similar cysts of the cartilages of the knee which are only locally removed (Chap. 89). Ganglia of the wrist and hand occur in two common situations: (a) Around the wrist, chiefly on the dorsum of the capitate and navicular bones; (b) on the flexor aspect of the fingers, where they are small and tense and often diagnosed as "sesamoids." They may also occur on the dorsum of the fingers in relation to the interphalangeal joints. Patients complain of a smooth, usually painless swelling which, however, as it becomes larger, may cause dull aching after use of the part. The swelling is rounded and fluctuant if not too tense. It is not adherent to the skin, but adherent to deeper structures. Its size may vary, and, if in the carpal region, may become tense with over use of the wrist.

As a ganglion sometimes disappears, immediate surgery is not indicated if it is small or of recent origin, but if a ganglion has been present for four to six months it is unlikely that it will subside spontaneously; it should therefore be removed, particularly if it is unsightly or interferes with function. The operation, though simple, should be carried out under good conditions through a transverse incision. A sphygmomanometer cuff is used as a tourniquet. It is important that all the cyst tissue and any prolongation of the cyst should be removed if recurrence is to be prevented. A few recur, but they are usually finally eradicated by a second operation. Methods of temporary relief consist of puncture with a tenotome, rupture (traditionally by a blow with the family Bible), or aspiration, but when the rent is sealed the cyst distends again. Injection of sclerosing fluids is often patchy in effect, and so recurrence is common.

This is now rarely seen. Chronic syphilitic tenosynovitis is also recorded.

Apart from specific treatment of the cause (*vide supra*), local treatment has also to be considered. The value of surgery is dependent on the amount of granulomatous tissue and the number of loose bodies formed. Surgical removal of both may be well worth while if they are excessive. Reliance must otherwise be placed on rest and physiotherapy.

**Trigger Finger and Thumb.** This interesting and easily recognised condition is due to a thickening in the tendon or the paratendinous tissue at the mouth of the digital sheath. At first some local pain suggestive of chronic tenosynovitis may be noted; then, as the mass gets too large to leave the mouth of the tunnel, there is difficulty in flexing the digit, and when it slips beyond the sheath there is difficulty in extending the digit. It is the sudden escape of the "tumour" into the mouth of the tunnel that causes the sharp flick of release so characteristic of the condition. There is always tenderness over the mouth of the digital tunnel, and the small mass may be felt to move with the flexor tendons. Fixed flexion of the thumb from this cause may be found in quite small babies, and due to the fatness of the hand it may be overlooked for some time, even by an observant mother. Treatment in all cases is simple and effective, and consists of a small incision in the line of the tendon and dividing and enlarging the mouth of the digital sheath, to allow freedom of movement to the thickened tendon. The mass, though not removed, subsides once the cause of irritation is removed. The condition usually comes on spontaneously, but sometimes it is caused by local injury which may be single or repeated, and may be occupational.

**Rupture of Tendons.** Tendons may rupture as the result of direct injury with a blunt or sharp object, or as the result of strain after partial division or degeneration. The long extensor of the thumb may rupture by a degenerative process, probably associated with injury to its vascular supply. This is the mechanism of late rupture after a Colles' fracture, but the condition may occur without a history of injury. The most common rupture is that of an extensor tendon of a terminal phalanx, following a blow on the tip of the finger (mallet finger). Two varieties are encountered: (a) Fracture of the bony insertion of the tendon, often accompanied by subluxation of the joint; (b) rupture of the tendon itself, distal to the joint line. The amount of drop of the terminal phalanx varies. The usual treatment of immobilisation in plaster with the finger in extension at the terminal interphalangeal joint and flexion at the proximal joint, only produces a decrease in the drop, but seldom a complete cure. This may be reasonable enough in the ring and little fingers, but better results may be demanded in the middle and index fingers, especially in skilled manual workers. A better result may be obtained by immediate suture of the tendon, though, owing to fraying of the ends, this may be difficult. When division is due to a clean section by a sharp object, the prognosis is much better. When the bone is fractured the fragment attached to the tendon should be

although they are usually completely eradicated at a second operation.

**Tenosynovitis around the Wrist.** Acute infective tenosynovitis in this region is rare, except as a spread of infection from the digital sheaths. Chronic tenosynovitis is, however, common, and three varieties are distinguishable: (a) The common sheath of the extensors may become distended unilaterally or bilaterally. The swelling may be confused with a ganglion, but is less tense, and when pressed, the fluid can be seen to run into the prolongations distally around each separate tendon. (b) *de Quervain's stenosing tenosynovitis* is confined to the sheath of the abductor pollicis longus in its tunnel along the radial styloid process (Fig. 575). The tendon sheath is distended with fluid, and is also thickened and granulomatous. A firm and, at times, almost cartilaginous swelling can be seen and felt over the radial styloid. Abduction of the thumb is painful, and pressure over the swelling increases the pain, or gives a sensation of crepitus.



FIG. 575. Sclerosing tenosynovitis (tenosynovitis stenosa or de Quervain's disease). The thickening of the tendon tunnel for the abductor pollicis longus where it crosses the radial styloid is well shown.

Though it may be relieved by rest in plaster, the quickest and most effective treatment is to incise the thickened tendon sheath and remove all the granulomatous synovial tissue and sheath necessary to free the tendon. (c) Tenosynovitis of the myotendinous junction of the abductor pollicis longus and the extensor pollicis longus. The pain is proximal to the wrist; the myotendinous junctions are tender, and crepitus may be

palpated. This is more likely to be relieved by rest in plaster, and usually subsides fairly quickly. Short-wave diathermy may help the more resistant cases.

**Tenosynovitis in the Hand.** Spread of acute infection from a septic focus in a digit, or from a wound of a digital tendon sheath into the radial or ulnar bursa is not the province of this section (see Chap. 106). Attention is, however, drawn to a more benign form of tendon sheath infection which may follow treatment of early acute tenosynovitis with antibiotics, for it may later be confused with the more chronic forms of tenosynovitis.

Chronic tenosynovitis of digital sheaths may be due to occupational causes, e.g. the prolonged use of heavy shears may inflame the digital sheath of the little finger. This cause is indicated by the development of appropriately placed callosities. Rheumatic tenosynovitis may occur at any stage of rheumatoid arthritis. Tuberculous tenosynovitis may involve the ulnar bursa with the formation of a fluctuant mass passing under the transverse carpal tunnel—the "compound palmar ganglion."

preserved and pain relieved in many cases by excision of the os multangulum majus, and the consequent formation of a fibrous false joint.

**Keinböck's Disease.** This is a degenerative condition of the lunate bone. Its origin remains obscure, though it is most likely due to partial avascular necrosis of the bone following a fissure fracture, for similar changes may follow known injuries. When the disease develops insidiously there may be an occupational cause, e.g. the use of compressed air percussion tools, or it may follow a minor injury. Cystic changes, patchy sclerosis, and compression of the bone occur (Fig. 577), followed by osteo-arthritis in the wrist. A similar condition, *Preiser's disease*, may affect the carpal navicular bone. *Treatment* depends on the stage of the disease. If the wrist is free from arthritis, excision of the lunate offers some amelioration, but if there is already arthritis of the radiocarpal joint, arthrodesis of the wrist is required.



FIG. 577. Sclerosis of the lunate following a fissure fracture—Kienböck's disease.

#### **Dupuytren's Contracture.**

This results from a proliferative thickening of the palmar fascia and its prolongations, so that the skin becomes wrinkled and firmly adherent to the fascia.

The prolongations of the fascia

into the fingers thicken and then flex the metacarpophalangeal and proximal interphalangeal joints (Fig. 578). This results in the affected finger being slowly drawn into the palm, and after a time secondary contracture of the affected joints occurs. The attachments of the palmar fascia to the metacarpals and the tendon sheaths are involved, and in severe cases the digital nerves may be inextricably involved in the fibrosis. If the *plantar* fascia is examined, nodules are found in it in 7 per cent. of patients with Dupuytren's contracture.

**Aetiology.** Localised contracture of the palmar fascia may follow injury especially if accompanied by infection. There is an hereditary factor, for the condition may occur through several generations. Occupations, such as the use of a bootmaker's awl, may, by irritating the fascia, cause contracture, although quite often the disease appears spontaneously in both hands of a sedentary worker. A most remarkable characteristic is the uneven distribution of the fibrosis, so that no two cases are truly alike. The ring finger is most commonly affected, and then the little finger, whereas the middle and index fingers are rarely involved. Men are more

excised if reduction is impossible, and the tendon then sewn to the terminal phalanx with braided steel wire. Rupture of the central slip of the extensor tendon to the middle phalanx produces the characteristic "buttonhole" injury in which the terminal phalanx is extended and the proximal interphalangeal joint flexed (Fig. 576). The middle slip of the tendon should be repaired, but this is often technically difficult, especially in late cases. Approximation of the two lateral slips to each other in the midline is usually possible and this improves the position and function.



FIG. 576. Deformity of the index finger following rupture of the middle slip of the extensor expansion.

**The Wrist Joint.** This consists of the lower radio-ulnar joint and the radio-carpal joint. Although both are commonly affected together, the radiocarpal joint may suffer alone. Subluxation of the radio-ulnar joint produces a prominent and tender lower end of ulna, with increased mobility. In severe cases, excision of a segment 1 inch long of the ulna above the joint to form a false joint may be of value. Most cases, however, are not very troublesome, and require at the most a wrist strap. The radiocarpal joint is commonly the seat of osteo-arthritis as a sequel to the following conditions: comminution of the radial articular surface, fractures of the navicular bone (scaphoid), especially if there is avascular necrosis, Kienbock's disease of the carpal lunate (*vide infra*), rheumatoid arthritis, or occupational stresses.

The joint is not a satisfactory one to treat because it is in constant use. Physiotherapy may temporarily relieve

mild cases. Immobilisation of the wrist in plaster for a few weeks is often of value, and is essential in acute cases. Advanced osteo-arthritis requires radiocarpal arthrodesis, which gives a strong and pain-free wrist, but limits mobility.

*Rheumatoid arthritis* commonly attacks this joint, and one of its effects may be synostosis of the carpal bones. This should not be confused with the rare congenital anomalous fusions. *Tuberculous* and *infective arthritis* of the joint are uncommon, and the physical signs and radiological changes are similar to those of infection of other joints with the same organisms.

The Metacarpocarpal joint of the thumb is often the site of a painful osteo-arthritis, as the result of injury, occupation, or rheumatoid arthritis. Immobilisation of the joint is a handicap; movement may, however, be

## CHAPTER 88

### THE HIP

**Clinical Examination.** This is often regarded as complicated because of the large number of available tests, but these can be reduced to a simple system, and the information obtained should often enable a diagnosis to be made before radiography is carried out. If the patient can stand, inspection of the joint is begun with the patient standing. As in the examination of the spine, the level of the pelvis is first checked by seeing that the anterior superior iliac spines are in the same horizontal line. A tilted pelvis may be due to shortening of one leg, adduction contracture, or very rarely fixation in abduction; the gait and stance are observed; then the tilted pelvis is levelled by placing blocks under the short leg, and some of the tests repeated—particularly the Trendelenberg test (*vide infra*). On walking, a stiff hip is swung from the lumbar spine. A fixed flexion deformity of the hip produces an increased lumbar lordosis, and there is a tendency for the patient to stand with the knee flexed. Movements of the lumbar spine may be conveniently tested at this stage to exclude pain referred to the buttock from a lumbar lesion. Alteration in the curve around the trochanter (producing a female silhouette) is noted in dislocation of the hip. In children, a rise in level of the homolateral gluteal fold and broadening of the perineum are noted in congenital dislocation of the hip.

**Trendelenberg's Test.** When a normal person stands on one leg, the pelvis is tilted upwards to bring the centre of gravity of the body over the hip joint being used. Any serious interference with joint function prevents this and the pelvis drops, i.e. tilts downwards, on attempting to balance on the affected hip (Fig. 579). Trendelenberg's test is nothing but a test of hip efficiency, which may be impaired by any one of a number of causes, e.g. paralysis of the abductors, dislocation, coxa vara, arthritis, or slipped epiphysis. When a patient with a positive Trendelenberg

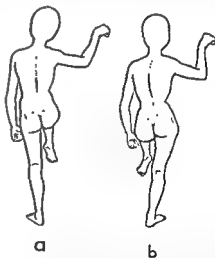


FIG. 579. Trendelenberg's test. (a) With the patient standing on the sound side the opposite buttock rises to bring the centre of gravity over the supporting hip joint. (b) With the patient standing on the unsound side, the pelvis drops on the opposite side. This is a test for efficiency of the hip joint and does not indicate any particular disease.



frequently affected than women, and old men more frequently than young men. The rate of development of the deformity is very variable; this fact, together with the age of the patient, should be taken into consideration in deciding treatment.

*Treatment.* In a mild slowly progressive contracture in an elderly man adequate function can often be maintained by subcutaneous division of the contracted bands with a tenotome or cataract knife, and the use of



FIG. 578. The classical appearance in Dupuytren's contracture. There is marked flexion of the fifth finger and developing contracture of the ring finger. Note in ulnar nerve palsy the metacarpophalangeal joints of the ring and little fingers are hyperextended.

night splinting. A very severely contracted little finger of long standing, with joint changes, may be beyond salvage, and is best amputated. The ideal procedure is early excision of the whole of the palmar fascia and the worst affected digital prolongations before much contracture has occurred. This, though tedious and requiring painstaking care, presents no real technical difficulty although adhesion of skin to the fascia and the contracture of the skin may result in skin necrosis after operation. This should be foreseen and Z incisions used in the digits, and Thiersch grafts in the palm, when the skin is devitalised. Operative success depends on careful dissection, planned skin incisions utilising skin creases, avoidance of hæmatomas and infection, and careful post-operative care.

**The Carpal Tunnel Syndrome (Median Nerve Compression).** See Chap. 61.

method, a tape is run obliquely across the abdomen and through the anterior superior iliac spines and the tip of the greater trochanter. Normally, the tape should lie above the umbilicus. In Bryant's triangulation, an imaginary line is dropped back from the anterior superior iliac spines and the distance of the great trochanter from it is measured.

The movements and position of the hip are now examined. Two main positions are adopted by the diseased hip: (a) Flexion with adduction, due to muscle spasm around a joint which has long been the seat of disease; (b) flexion with abduction when a hip joint is distended with



FIG. 581. In children, shortening of the femur is best estimated by flexing both hips and knees to 90 degrees and comparing the level of the patellæ.

fluid, or distorted by psoas spasm. The examination is carried out with the patient supine. First, fully flex the sound hip to flatten the lumbar spine. If there is any flexion contracture of the opposite hip, the limb rises off the couch to a degree equivalent to the contracture (this can be checked by turning the patient into the prone position and showing that hyperextension of the diseased hip is limited). Rotation of the hip can be gently carried out by rolling the relaxed leg under the palm of the hand. This method, by producing minimal movement of soft tissues around the joint, distinguishes painful intra-articular lesions from those outside the joint. Rotation may be more forcibly tested by flexing the hip and using the leg as a lever. Limitation of internal or external rotation may be found or rotation may be painful at its extreme limits. In babies and young children, abduction of the hips is tested by flexing both hips and separating the legs; when the hips are normal the knees

sign walks, there is a dipping gait on the affected side, and this becomes a waddle if the weakness is bilateral.

The hip is now carefully palpated with the patient lying down. In dislocation of the hip, elevation of the greater trochanter may be noted with relaxation of the fascia lata above it. Furthermore, loss of resistance may be felt anteriorly making it difficult to palpate the femoral artery. The head of the femur may be detected in the buttock, particularly if the muscles are wasted. On rotation of the leg with the flat of the hand, the centre of rotation at the pelvis may be felt to lie outside the hip joint. If the head of the femur is dislocated and unfixed, telescoping of the leg may be elicited.

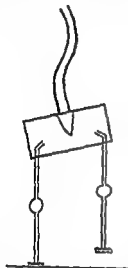


FIG. 580. Abduction of the hip joint causes apparent lengthening; conversely adduction produces apparent shortening. Unless care is taken to measure the two legs in a similar relationship to the transverse axis of the pelvis, false measurements may be made.

With the patient supine, the pelvis is now levelled by placing the legs in the appropriate position. This will show whether any pelvic obliquity is due to shortening of a leg, or to contracture of soft tissues around the hip. If there is contracture, the leg on the normal side must be placed in the same position as the abnormal leg, for comparative measurements to be accurate. The position of the leg alters the apparent length of the limb; thus adduction appears to shorten it and abduction to lengthen it (Fig. 580). However, when the above precautions have been taken, true shortening is estimated by comparing the measurement from the anterior superior iliac spine to the medial malleolus on the two sides. A combination of true and false shortening may be encountered. Shortening cannot be measured in a baby by this method; therefore the length of the femora

are compared with the buttocks flat on the couch and the hips and knees flexed to a right angle. Any shortening is shown by alteration in level of the patellæ (Fig. 581).

If a leg is found to be short, supplementary measurements are made to determine the site of the shortening. The length of the tibia may be checked by measuring from the inner aspect of the tibial plateau (joint surface) to the medial malleolus. If the tibiae are of equal length, any shortening of the limb is in the shaft or neck of the femur. To determine which of these two sites is involved, the relations of the greater trochanter to the pelvis are investigated. (Accurate determination of the trochanteric level may be impossible in fat patients.) In the simplest method, one tape is run across both anterior superior iliac spines and another across the tips of both greater trochanters. If there is no shortening of the head and neck of the femur, the tapes should be parallel. In Shoemaker's

method, a tape is run obliquely across the abdomen and through the anterior superior iliac spines and the tip of the greater trochanter. Normally, the tape should lie above the umbilicus. In Bryant's triangulation, an imaginary line is dropped back from the anterior superior iliac spines and the distance of the great trochanter from it is measured.

The movements and position of the hip are now examined. Two main positions are adopted by the diseased hip: (a) Flexion with adduction, due to muscle spasm around a joint which has long been the seat of disease; (b) flexion with abduction when a hip joint is distended with

FIG. 581. In children, shortening of the femur is best estimated by flexing both hips and knees to 90 degrees and comparing the level of the patellæ.



fluid, or distorted by psoas spasm. The examination is carried out with the patient supine. First, fully flex the sound hip to flatten the lumbar spine. If there is any flexion contracture of the opposite hip, the limb rises off the couch to a degree equivalent to the contracture (this can be checked by turning the patient into the prone position and showing that hyperextension of the diseased hip is limited). Rotation of the hip can be gently carried out by rolling the relaxed leg under the palm of the hand. This method, by producing minimal movement of soft tissues around the joint, distinguishes painful intra-articular lesions from those outside the joint. Rotation may be more forcibly tested by flexing the hip and using the leg as a lever. Limitation of internal or external rotation may be found or rotation may be painful at its extreme limits. In babies and young children, abduction of the hips is tested by flexing both hips and separating the legs; when the hips are normal the knees

nearly touch the couch. Abduction in adults is tested by holding the pelvis level and pulling the knee outwards as far as possible ; the degree of abduction is compared on the two sides. Adduction is tested by crossing one leg over the other ; the leg on the side being tested should normally cross the middle third of the opposite thigh. The patient is then turned into the prone position and extension tested by lifting the leg with the knee extended. This produces tension in the Y-shaped (ilio-femoral) ligament, and a rise in pressure in the joint. A normal range of painless hyperextension is strong evidence against the presence of any active disease of the hip joint.

**Diseases of the Hip.** These are closely related to age and may be grouped as follows :

<i>Infants</i>	<i>Children</i>	<i>Adults</i>
Congenital Dislocation. Acute Infective Arthritis.	Transient Synovitis. Perthé's Disease. Slipped Femoral Epiphysis (Adolescent Coxa Vara). Tuberculous.	Osteo-arthritis.

**Congenital Dislocation.** This arises from a developmental defect of the acetabulum, which permits the femoral head to escape from it at various stages in the growth of the child, depending on the degree of shallowness of the socket. It is an hereditary defect, and is common in certain parts of the world, notably in Brittany and around Bologna in Italy. It is seven times more common in females than males, and unilateral dislocation is more common than bilateral dislocation. The characteristic abnormalities in the bones are seen in plain X-rays, but the important changes in the soft tissues are better determined by arthrography. The younger the baby the less obvious is the acetabular defect. There is a failure of development of the roof of the acetabulum, and a secondary failure of development of the femoral head, proportionate to the degree of instability and the length of time the dislocation has existed. The epiphyseal centre for the head of the femur is smaller than normal, and the anteversion of the neck of the femur alters the appearance of the neck (Fig. 582). In long-standing cases, the size of the whole limb is reduced. If a line is drawn through the central acetabular cartilage of both hips horizontally, and lines are dropped at right angles to this from the outer lip of each acetabulum, four quadrants centered just above each femoral head are formed (Fig. 582). In the normal hip, the epiphyseal centre lies in the lower inner quadrant. In congenital dislocation it lies in one of the outer quadrants, often the one above the horizontal line.

**Clinical Features.** The condition may be suspected before the child begins to walk, because of perineal broadening in bilateral cases, and of changed levels of the gluteal folds in unilateral cases. Shortness of the leg may be noted by an observant mother, especially if there is a family history of the disorder. Walking is often delayed and a limp, which is

characteristic waddle in bilateral disease, may appear. When the dislocation is complete Trendelenberg's sign, real shortening of the leg, and telescopic movement are present, and the head of the femur may be palpated in an abnormal position.

*Treatment.* The aim is reduction of the dislocation and retention of the hip in the reduced position for long enough to stretch all soft tissue obstructions and to allow the acetabulum to develop. The ease with which this can be done is variable, and depends on the presence or absence of soft tissue obstruction in the form of an hour-glass contracture of the capsule or fibrous tissue in the acetabulum, and the length of time the

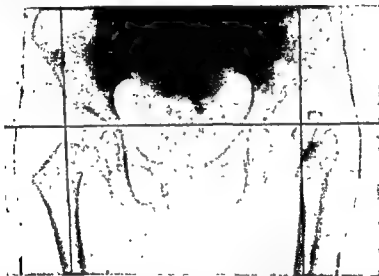


FIG. 582. Congenital dislocation of the left hip and the relationship of the head of the femur to Bruce's lines. On the right (reader's left) the epiphyseal centre can be seen to lie in the lower inner quadrant. On the dislocated side (reader's right) the epiphysis is reduced in size and lies in upper outer quadrant. The horizontal line is drawn through the cartilagenous centre of each acetabulum. A vertical line is dropped through the outer edge of the roof of each acetabulum at right angles to the horizontal line.

dislocation has existed. Up to the age of three years, manipulative reduction is usually possible, though it may have to be accomplished in stages, with preliminary stretching of the hip in increasing abduction. Sometimes reduction is difficult or unstable. Arthrography gives valuable information about the state of the soft tissues, and enables a decision to be made between persistence in conservative treatment and open operation. Between three and six years, the success of closed reduction is more problematical, and over six years deepening the acetabulum by an osteotomy above it and the insertion of a wedge, or some form of reconstruction has to be carried out. Often above this age a false acetabulum has already formed, and it is therefore unwise to interfere with it till osteo-arthritis develops.

Manipulative reduction is carried out, preceded, when necessary, by a period of traction to bring the head of the femur opposite the acetabulum. The head of the femur is lifted forward while the hip is flexed and widely abducted. The head of the femur is retained in the acetabulum in the most stable position by means of a plaster—usually in the “frog” position (Fig. 583). Reduction is confirmed by radiography and maintained by plaster, renewed as required for six to nine months. The degree of abduction is then slowly decreased and freedom of movement increased as each new plaster is applied until it is felt safe to leave the legs free. Although a satisfactory reduction may be achieved, and the radiological appearances may be good, only 75 per cent. of patients

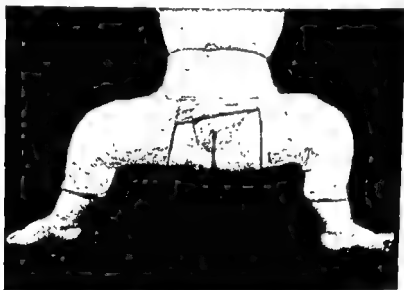


FIG. 583. A “frog plaster” for bilateral congenital dislocation of the hip joints.

obtain a good functional result, and many of these patients eventually develop osteo-arthritis. The treatment of the late unreduced congenital dislocation is difficult; sometimes a cup arthroplasty or alteration in the joint stresses by a Lorenz or McMurray osteotomy is indicated.

**Acute Infective Arthritis.** The infection of the joint probably often arises from bone close to the joint although direct infection of the hip does occur. The disease is secondary to sepsis elsewhere, e.g. tonsillitis, infective adenitis, impetigo, or distant osteomyelitis. Infection of bone or soft tissues may occur near the hip, without the joint being involved, but the pain and irritation may make a differential diagnosis difficult in the early stages.

The clinical features are those of a severe infection (high temperature and leucocytosis) and extreme pain on the slightest movement of the hip, which is flexed and abducted. Extra-articular abscess formation is common and may require incision. An abscess may point in the buttock.

Rest and antibiotics are the fundamentals of treatment. Rest may be secured by simple skin traction on the leg. Penicillin is usually tried first, and is initially given in massive doses. Aspiration of the joint and instillation of penicillin may be needed. Frank pus in the joint means severe destruction of the cartilage, and the inevitable development of osteo-arthritis. In babies, the whole epiphyseal centre for the head may be lost. A long period of fixation of the hip may occasionally result in fusion, but generally the result is an unstable arthritic hip demanding further surgery in the chronic stage.

**Transient Synovitis.** This is of comparatively frequent occurrence, and causes alarm because the symptoms of early tuberculosis of the hip are similar. The correct diagnosis can only be established by a period of rest and observation, which are essential to avoid mistakes. A history of injury is often given though it is probably seldom relevant. The child limps from muscle spasm around the hip; movements of the hip are painful, but often only in extreme positions; the hip is held flexed. The child should be put to bed with skin traction on the leg, and the temperature, E.S.R. and the white cell count noted. Radiological examination shows a normal hip. Recovery in a few days to a fortnight is the rule, without any after-effects.

**Perthés' Disease.** This is one of the best examples of the growth disturbance of an epiphysis, *osteochondritis juvenilis*, which may be met with in other epiphyseal centres, e.g. Schlatter's disease of the tibial tubercle, Köhler's disease of the tarsal navicular, Calvé's disease of the spine and Sever's disease of the calcaneal epiphysis, in all of which the sequence of changes is similar. The basic cause of the condition is unknown, though there is obviously considerable disturbance of the vascular supply to the epiphysis, but whether this is primary or secondary is speculative (Fig. 584). In osteochondritis of other epiphyses, the sequence of changes to be described occurs, but they are not so marked, and they are often of shorter duration.

The disease is most common in boys, between the ages of four to ten



FIG. 584. Bilateral Perthés' disease. The changes in both hips are advanced. The age of onset in this case was three years.



Manipulative reduction is carried out, preceded, when necessary, by a period of traction to bring the head of the femur opposite the acetabulum. The head of the femur is lifted forward while the hip is flexed and widely abducted. The head of the femur is retained in the acetabulum in the most stable position by means of a plaster—usually in the "frog" position (Fig. 583). Reduction is confirmed by radiography and maintained by plaster, renewed as required for six to nine months. The degree of abduction is then slowly decreased and freedom of movement increased as each new plaster is applied until it is felt safe to leave the legs free. Although a satisfactory reduction may be achieved, and the radiological appearances may be good, only 75 per cent. of patients

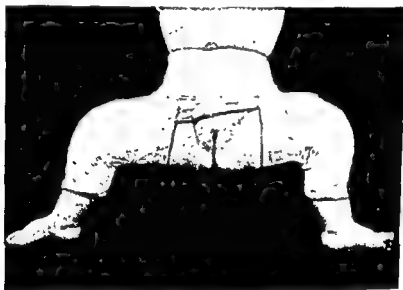


FIG. 583. A "frog plaster" for bilateral congenital dislocation of the hip joints.

obtain a good functional result, and many of these patients eventually develop osteo-arthritis. The treatment of the late unreduced congenital dislocation is difficult; sometimes a cup arthroplasty or alteration in the joint stresses by a Lorenz or McMurray osteotomy is indicated.

**Acute Infective Arthritis.** The infection of the joint probably often arises from bone close to the joint although direct infection of the hip does occur. The disease is secondary to sepsis elsewhere, e.g. tonsillitis, infective adenitis, impetigo, or distant osteomyelitis. Infection of bone or soft tissues may occur near the hip, without the joint being involved, but the pain and irritation may make a differential diagnosis difficult in the early stages.

The clinical features are those of a severe infection (high temperature and leucocytosis) and extreme pain on the slightest movement of the hip, which is flexed and abducted. Extra-articular abscess formation is common and may require incision. An abscess may point in the buttock.

are added varying degrees of slipping of the epiphysis. The head of the femur, as in fractures of the femoral neck in the so-called abducted position, slips downwards and backwards; thus when slipping is severe anteroposterior radiographs give a view looking directly through the base of the hemispherical femoral epiphysis. However, the deformity is best seen in the lateral radiographs, which must never be omitted in diagnosis or treatment, for differing degrees of internal rotation of the femur may disguise the degree of displacement in anteroposterior films.

The disease is most common between the ages of twelve and fifteen years, and slightly more common in boys than in girls. It may be unilateral or bilateral, and if one hip has slipped watch must be kept on the opposite side. There is often an associated endocrine disturbance, giving the child a fat and infantile appearance. No certain cause is known; examination of tissue removed from the epiphyseal line sheds no light on the condition. Trauma may play one of the following roles: (1) Severe trauma may separate a normal epiphysis (rare). (2) An epiphysis the site of disease may be susceptible to minor trauma which may precipitate the symptoms complained of without actually being responsible for displacing the epiphysis. (3) An epiphysis the site of the disease may be considerably displaced by trauma. At times it may be difficult to distinguish a gradual slip with severe deformity from a recent acute slip. A decision may be reached in such cases following a preliminary period of traction in moderate abduction. An acutely displaced epiphysis is at least partly restored to its normal position by this means whereas an epiphysis which has slipped slowly is fixed by adhesions and does not change its position.

*Treatment* is governed by two important considerations: (a) The need to restore and retain the epiphysis in its normal position; (b) the need to preserve the precarious blood supply to the epiphysis, dependent on its unstretched posterior attachments. Manipulation cannot be permitted, as it may damage the blood supply and produce avascular necrosis of the epiphysis. In the pre-slipping stage or when the epiphysis has only slightly slipped, further displacement should be prevented by insertion of a Smith-Petersen pin. A pin with sharp fins should be used and care taken that it does not carry the head away from the diaphysis.

When the epiphysis has slipped so that its position cannot be accepted it must be replaced. In the rare acute cases this may be accomplished by skeletal traction applied by means of a pin in the tibial tuberosity; the hip is maintained in flexion and adduction. If reduction is satisfactory, the femoral head may be pinned in this position. When traction fails to reduce the displacement, open operation is necessary. The anterior approach of the Smith-Petersen type, between the tensor fascia lata and the sartorius, is used. It is deepened and the iliacus retracted. The hip joint is opened, and the femoral neck exposed. A T-shaped incision is now made in the fibrous and periosteal tissue over the neck. The

years, but it may be encountered from three to sixteen years. It is usually unilateral, but may be bilateral. The symptoms are pain and limping. Clinical examination in an early case may be almost negative, except for some limitation of abduction. The disease sorts itself into three stages, with corresponding radiological changes.

(1) <i>Stage of Onset.</i> Pain is variable, and may be slight or intermittent; limping; and limited movements.	<i>Radiographic Changes.</i> Altered epiphyseal shape, increased joint space, and increased epiphyseal density.
(2) <i>Stage of Activity.</i> The manifestations may remain the same as above. Restriction of activity may be minimal; thus the child may only complain after a long day. This stage lasts for up to two years.	Irregular condensations in epiphysis. Patchy rarefaction, with cavities. Fragmentation of the epiphysis. Flattening and thinning of the epiphysis. Broadening of the femoral neck (Fig. 584).
(3) <i>Stage of Recovery.</i> Little interference with hip function, but on examination restriction of hip movement, particularly abduction.	Slow reconstitution of the trabecular structure of the epiphysis. Restitution of even and uniform density of the bone. Permanent alteration in epiphyseal shape.

**Treatment.** There is no way of influencing the course of the disease, which burns itself out in two to four years. It is, however, important that the hip should be freed from weight-bearing during the active stage of the disease. In bilateral cases there is no alternative but bed rest; skin traction or a frame may be needed to keep the child free from pain. In unilateral cases activity may be permitted, but the hip should be freed from weight-bearing by a weight-relieving caliper and a patten on the opposite shoe, or by the simpler method of the patient carrying the foot on the affected side in a shoulder sling and using crutches. The hip joint is rested thus till the epiphysis has fully recovered its normal density. The disease may be mild or severe, and the final alteration in the shape of the epiphysis is related to this and not to any particular treatment. Mild cases end with an almost normal hip, whereas severe ones have such a deformity of the femoral head that the early onset of osteo-arthritis is inevitable.

**Slipped Femoral Epiphysis (Adolescent Coxa Vara).** Decrease in the angle between the femoral shaft and neck, normally 130 degrees, is known as *coxa vara*. It may result from fracture of the femoral neck or trochanter, diseases which soften the bone, such as rickets or osteomalacia, or it may be a congenital deformity—sometimes bilateral.

Adolescent coxa vara is due to slipping of the femoral epiphysis secondary to changes on the metaphyseal side of epiphyseal plate, seen radiologically as rarefaction with occasional loculi and patches of dense bone; there is also widening of the epiphyseal line. To these appearances

Gonorrhoeal arthritis may be met with in varying degrees of severity. A juxta-articular lesion such as a Brodie's abscess or a tumour may cause difficulty in diagnosis. Syphilis is now a rare cause of difficulty; it may take the form of an epiphysitis in the congenital disease, or a gumma in the acquired disease. Charcot's disease may also be met with in the hip. Children are prone to develop limps from minor causes, which may persist for some time, usually to excite interest. Such limps tend to be peculiar, variable, and accompanied by voluntary resistance to examination.

**Treatment.** The general principles are the same as those for the treatment of tuberculosis of the spine (Chap. 84). Tuberculous abscesses of the hip are also treated by aspiration. Fixation of the joint in a child is maintained by a double abduction frame, and in the adult probably by a hip spica. When the patient's general condition has been restored, as judged by the temperature, E.S.R., weight, and general appearance, a decision must be made, with the object of shortening treatment, as to whether a movable or fixed joint is to be the aim. In adults, the decision is simple, for nothing less than a soundly arthrodesed hip is safe. If bony ankylosis is unlikely to occur, extra-articular fixation (which avoids disturbance of the infected area) is the best procedure. The fixation may be iliofemoral, i.e. from the greater trochanter to the ilium, or, better, ischiofemoral by inserting a tibial graft through a subtrochanteric osteotomy into the ischium. The latter method, perhaps by isolating the femoral head and neck from all movement for a time, results in frequent fusion of the hip. If a mobile joint is the aim, immobilisation in the frame or plaster is changed to simple leg-traction and eventually gentle movements of the joint are begun. Weight-bearing is permitted only when there is radiological evidence of restoration of the hip joint. Crutches, and a patten on the sound leg may be used temporarily while waiting for proof of cure.

**Osteo-arthritis.** This may arise as part of generalised osteo-arthritis or it may be secondary to hip disease or injury. Sometimes osteo-arthritis affects both hips without involving other joints (*malum coxae senilis*). The distribution of the pathological changes varies with the primary cause, but there is usually a combination of erosion of cartilage, eburnation of bone, cystic changes under the joint surfaces, and osteophytes at the margins of the joint (Fig. 585). Eventually, erosion of the head of the femur and the acetabulum may cause subluxation of the joint.

The primary causes of the degeneration of the joint are as follows:

- (1) *Trauma*.—Dislocation of the hip. Fracture of the femoral neck, often with partial avascular necrosis. Malunion of a fracture of the shaft of the femur or trochanter. Fracture of the acetabular floor.
- (2) *Congenital dislocation of the hip*.
- (3) *Perthés' Disease*.—The head is flattened.
- (4) *Slipped Femoral Epiphysis*.—Rotation of the head and broadening of the neck.
- (5) *Infection*.—Old healed tuberculosis. Rheumatoid arthritis,

epiphysis is replaced by nibbling away a triangular wedge of bone on the raw surface of the neck, for it would be dangerous to the remaining blood supply of the epiphysis to lever it back in position. When enough bone has been removed, the epiphysis is transfixed with a small Steinmann's pin and restored to alignment with the neck. It is then fixed in this position with a Smith-Petersen pin, by the usual technique (Chap. 100). The wound is then closed. In spite of every care, the blood supply to the epiphysis is sometimes damaged, and avascular necrosis of the femoral head occurs, later to be followed by osteo-arthritis. Otherwise the outlook for the hip joint depends on the accuracy of reduction.

**Tuberculous Arthritis.** This is a disease of the first ten years of life, during which time 90 per cent. of the cases occur. It is secondary to a focus elsewhere in the body—usually the hilar glands—and the hip joint is involved due to spread of infection from a focus in the acetabulum or neck of the femur. The progress of the disease in the joint may be divided into the stage of synovial infection, the stage of cartilaginous destruction and the stage of bone destruction. If the disease can be cured in the synovial stage, movements may be retained. If the cartilage is destroyed, there will be at least a fibrous ankylosis. If bone is involved, then bony ankylosis will probably occur; moreover, a firm bony ankylosis is the aim, as the hip would otherwise be painful, and, furthermore, there is a grave risk of recurrence of infection in an unfused hip. The outlook is more serious in adults, in whom firm bony ankylosis is the only satisfactory result.

The onset of the disease is insidious; a limp develops, but at first it is obvious only when the child is tired. Night starts occur. The child's general condition is poor, and there may be enlargement of the inguinal glands. As destruction of the joint progresses, there is increased flexion deformity of the hip, wasting of muscles, and muscle spasm. Real shortening and extra-articular abscess formation are signs of long-existing disease. Abscesses occur in half the cases.

Suspicious signs are an indication for admission to hospital for observation. Early radiography may be indefinite. A narrow joint space or a small erosion near the joint may be seen. The bones around the joint become osteoporotic. If an immediate diagnosis is impossible radiographs should be repeated periodically while the child is kept at rest on an abduction frame. An X-ray of the lungs is essential, for a primary focus there would favour the diagnosis and it might need treatment. The temperature and E.S.R. are observed. Biopsy of enlarged inguinal glands may give evidence of tuberculosis. A negative Mantoux test usually excludes tuberculosis.

**Differential Diagnosis.** This includes conditions already mentioned, Perthe's disease, slipped femoral epiphysis, and transient synovitis of the hip. Subacute non-tuberculous arthritis occurs in the hip, but it may prove impossible to establish the nature of the infecting organism.

osteotomy (McMurray) and slipping the upper end of the femoral shaft nearer the mid-line (Fig. 586). Decreased pain, but little increase in movement is to be expected, but the hip remains stable. *Arthrodesis* of the joint is the best procedure in young patients. A strong stable painless limb is ensured, enabling the patient to work. A sound knee and lumbar spine are necessary prerequisites. The hip joint is difficult to fuse. The arthrodesis may be an intra-articular one, in which the joint is cleared of cartilage and debris, and then fixed by transfixion with a long Smith-Petersen pin, or an extra-articular one of which the ischiofemoral type is the better.

*Arthroplasty* is the most uncertain of the methods used, but has advantages which justify its continued use. If there is a stiff lumbar spine, or the disease is bilateral, it is the only choice. The rheumatoid hip, the hip with prolapsus acetabuli, and the hip with marked cystic changes do badly when attempts are made to reform the hip joint. These types of osteo-arthritis are better treated by an excision arthroplasty, in which the head and neck of the femur are removed, together with the overhanging lip of the acetabulum. A false fibrous joint forms between the remaining surfaces, and although a little unstable, is often surprisingly painless. The procedure may be combined with an angular osteotomy of the upper end of the femur to give a broader support to the pelvis.

Two main forms of reconstruction arthroplasty are practised. In the first (Smith-



FIG. 586. A successful end result of a McMurray trachea-osteotomy for osteo-arthritis of the hip joint.



FIG. 587. Cup arthroplasty of the hip (Smith Petersen)

either in the active state or when the disease is burnt out. Old acute infective arthritis of childhood. (6) *General diseases* affecting the hip. Paget's disease. Spondylitis ankylopoetica. Osteitis fibrosa cystica. (7) *Local disease* near the hip. Osteomyelitis. Brodie's abscess. (8) *Malum Coxæ Senilis*.—Both hips affected, but other joints not involved. May be accompanied by prolapse of the acetabular floor. (9) *Neurotrophic Joint*.—Charcot's disease.

Difficulties in treatment are apt to arise from the bilateral nature of the



FIG. 585. Osteo-arthritis of the hip, showing a narrow irregular joint space and marginal osteophytes.

disease, the additional strains thrown on the spine and the knee which may therefore themselves be similarly affected, muscular atrophy after long disuse, and excessive weight due to lack of exercise and endocrine disturbances.

*Symptoms and Signs.* Pain after use is the characteristic symptom. The joint stiffens after rest, and is thus stiff on getting up in the morning. Pain bears no relation to the anatomical deformity. A gross deformity is consistent with little pain, and vice versa. A limp eventually develops due to an adduction or flexion deformity

of the hip, with real or apparent shortening. Pain is most often referred to the thigh and knee, and this may result in the knee being blamed erroneously for the pain, especially if it also is the site of arthritic changes. As the disease advances there are short remissions of pain, and exacerbations produced by minor injury or overuse.

*General treatment* consists of raising the standard of the patient's general health, by dieting, fresh air and exercises. The weight should be reduced. Aspirin derivatives may be used to reduce pain. Butazolidin (200 mg. t.d.s. reduced in a month to 100 mg. t.d.s.) is useful in generalised osteo-arthritis, and may be of value in relieving pain in localised osteo-arthritis. *Operative Treatment*.—No generally successful operation has been devised. The chief difficulty is pain, which tends to persist despite apparently good functional results. Joint denervation alone has proved of little value, although it is one of the effects of capsulectomy carried out during some of the procedures mentioned below. Local resection of osteophytes (*cheilotomy*) may occasionally be of benefit in increasing the range of movement and diminishing pain. A relatively successful procedure is to alter the stresses on the diseased joint by a subtrochanteric

## CHAPTER 89

### THE KNEE

**Clinical Examination.** Much may be learnt by inspection of the knee and the muscles acting on it. The efficiency of the knee joint is reflected in the condition of the quadriceps, and there is no better guide to the degree of disturbance of joint function than the amount of wasting and hypotonia of this muscle group. Conversely paralysis of the quadriceps leads to secondary changes in the joint. A knee joint which is distended with fluid shows an obvious filling of the supratellar pouch. The patella tap can be elicited if there is visible fluid, but a more sensitive test for small quantities of fluid is the stroking test. If the hand is rubbed firmly up the inner surface of the knee and then down the outer surface, a fluid wave can be seen to run from under the patella and distend the medial part of the quadriceps expansion.

If the patient can relax the muscles around the joint, and the flexed knee is then allowed to fall back into extension with the leg supported by the examining hands, a "chunky" sensation is felt as cartilaginous contact stops movement. If there is any obstruction to full extension, this is appreciated by the examining hand as an altered feeling at the end of extension, or pain or muscle spasm is produced. Greater degrees of limitation of extension are clearly visible.

The collateral ligaments of the knee joint are easily stretched by forced abduction or adduction of the knee when the joint is extended. The cruciate ligaments are tested with the knee flexed to a right angle and the foot steadied by the examiner sitting on it; the examiner grasps the upper end of the tibia and draws it backwards and forwards under the femoral condyles. The anterior cruciate ligament resists forward movement, and the posterior cruciate ligament backward movement.

The range of active and passive movements of the joint is assessed. Palpation of the joint line is important in injuries to the cartilages and marginal arthritis. It leads backward from the triangular depression on either side of the patellar ligament. Tenderness over the posterior horn of the meniscus may be the only sign of meniscus injury. This part of the cartilage may be further tested by *McMurray's test* (Fig. 589). With the knee and the hip almost fully flexed and the foot firmly gripped in the examining hand, the posterior horn of the cartilage is ground between the femoral condyle and the posterior margin of the tibial plateau by internal and external rotation of the foot and firmer flexion of the knee. In normal knees, this elicits a painless click. In abnormal knees, the click may be absent or modified; it may be painful, or provoke muscle



Petersen), the femoral head and acetabulum are reshaped into a ball and socket and a vitallium or stainless steel cup is inserted between the two



FIG. 588. The metal Judet prosthesis for arthroplasty of the hip.

surfaces to prevent cross union (Fig. 587). Although a good range of movement and a stable joint usually results, pain may persist. In the second type of reconstruction (Judet), the whole head and part of the neck is excised and replaced with a steel prosthesis (Fig. 588). The prosthesis tends to loosen at its junction with the femoral neck, and in spite of many devices, this essential weakness has not been completely overcome. The weakness inherent in the plastic type of prosthesis is avoided by using vitallium or stainless steel. The immediate post-operative results are excellent; there is a rapid return of function. The procedure is of value in elderly patients who are not

likely to put much strain on the hip, and in patients in whom the femoral head is lost or diseased, such as in avascular necrosis after fracture of the femoral neck. It is not to be lightly recommended for young people for it seriously interferes with any further operation on the hip; cup arthroplasty is the better procedure for these patients.

of cartilage cells in synovial chondromatosis, or degenerative arthritis. *Bony*.—Fragments detached by acute trauma (osteocartilaginous). Osteophytic new bone formation in chronic osteo-arthritis. Bone formation in pedunculated cartilaginous tumours. *Meniscal*.—Fragments of torn cartilage : (a) Free (rare) ; (b) pedunculated.

True new bone formation in a loose body means that it must have a blood supply or that it *had* a blood supply. Bone does not form without access to the blood stream. Cartilaginous and fibrous tissue, however, can grow in the joint fluid without a blood supply. A cartilaginous loose body may become adherent and develop a blood supply and then bone may form in it.

**Injuries to the Menisci.** These occur most commonly when the knee is flexed and under strain. A footballer making a sharp turn with his foot fixed in mud may suffer this injury. Collisions with other players are more likely to damage the collateral ligaments of the knee, and meniscus injuries, though they may be associated with this injury, are more commonly associated with injury to the cruciate ligaments. Coal mining is another source of injured menisci. The severe pain and the displacement of the torn part of the cartilage prevent full extension of the knee ; the patient therefore limps on the flexed knee, or may not be able to get about without assistance. Within twelve hours there is considerable swelling of the joint. The knee may gradually straighten, but if the cartilage is caught between the femoral condyle and the tibial plateau, interference with full extension may continue until relieved by manipulation or open operation.

The degree of joint disturbance and its characteristics depend on the degree of tearing of the cartilage, and the amount of freedom left to the torn portion. The lesser injury becomes greater with each successive incident. Cartilage injuries may be classified in the following sequence : (a) Small tear or detachment of the posterior horn. Minimal joint disturbance and healing with no further incidents. (b) Small posterior horn split. Minimal immediate disturbance, further increase in length of split with acute symptoms after perhaps a relatively trivial injury. (c) A  $\frac{3}{4}$ -inch tear. This permits a section of the cartilage to pass to the inner side of the femoral condyle, and the binding down of the ends produces a firm lock with little movement in either direction. Manipulation is often required to reduce it. Careless manipulation may partially free the joint by extending the tear further. (d) A longer tear. This may produce recurrent incidents which the patient learns how to reduce by manipulation. (e) The complete split or the " bucket handle " tear. The torn part may find accommodation in the intercondylar notch. Full extension may then be possible, but the movement of the torn part results in frequent " giving " incidents when it is nipped. There is no severe locking, and the incidents become less and less troublesome as the joint becomes habituated to them. (f) Eventually the bucket handle

spasm. After this test the knee is slowly extended, and any clicks or grating at other stages in the range of extension are noted.

**Internal Derangements.** This useful term comprises those conditions which partly affect the function of the knee joint, without depriving the patient of all use of the limb, and which produce little external change in the appearance of the joint. The interference with function is often temporary, and is most commonly due to a loose body or fragment of attached tissue being caught between the femoral condyles and the tibial plateau. The characteristic features are pain, sudden inhibition of the quadriceps, and "giving" of the knee. Depending on the cause of the obstruction, it may then move out of the way, or be moved out of the



FIG. 589. Performing McMurray's test. See text.

way by some manipulation on the part of the patient, or may remain, causing persistent loss of full extension of the knee, often described as "locking," though in most cases some flexion of the knee is possible. Only in a few cases in which a small torn fragment of cartilage is jammed on both sides of the femoral condyle is there complete limitation of movement. If a loose body is tethered, i.e. pedunculated, as is usual with a torn cartilage, the incidents assume a stereotyped pattern, which is graphically described by the patient, often with the appropriate actions, as "going out" and "going in." If a loose body is free, the patient may feel it moving around the joint in various situations, often in the suprapatellar pouch or on either side of the patella.

*Loose bodies* arise in joints from the tissues which compose it, and may be one of the following: *Fibrous*.—Condensations of cells in organised clot, or after inflammation. Detached synovial villi (villous synovitis). *Cartilaginous*.—Fragments detached by acute trauma. Fragments detached as the result of osteochondritis dissecans. Free multiplication

of cartilage cells in synovial chondromatosis, or degenerative arthritis. *Bony*.—Fragments detached by acute trauma (osteocartilaginous). Osteophytic new bone formation in chronic osteo-arthritis. Bone formation in pedunculated cartilaginous tumours. *Meniscal*.—Fragments of torn cartilage : (a) Free (rare) ; (b) pedunculated.

True new bone formation in a loose body means that it must *have* a blood supply or that it *had* a blood supply. Bone does not form without access to the blood stream. Cartilaginous and fibrous tissue, however, can grow in the joint fluid without a blood supply. A cartilaginous loose body may become adherent and develop a blood supply and then bone may form in it.

**Injuries to the Menisci.** These occur most commonly when the knee is flexed and under strain. A footballer making a sharp turn with his foot fixed in mud may suffer this injury. Collisions with other players are more likely to damage the collateral ligaments of the knee, and meniscus injuries, though they may be associated with this injury, are more commonly associated with injury to the cruciate ligaments. Coal mining is another source of injured menisci. The severe pain and the displacement of the torn part of the cartilage prevent full extension of the knee ; the patient therefore limps on the flexed knee, or may not be able to get about without assistance. Within twelve hours there is considerable swelling of the joint. The knee may gradually straighten, but if the cartilage is caught between the femoral condyle and the tibial plateau, interference with full extension may continue until relieved by manipulation or open operation.

The degree of joint disturbance and its characteristics depend on the degree of tearing of the cartilage, and the amount of freedom left to the torn portion. The lesser injury becomes greater with each successive incident. Cartilage injuries may be classified in the following sequence : (a) Small tear or detachment of the posterior horn. Minimal joint disturbance and healing with no further incidents. (b) Small posterior horn split. Minimal immediate disturbance, further increase in length of split with acute symptoms after perhaps a relatively trivial injury. (c) A  $\frac{3}{4}$ -inch tear. This permits a section of the cartilage to pass to the inner side of the femoral condyle, and the binding down of the ends produces a firm lock with little movement in either direction. Manipulation is often required to reduce it. Careless manipulation may partially free the joint by extending the tear further. (d) A longer tear. This may produce recurrent incidents which the patient learns how to reduce by manipulation. (e) The complete split or the " bucket handle " tear. The torn part may find accommodation in the intercondylar notch. Full extension may then be possible, but the movement of the torn part results in frequent " giving " incidents when it is nipped. There is no severe locking, and the incidents become less and less troublesome as the joint becomes habituated to them. (f) Eventually the bucket handle

tear may become detached at one end and become a pedunculated tag. The incidents then are more frequent and more trivial, for the tag slips easily in and out.

This is the sequence of events in longitudinal tears. Transverse tears, detachments of the anterior and posterior horns, and "parrot beak" tears (i.e. sickle-shaped tears, with a broad base), may produce minor painful incidents, or sudden giving from which recovery is rapid.

*Diagnosis of Meniscus Injuries.* Accurate diagnosis is based on a careful history. Examination of the knee may be negative, but the diagnosis may nevertheless be certain. Physical signs may be confirmatory, e.g. the palpable tag of pedunculated cartilage, or may be merely suspicious, but it is by the history that the diagnosis is most often made. Significant points are: (1) Occurrence of a painful twisting injury. (2) Immediate incapacity. (3) Loss of full extension of the knee. (4) Development of an effusion, and its persistence for some three weeks. (5) Presence of a click. (6) Incidents of "giving," or blocking of extension—rarely of complete locking. (7) The patient may devise a method of putting the torn cartilage back. (8) Decreasing effusion with each incident, and shortened disability after it.

*Examination.* If the torn cartilage has moved back into its normal position and has remained undisplaced for some time, there may be nothing to detect on clinical examination. If there has been a recent incident and the fragment is undisplaced, there is a small excess of fluid in the joint, tenderness over the affected cartilage, a painful posterior horn with perhaps a positive McMurray sign (*vide supra*), and slight wasting of the quadriceps. If the cartilage has remained displaced, there is loss of full extension and there is more wasting of the quadriceps, and attempts to obtain full extension produce pain. If the lesion has been present for some time and there have been frequent effusions, there is marked quadriceps wasting. The medial meniscus is injured three times more frequently than the lateral meniscus. The diagnosis of lateral meniscus tears is based on the same principles, but is more difficult, as the pain from a torn lateral cartilage is often referred to the back of the knee, and the anterior end of the cartilage and the lateral joint line is not so easily palpated. Furthermore, there is no natural posterior horn click, though the posterior horn may be sensitive in the McMurray test.

In difficult cases, further information may be obtained by: (a) Waiting for further incidents to clinch the diagnosis; (b) watching the progress of the knee during a course of physiotherapy (which may clear up many minor causes of disability); or (c) arthrography of the knee. Although the latter investigation is very reliable, a very small number of cases remain in which exploratory arthrotomy is justified.

*Differential Diagnosis.* The chief cause of confusion is loose bodies (*vide supra*). The majority of these are radio-opaque and therefore routine radiography of the joint is essential. "Giving" incidents,

without preliminary pain, may be caused by *weak quadriceps*, due to muscular injury, poliomyelitis or the long-continued mild irritation of osteo-arthritis. There is no locking or "putting the joint in," and the condition responds to a course of quadriceps exercises. Osteo-arthritis of the knees, especially in the presence of loose bodies, may mimic meniscus tears. If symptoms referable to the knee occur without a history of injury, an arthritic origin is suspected in patients over forty-five. Radiography and the response to physiotherapy may help in the diagnosis of doubtful cases when joint crepitus cannot be felt. The presence of a torn

cartilage in an arthritic joint may also give rise to difficulty; arthrography is of great value in difficult cases. *Cysts* of the semilunar cartilages cause pain and a localised swelling over the joint line, and are associated with trauma. They produce a characteristic triad of symptoms (*vide infra*). *Chondromalacia patellæ* is a degenerative condition of the articular surface of the patella producing confusing vague symptoms sometimes associated with "giving," particularly on stairs, and occurring in younger patients (Fig. 590). The pain is localised around the patella, and is increased on



FIG. 590. The eroded joint surface of the patella in chondromalacia patellæ.

pressing the patellæ against the femoral condyles. Oblique radiographs of the knee joint may show arthritic changes on the patellar surface in late cases. For a few days after injury of the medial collateral ligament there is tenderness over the joint line and there may be loss of full extension of the knee. This may be mistaken for a displaced bucket handle tear. The ligament is painful on forced abduction of the tibia. In *Pellegrini-Steida's disease*, in which there is new bone formation in the vicinity of the femoral attachment of the medial collateral ligament, the tenderness is above the joint line, and there may be a palpable mass in which bony change is confirmed by radiography. *Lax Cruciate Ligaments*.—These ligaments may be injured as a cartilage is torn, but if very loose may, perhaps, by permitting nipping of normal cartilages, produce incidents resembling the "giving" in cartilage injuries. Patients with relaxed cruciates should therefore be examined with great care, and if operation is contemplated, patients should understand that the laxity of the cruciates will remain as a source of minor disability. *Congenital discoid meniscus* gives rise to symptoms of internal derangement of the knee joint (*vide infra*).

*Treatment of Cartilage Injuries.* The only effective treatment is removal of the deranged part of the cartilage. Ideally this should be preceded by a short course of quadriceps exercises to familiarise patients with them, for they are required post-operatively to rehabilitate the joint. The operation is carried out in a bloodless field obtained by means of a tourniquet. A short oblique incision is made centered on the joint line on the appropriate side of the patella. The operation is best performed with the patient's knee flexed to a right angle and the surgeon sitting in front of the joint, steadying the patient's foot in his lap. The skin is protected by stockinette glued on with Mastisol. The two layers formed by the patellar retinaculum and the synovial membrane are incised in the same line. Retractors are inserted into the intercondyloid fossa and between the collateral ligament and the femoral condyle; the cartilage is then carefully inspected. Small posterior tears may not be visible from in front, but they require total meniscectomy; hence the need for certainty in diagnosis. The anterior horn is detached and the loose anterior end is grasped by cartilage forceps and pulled forward. The margin of the cartilage is then dissected off the capsule of the joint, passing as far back as can be reached. If there is a posterior tear, the dissection is completed by entry of the cartilage knife into this and the cartilage immediately slips under the femoral condyle and lies in the intercondyloid fossa. It is then cut from its posterior attachment. Only if the cartilage is intact is there any difficulty in detachment of the posterior third from the capsule. Loss of continuity of the cartilage from inadvertent section or a transverse tear may also make operation difficult by depriving the operator of the means of pulling the cartilage forward. This may necessitate a small posterior incision to complete the removal. After inspection of the articular surfaces, the joint is closed by a running catgut stitch down the synovial membrane and up the capsule of the joint. The skin is then closed, and the knee supported by a Robert Jones' bandage (wool, calico bandage, wool and a second calico bandage). The tourniquet is then released.

Post-operatively, a course of quadriceps exercises is given beginning with static quadriceps contraction on the second day (i.e. contraction of the muscles without moving the knee). The patient is then encouraged to bend the knee against the resistance of the bandage and slowly to work the dressing loose. Stitches are removed about the tenth day and a lighter bandage substituted. Flexion of the knee to a right angle or beyond should be attained in the third week, when there is still a variable effusion of fluid into the joint. Full strong extension should be maintained at all times. The patient should be able to get about freely from the sixteenth to the twentieth day. In the absence of ligamentous damage there should be no post-operative disability. Infection is now a very rare post-operative complication; it should be treated by systemic antibiotics, and local antibiotics injected into the joint. The most common complica-

tion is a hamarthrosis, either developing immediately as the result of the operation, or about the end of the first week following too active flexion of the joint. It may, if tense, require aspiration. Generally it absorbs, but results in delay in subsidence of the post-operative effusion.

**Cysts of the Menisci.** These have the same pathological features as ganglia; they are formed by hypertrophy of synovial cells at the junction of the meniscus and the capsule. They differ from simple ganglia in being multilocular. At first they are composed of many small cysts, one of which

becomes large and produces a fluctuant swelling. These cysts have a traumatic origin and may be associated with tears of the cartilage. They are three times as common in the lateral as in the medial cartilage. Most of the cysts appear by herniating around the posterior margin of the lateral or medial ligament of the knee joint, though a few appear anteriorly and posteriorly in the joint. The patient presents a distinctive syndrome: (1) Deep aching pain, often likened to "toothache." This pain is: (a) Worse after exercise; (b) worse in bed, and the patient has to adopt a special position to sleep in; (c) subject to remissions for weeks or even months. (2) The



FIG. 591. A cyst of the lateral meniscus. Note the rounded swelling centred over the posterior third of the cartilage.

presence over the joint line of a rounded swelling which is rubbery or tensely fluctuant (Fig. 591). (3) The patient accurately localises the pain to the affected site where pressure of the examining finger reproduces the pain. The *differential diagnosis* includes tumours in the skin and subcutaneous tissues, marginal exostoses, foreign bodies caught between the cartilage and the tibial plateau, tags of torn menisci caught in the same place, and degenerative cysts in association with advanced osteo arthritis. The only effective *treatment* is total meniscectomy. This is a little more difficult than usual as fibrosis around the cyst ties the cartilage down and makes retraction difficult. Large cysts may need local removal as well. Small cysts may not be entirely removed from the interior of the joint, but disappear after meniscectomy. Local removal of a cyst alone is inevitably followed by rapid recurrence.

**Congenital Discoid Meniscus.** The central tissue composing the meniscus sometimes fails to absorb during development, leaving a solid ovoid



meniscus. The thickened tissue is rolled ahead of the femoral condyles in extension of the knee and then flicks back with an audible click when the limit of its elasticity is reached. This painless click may be encountered in children. Eventually degenerative changes occur and signs of internal derangement of the knee appear. Operative removal of the meniscus is then necessary. The condition is often bilateral, and though most common in the lateral meniscus, has also been reported in the medial meniscus.

**Osteochondritis Dissecans.** The best examples of this disease are seen



FIG. 592. Osteochondritis dissecans of the knee joint. The sclerotic flake of bone is lying in the medial femoral condyle.

in the knee, but other joints with a similar structure may also be affected. The basic condition is the slow separation from the articular surface of an osteocartilaginous flake which eventually becomes a loose body. In some instances, multiple loose bodies may form, due to fragmentation of the original one. The cause of the condition is not fully understood, but there is an undoubted relation to trauma; subchondral fracture followed by non-union is a reasonable explanation of the disease. Occasionally the disease is bilateral, and is then more difficult to explain. It has been suggested that a minor

degree of malformation of the affected joint results in undue pressure on the part of the articular surface which eventually becomes detached. The affected part of the joint is always: (1) Convex; (2) covered with a thick layer of articular cartilage; (3) in contact with another articular surface; and (4) subject to constant motion or pressure. The sites of the disease are, the femoral condyles (Fig. 592), the capitellum of the humerus, the upper surface of the talus, and the head of the second metatarsal. In the knee joint, the disease is most commonly seen in the medial femoral condyle in close relation to the attachment of the posterior cruciate ligament, to which the loose fragment may be attached. The condition may also be found in association with a torn medial meniscus, which may be responsible for the uneven articular pressure postulated above as being an aetiological factor.

There are three stages of the disease, and the clinical manifestations vary accordingly: (1) Radiography shows an area of softening of the condylar surface, with a subjacent denser flake of bone. The symptoms

are a vague aching in the knee, mild interference with function, and transitory small effusions. Diagnosis depends entirely on radiography. (2) A loose body has separated, but remains attached either by a piece of unbroken cartilage, or by adhesions. No loose body is palpable, but the symptoms resemble those of nipping of a meniscus tag; they are frequent and of short duration and cause minor disability. (3) The flake has completely separated as a loose body which may be palpated; there are frequent locking and "giving" incidents.

*Treatment.* Attempts to prevent separation of the flake, by multiple drilling through the softened cartilaginous area, are not usually successful, though occasional benefit has been reported. Operation should be delayed till the flake is about to separate or is actually loose in the joint. The affected area is inspected and any soft cartilage removed, leaving a saucerised cavity. All loose bodies are removed. As after meniscectomy, a post-operative course of quadriceps exercises is instituted. The prognosis depends on the degree of damage to the articular surface. If this is small, the function of the knee may not be affected for years; if large, a degenerative arthritis slowly develops.

**Effusions into the Knee Joint.** The causes of effusion are classified as follows: (1) *Trauma*.—Transitory with strains, sprains, and partial ligamentous tears, or recurrent with the internal derangements of the knee (e.g. torn cartilage). (2) *Infection*.—(a) With a recognisable organism, e.g. streptococcus, staphylococcus, gonococcus, or the tubercle bacillus; (b) with no recognisable infecting agent, e.g. rheumatoid arthritis and subacute infective synovitis; (c) syphilitic. (3) *Hæmorrhage*.—As the result of injury, particularly in hæmophilia, in which repeated hæmorrhages may eventually destroy the joint. (4) *Osteo-arthritis*.—The most common cause of effusion in later life. (5) *Anaphylactic*.—Effusions may be found in serum sickness, but in women recurrent effusions may be related to the hormonal state of the menstrual cycle.

Irritation of the synovial membrane of the knee joint is frequently accompanied by irritation of the semimembranosus bursa; distension of the bursa subsides when the source of irritation is removed. Effusion into the semimembranosus bursa is common, and may be encountered in children. A rounded fluctuant swelling forms on the inner aspect of the insertion of the semimembranosus tendon. It may cause aching due to interference with full extension of the knee joint. As the effusion may subside, it is best to observe cases for a few months before deciding on operative removal.

Aspiration of the joint may be the only means of elucidating the diagnosis, especially in the subacute infective conditions. The quality of the fluid, whether it is clear, opalescent, semipurulent or purulent is observed. In lesser degrees of infection, the cell count is valuable. Always culture the fluid for organisms. Advantage may be taken of the aspiration to introduce an antibiotic into the joint. In rheumatoid

meniscus. The thickened tissue is rolled ahead of the femoral condyles in extension of the knee and then flicks back with an audible click when the limit of its elasticity is reached. This painless click may be encountered in children. Eventually degenerative changes occur and signs of internal derangement of the knee appear. Operative removal of the meniscus is then necessary. The condition is often bilateral, and though most common in the lateral meniscus, has also been reported in the medial meniscus.

**Osteochondritis Dissecans.** The best examples of this disease are seen



FIG. 592. Osteochondritis dissecans of the knee joint. The sclerotic flake of bone is lying in the medial femoral condyle.

in the knee, but other joints with a similar structure may also be affected. The basic condition is the slow separation from the articular surface of an osteocartilaginous flake which eventually becomes a loose body. In some instances, multiple loose bodies may form, due to fragmentation of the original one. The cause of the condition is not fully understood, but there is an undoubted relation to trauma; subchondral fracture followed by non-union is a reasonable explanation of the disease. Occasionally the disease is bilateral, and is then more difficult to explain. It has been suggested that a minor

degree of malformation of the affected joint results in undue pressure on the part of the articular surface which eventually becomes detached. The affected part of the joint is always: (1) Convex; (2) covered with a thick layer of articular cartilage; (3) in contact with another articular surface; and (4) subject to constant motion or pressure. The sites of the disease are, the femoral condyles (Fig. 592), the capitellum of the humerus, the upper surface of the talus, and the head of the second metatarsal. In the knee joint, the disease is most commonly seen in the medial femoral condyle in close relation to the attachment of the posterior cruciate ligament, to which the loose fragment may be attached. The condition may also be found in association with a torn medial meniscus, which may be responsible for the uneven articular pressure postulated above as being an ætiological factor.

There are three stages of the disease, and the clinical manifestations vary accordingly: (1) Radiography shows an area of softening of the condylar surface, with a subjacent denser flake of bone. The symptoms

**Osteochondritis Juvenilis of the Tibial Tubercle** (*Osgood-Schlatter Disease*). A similar series of changes, though not so distinctly marked as those in Perthes' disease, may occur in the tibial tubercle in children from the age of ten to eighteen years. It is more common in boys. The complaint is of pain around the tibial tubercle which is swollen and tender. The disease is not infrequently bilateral, but one tubercle is in a different stage of the disease from the other. Radiological examination



FIG. 593. Osgood-Schlatter disease. The fragmentation of the epiphysis has left one fragment un-united.

shows enlargement of the tibial tubercle and patchy sclerosis and fragmentation (Fig. 593). Comparison with the tubercle of the opposite side is valuable, not only because of the occasional bilateral occurrence of the disease, but because the normal tubercle is variable in its development. The disease grumbles on for many months or years; eventually it settles down, leaving a permanently enlarged tubercle which in later life may sometimes be the source of pain after exertion. An acute onset is occasionally seen; then rest in a knee fixation plaster for a few weeks is indicated. Ordinarily, however, reassurance combined with restriction of severe exertion is all that is required.

**Prepatellar Bursitis (Housemaid's Knee).** The prepatellar bursa is a

arthritis, the effusion is usually bilateral, massive, and accompanied by severe wasting of the muscles around the knee. The aspirated fluid is clear.

Subacute infective synovitis is a convenient term for those cases in which there is a hot, red and swollen knee, with some palpable synovial thickening, and perhaps some general disturbance, in the form of a raised temperature and white cell count. The condition subsides rapidly with rest in the majority of cases. Aspiration of the fluid shows an increase in polymorphonuclear cells, but no organisms are found. In a few cases, the condition grumbles on perhaps for years, with a small effusion and thickened synovial membrane. Eventually degenerative arthritic changes supervene.

**Rupture of the Extensor Expansion.** The extensor apparatus of the knee is liable to rupture at one of several levels. The rectus femoris may be detached from its upper origin; it may rupture through the belly of the muscle, or musculotendinous junction; and in the elderly may be detached from the patellar insertion. The incident is sudden; it is accompanied by pain, swelling and often bruising, and is recognised by the distorted shape of the quadriceps, accentuated by its contraction. Except for detachments from the patella, repair is not needed, for the impairment of function seems to be of little moment to the patient. Fractures of the patella by muscular violence may be regarded as a rupture of the extensor expansion at a lower level. Very rarely avulsion injury of the tibial tubercle may occur, although pain and irregularity in this region is most commonly due to osteochondritis juvenilis of the tibial tubercle (Schlatter's disease).

**Recurrent Dislocation of the Patella.** This is due to a relaxation of the quadriceps expansion on the medial side permitting lateral displacement; it may be combined with other anomalies which facilitate the slipping of the patella laterally (underdevelopment of the lateral femoral condyle and the patella, or excessively lax patellar retinaculæ associated with a poorly developed quadriceps and knock-knee deformity). Recurrent dislocation eventually results in a patellofemoral arthritis, and osteoarthritis of the rest of the joint. *Treatment* is divided into two stages: (a) Before the onset of arthritis, when attempts to correct the condition may be made. (b) After the development of patellofemoral arthritis, when excision of the patella is advised. Corrective procedures which may be used alone or in combination are: (a) Transplantation of the tibial tubercle and quadriceps insertion to a lower and more medial position. (b) Shortening by plication of the patellar retinaculum on the medial side. (c) Transplantation of the vastus medialis insertion to a more lateral position on the patella. (d) Correction of any knock-knee deformity. In the younger patient in whom growth assists in balancing patellar pressures, transplantation of the tibial tubercle is best. In older patients, excision of the patella is probably the best method, for patellofemoral arthritis is commonly present.

incidents of locking and "giving." Eventually movement is restricted, and the knee becomes permanently flexed. The course of the disease is irregular. In the early stages, painful periods may follow minor strains, although there are intervals of comparative freedom from trouble. Patients complain of grating on movement, and when the arthritis is limited to the patellofemoral joint, there is increased pain on ascending or descending stairs.

*Treatment.* In the early stages, much relief may be given by a course of radiant heat, massage and exercise. Maintenance of the tone and development of the quadriceps is most important. A fixed flexion deformity must be avoided by rest in bed and traction on the leg or, if necessary, by splinting. Limited operative procedures are sometimes of value, e.g. removal of loose bodies, excision of a torn cartilage, or occasionally removal of marginal exostoses. When the arthritis is confined mainly to the patella, or when condylar osteophytes catch on the patella, removal of the patella is of value. Arthrodesis of the joint is a comparatively certain and straightforward procedure, if the joint surfaces are not excessively sclerosed as in long-standing neuropathic joints. An invaluable aid to rapid union is the use of compression, applied by screws attached to Steinmann's pins transfixing the bone on each side of the joint. A single stiff knee is not a great handicap; therefore arthrodesis may be indicated in young subjects in whom the arthritis would be a continued handicap. In older people, the indication is usually pain, or a fixed flexion deformity. With good hips and ankles it is possible to get about with both knees fused. The use of a knee-flexion caliper to relieve the joint from strain is sometimes of value, though the additional weight of the appliance may be a handicap. In patients with progressive lateral angulation, the appliance may assist in controlling it. Manipulation, except for the relief of locking, is of little value, and improvement in position is best achieved by continuous traction and persistent exercises. The relief of pain is often a problem, and in early arthritis in elderly patients, particularly those with much oedema and tenderness over the medial collateral ligament, Butazolidin (200 mg. t.d.s. p.c. reduced to 100 mg. in a month) is often of value and always worth a trial.

**Rheumatoid Arthritis.** This disease may be seen in the knees in the acute phase as large tense bilateral effusions, accompanied by joint involvement in the hands, and the manifestations of a febrile disease. Often the condition burns itself out, but osteo-arthritis supervenes and needs treatment along the lines already discussed.

**Syphilis** may affect the knee at varying ages. Thus congenital syphilis may result in an acute epiphysitis with separation of the epiphyses, resembling scurvy in young children, and later in life there may be a less acute bilateral synovitis with some periosteal reaction (Cluttons' joints). In acquired syphilis, local gummas may involve the knee, but the most commonly seen manifestation is a degenerative arthritis of the joint due

pathway of lymphatic return from the leg. Infections below the knee are therefore liable to produce acute bursitis. This may be controlled by systemic penicillin and local aspiration and penicillin replacement. Occasionally incision and drainage are required. Either following infection or as the result of the repeated trauma of kneeling, the bursa may become thickened or the seat of a chronic effusion. Occasionally the bursa slowly becomes replaced by a fibrous mass or even partly calcified. Under these conditions it should be excised.

**Tuberculosis of the Knee Joint.** This is a disease of childhood of increasingly infrequent occurrence. It manifests itself by the usual evidence of knee joint dysfunction, namely, limping, pain and swelling. Muscle wasting is rapid. Synovial spread in the suprapatellar bursa produces a thickened roll of tissue above the patella. A flexion deformity of the knee occurs and steadily increases. A child with a suspicious effusion should be admitted to hospital for observation. The knee is steadied by skin traction. Observation of the child's general condition, X-ray of the lungs, the Mantoux test, the results of aspiration of the knee, and the radiological appearances of the joint usually enable a diagnosis to be made. In a few cases, biopsy of an enlarged inguinal gland may be confirmatory. There are few conditions which can mimic a tuberculous knee. One of these, however, is congenital syphilis, but the arthritis is usually bilateral or soon becomes so, and there is a positive Wassermann reaction. A subacute infective synovitis, or a penicillin-controlled acute infection may occasionally cause doubt. *Treatment* consists of the same general regime as for tuberculosis in other sites and immobilisation of the knee either on a Thomas's splint or in plaster of Paris. In children, an infection limited to synovial membrane may heal with retention of some movement. In adults, arthrodesis is the aim, and this may need a preliminary excision of the joint when the infection is controlled. Tuberculous osteitis and arthritis are discussed in Chap. 80.

**Osteo-arthritis of the Knee Joint.** This may be part of generalised osteo-arthritis, usually in patients of characteristic short heavy build. Changes are then seen in other joints, notably the spine, and there may be typical enlargement of the terminal interphalangeal joints (Heberden's nodes). Both knees are usually affected though not to the same degree.

*Monarticular osteo-arthritis* may be due to one of many causes which throw an increased strain on the knee joint. It is found in association with a stiff arthritic hip, due to the added stresses the knee has to bear. It may be the sequel to subacute infection of the joint, repeated injury produced by a torn meniscus, a fracture into the joint, or osteochondritis dissecans. The pathology of osteo-arthritis is described in Chap. 81.

The disease usually starts after middle age. At first there is stiffness after rest, and muscular fatigue and aching after exercise. Small synovial effusions occur. With the marginal degeneration of the joint, alterations in its contours are produced, and loose bodies may appear and cause

incidents of locking and "giving." Eventually movement is restricted, and the knee becomes permanently flexed. The course of the disease is irregular. In the early stages, painful periods may follow minor strains, although there are intervals of comparative freedom from trouble. Patients complain of grating on movement, and when the arthritis is limited to the patellofemoral joint, there is increased pain on ascending or descending stairs.

*Treatment.* In the early stages, much relief may be given by a course of radiant heat, massage and exercise. Maintenance of the tone and development of the quadriceps is most important. A fixed flexion deformity must be avoided by rest in bed and traction on the leg or, if necessary, by splinting. Limited operative procedures are sometimes of value, e.g. removal of loose bodies, excision of a torn cartilage, or occasionally removal of marginal exostoses. When the arthritis is confined mainly to the patella, or when condylar osteophytes catch on the patella, removal of the patella is of value. *Arthrodesis* of the joint is a comparatively certain and straightforward procedure, if the joint surfaces are not excessively sclerosed as in long-standing neuropathic joints. An invaluable aid to rapid union is the use of compression, applied by screws attached to Steinmann's pins transfixing the bone on each side of the joint. A single stiff knee is not a great handicap; therefore arthrodesis may be indicated in young subjects in whom the arthritis would be a continued handicap. In older people, the indication is usually pain, or a fixed flexion deformity. With good hips and ankles it is possible to get about with both knees fused. The use of a knee-flexion caliper to relieve the joint from strain is sometimes of value, though the additional weight of the appliance may be a handicap. In patients with progressive lateral angulation, the appliance may assist in controlling it. Manipulation, except for the relief of locking, is of little value, and improvement in position is best achieved by continuous traction and persistent exercises. The relief of pain is often a problem, and in early arthritis in elderly patients, particularly those with much oedema and tenderness over the medial collateral ligament, Butazolidin (200 mg. t.d.s., p.c. reduced to 100 mg. in a month) is often of value and always worth a trial.

*Rheumatoid Arthritis.* This disease may be seen in the knees in the acute phase as large tense bilateral effusions, accompanied by joint involvement in the hands, and the manifestations of a febrile disease. Often the condition burns itself out, but osteo-arthritis supervenes and needs treatment along the lines already discussed.

*Syphilis* may affect the knee at varying ages. Thus congenital syphilis may result in an acute epiphysitis with reparation of the epiphyses, resembling scurvy in young children, and later in life there may be a less acute bilateral synovitis with some periosteal reaction (Clutton's joints). In acquired syphilis, local gummas may involve the knee, but the most commonly seen manifestation is a degenerative arthritis of the joint due



to tabes (Charcot's joint). The deprivation of the joint of its sensibility (occurring also in other nervous diseases) results in repeated injuries which receive scant care, and eventually a completely unstable joint is produced. This requires an appliance to support the joint, or arthrodesis, but fusion is not easily accomplished in the sclerotic and very degenerated joint.

**Knock-knee and Bow-legs (Genu Valgum and Genu Varum).** Rickets was the common cause of both conditions in times past. Though this disease has been almost abolished, minor degrees of both deformities are still commonly seen. Knock-knee may be regarded as a stage in the development of some children, who form the majority of cases seen, but there are still a few in whom there is a more marked growth disturbance than can be corrected by conservative means. In both types, the cause is unknown and is termed "idiopathic" knock-knee. The child is healthy and often fat, and stands with the feet wide apart. There is often an accompanying eversion of the feet with apparent flattening of the arches. In assessing the degree of knock-knee, the child is seated on the mother's lap, and the extended knees are brought together so that they just touch. The distance between the medial malleoli is then measured. It will be noted that this distance is usually much less than the distance between the feet when the child stands. The exaggeration on standing is due to the ability of the child to hyperextend the knee joints, and this may be verified by hyperextending the knees when the child is seated. It will also be noted that the deformity disappears with slight flexion of the knees. The deformity steadily decreases as the child grows and the knee joints become firmer. So long as there is hyperextensibility of the knee there remains the possibility of spontaneous recovery—usually up till six to eight years of age. *Epiphyseal Knock-knee.*—In these children there is no hyperextensibility of the knee, or it is inadequate to account for the deformity which is severe, i.e. over 4 inches between the malleoli. An X-ray confirms bony curvature, often accompanied by "flaring" of the epiphyseal plates (i.e. the epiphyseal edges tend to taper and to overhang the metaphysis), which may be at an angle to the shaft of the bone. The main deformity may be above the knee, rarely below it. Such cases do not do so well on conservative treatment, and subsequently often require an osteotomy to obtain complete correction.

*Treatment* is summarised as follows: If the knees are hyperextensible always treat conservatively. If the knees are not hyperextensible start with conservative treatment. Under two years of age, and under two inches between the malleoli: wedge sole and heel  $\frac{1}{2}$  inch, or, better, provide a valgus insole as in "Inneraze" shoes. Under three and under three inches: order valgus insoles and night splints. Splints must obtain firm control of the knee, so use a good pattern such as the Ellis splint. Over four and over four inches: if hyperextensible continue conservative therapy as for under three's; if not hyperextensible, then correct by osteotomy of the femur, or rarely by tibial osteotomy.

*Bow-legs* are much less common than knock-knees. They may be due to : (a) bowing of the tibia, sometimes more marked in one leg than the other ; (b) uneven femoral condylar growth and capsular laxity, the reverse of that seen in "idiopathic" knock-knee ; or (c) epiphyseal angulation with flaring and increased density of the medial tibial cortical bone. Mild cases may be treated by wedging the outer side of the heel and sole, though this is probably more of a parental placebo than an effective procedure. Most cases recover spontaneously. If the deformity is severe, or if one tibia is markedly bowed, and there is failure of spontaneous correction, tibial osteotomy or more rarely femoral osteotomy should be performed before patients reach the age of five years.

to tabes (Charcot's joint). The deprivation of the joint of its sensibility (occurring also in other nervous diseases) results in repeated injuries which receive scant care, and eventually a completely unstable joint is produced. This requires an appliance to support the joint, or arthrodesis, but fusion is not easily accomplished in the sclerotic and very degenerated joint.

**Knock-knee and Bow-legs (Genu Valgum and Genu Varum).** Rickets was the common cause of both conditions in times past. Though this disease has been almost abolished, minor degrees of both deformities are still commonly seen. Knock-knee may be regarded as a stage in the development of some children, who form the majority of cases seen, but there are still a few in whom there is a more marked growth disturbance than can be corrected by conservative means. In both types, the cause is unknown and is termed "idiopathic" knock-knee. The child is healthy and often fat, and stands with the feet wide apart. There is often an accompanying eversion of the feet with apparent flattening of the arches. In assessing the degree of knock-knee, the child is seated on the mother's lap, and the extended knees are brought together so that they just touch. The distance between the medial malleoli is then measured. It will be noted that this distance is usually much less than the distance between the feet when the child stands. The exaggeration on standing is due to the ability of the child to hyperextend the knee joints, and this may be verified by hyperextending the knees when the child is seated. It will also be noted that the deformity disappears with slight flexion of the knees. The deformity steadily decreases as the child grows and the knee joints become firmer. So long as there is hyperextensibility of the knee there remains the possibility of spontaneous recovery—usually up till six to eight years of age. *Epiphyseal Knock-knee.*—In these children there is no hyperextensibility of the knee, or it is inadequate to account for the deformity which is severe, i.e. over 4 inches between the malleoli. An X-ray confirms bony curvature, often accompanied by "flaring" of the epiphyseal plates (i.e. the epiphyseal edges tend to taper and to overhang the metaphysis), which may be at an angle to the shaft of the bone. The main deformity may be above the knee, rarely below it. Such cases do not do so well on conservative treatment, and subsequently often require an osteotomy to obtain complete correction.

*Treatment* is summarised as follows: If the knees are hyperextensible always treat conservatively. If the knees are not hyperextensible start with conservative treatment. Under two years of age, and under two inches between the malleoli: wedge sole and heel  $\frac{1}{8}$  inch, or, better, provide a valgus insole as in "Inneraze" shoes. Under three and under three inches: order valgus insoles and night splints. Splints must obtain firm control of the knee, so use a good pattern such as the Ellis splint. Over four and over four inches: if hyperextensible continue conservative therapy as for under three's; if not hyperextensible, then correct by osteotomy of the femur, or rarely by tibial osteotomy.

plantigrade foot is achieved, the uneven muscle balance results in partial redevelopment of the deformity when support is relaxed. Abnormalities of the bones prevent a normal foot posture ever being achieved. The prognosis is often bad, and though a sound functional foot may be obtained, it is always associated with some rigidity and some deformity.

The malposition of the foot is described by reference to four main deformities, but these are often combined. The four deformities are: *Talipes Equinus*.—Plantar flexion of the ankle so that the child walks on the toes. *Talipes Calcaneus*.—Dorsiflexion of the ankle so that the child walks on the heel. *Talipes Valgus*.—The foot is everted and abducted. *Talipes Varus*.—The foot is inverted and adducted. These deformities include the fore-foot as well as the ankle. Adduction of the fore-foot may be met with alone, without any ankle deformity. Abduction

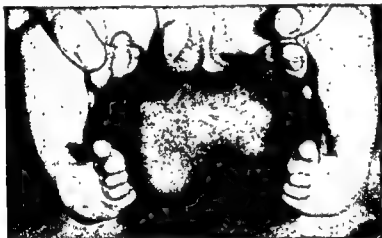


FIG. 595. Severe bilateral talipes equino-varus due to defective development of the musculature of both calves.

of the first metatarsal from the second (*metatarsus primus varus*) may be a limited form of metatarsus varus, and is the basic cause of hallux valgus in many cases. There is a palpable separation of the first and second metatarsal heads.

The normal baby's foot is held in mild talipes calcaneo-valgus, but in a very short time assumes a normal position. In *talipes calcaneo-valgus*, the position cannot be easily corrected by manipulation and there is usually some contraction of the skin on the outer side of the ankle. This is a relatively uncommon deformity. *Seventy-five per cent.* of club feet lie in the position of *equinovarus*. The ankle is inverted, and in the most severe cases the metatarsus is adducted and the foot lies along the inner aspect of the tibia (Fig. 595).

*Talipes Equinovarus*. The object of treatment is to obtain by manipulation and splinting a normal plantigrade position of the foot. The primary treatment to be adopted depends on the severity of the

## CHAPTER 90

### THE ANKLE AND FOOT

**Club-foot (Talipes).** These congenital deformities of the foot are divisible into two main clinical groups : (1) *Postural*.—The foot lies in a deformed position, but the calf is normally developed and the structure of the foot is normal. It follows from this that the later development of the foot shows a normal bone complement. The deformity, though it may be marked, is not as severe as in developmental talipes (*vide infra*).



FIG. 594. Mild postural talipes equino-varus, more marked on the right (reader's left) than on the left.

The condition may be unilateral or bilateral ; when bilateral both feet may not be similarly deformed (Fig. 594). The deformity is due to malposition *in utero*, and when the foot is straightened there is normal balance of the musculature to maintain it in position. The condition is often easily corrected, and the correction easily maintained. The prognosis is good. The majority of cases are of this type. (2) *Developmental*.—The foot is more markedly deformed (Fig. 595). There is failure of development of the calf, and alterations in the shape of the foot and the skin markings of the sole. Other congenital abnormalities may be associated with the deformity such as absence of one or more toes or tarsal bones, and, as the bones become defined radiologically, abnormalities of shape appear, partly due to maldistribution of pressure and impaired function of the foot, but also to congenital malformation. The condition may have an hereditary tendency. The deformity is much more difficult to correct and to retain than in the postural type, and if a normal-looking

it may only require boots with suitable wedging of the soles and heels. If the inversion deformity is the most troublesome, provision of an inside iron and outside T strap may be satisfactory. When the deformity is so severe that normal footwear is impossible, a choice must be made between continuation of manipulations followed by application of plaster of Paris, or an operation on the soft tissues. An ingenious modification of the wedged-plaster principle known as Kite's plaster may be used in the foot to give gradual correction at the required level. The equinus deformity can often be corrected by lengthening the tendo Achilles, and this also has the advantage of improving the persistently inverted heel. Correction of the subtaloid and mid-tarsal deformity is more difficult and is done by planned soft tissue dissection on the medial aspect of the sole so that the bones can be swung into place and retained by plaster (Brockman's operation).

When, in spite of these measures, correction is still inadequate, removal of a wedge of bone from the tarsus is necessary. There has been general agreement that this method should be reserved for older children (over the age of eight). When the deformity is such that retentive appliances are unsatisfactory, there is much to be said for doing the operation earlier, even if a further operation has to be done at a later date because of the uneven development of the bones of the foot. Early operation avoids soft tissue strains, uneven pressure on bones and joints, the bad psychological effects of unsightly appliances, and encourages bone and muscle development by the use of a plantigrade foot.

*Talipes calcaneo-valgus* or *rarus* can easily be corrected by strapping the leg and foot to a padded metal splint along the front of the limb or to an L-shaped splint along the side of the limb. The contraction mainly affects the soft tissues.

**Other Congenital Deformities of the Foot.** Many congenital deformities are encountered in the foot, and the minor ones are common. Absence of the forefoot, absent bones of the tarsus, congenital fusion of bones, e.g. the calcaneo-navicular bar mentioned below, occur. They may be part of a club-foot and make its retention in the corrected position difficult and restitution to normal impossible.

Rare hereditary defects, such as the "lobster claw" deformity, also occur. There is a familial factor in the development of metatarsus primus varus, a precursor of hallux valgus. The most commonly encountered deformities are those of the toes. As in the hand, supernumerary digits may be encountered. The great toe may be double, and the degree of separation of the two parts is very variable. A rudimentary sixth toe may also be present. The most common defect of the fifth toe is a contracture causing it to overlie the fourth. This defect should be corrected about the age of four years by a plastic procedure, in which the extensor tendon of the fifth toe is severed high up on the dorsum of the foot and passed around the inner aspect of the proximal phalanx and under

deformity. One of the best and most universally accepted methods is the application of a Denis Browne splint, but for its successful use the foot must not be severely deformed (Fig. 596). If it is so deformed that it will not fit the splint without excessive pressure on the soft tissues, there is a grave risk of pressure sores. In these circumstances, preliminary treatment consists of manipulation followed by the application of plaster of Paris until a reasonable position is achieved; then further treatment is continued with the Denis Browne splint. This consists of sole plates with side bars up the outer sides (Fig. 596). Bolts are attached to these below

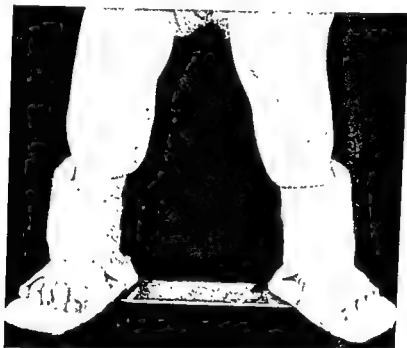


FIG. 596. Method of application of Denis Browne splint with strapping.

the heels so that they can be bolted to a cross bar. Correction of each foot is obtained partly by the foot-piece, and is further braced by the attachment to the other foot, and the child is still free to kick and develop his muscles. If the child grows too big and strong for strapping to be effective, lace up boots without toe caps may be substituted for the sole plates and side bars. The length of time the splint needs to be worn depends on the case with which correction is obtained. It may be dispensed with when the child maintains a normal foot posture with the splint removed and the surgeon can obtain over-correction easily by manipulation. After removal of the splint the foot must be watched, and any recurrence treated by further splinting or alterations in the footwear.

When the child has grown beyond the stage of control with the Denis Browne splint and lace-up boots, a decision on future treatment must be made. If the child has a plantigrade foot with relatively little deformity,

tendo Achilles, or the woman used to high heels or with pes cavus is uncomfortable with low heels. Raising the heel results in more pressure being borne by the heel until the rise is such that the foot slips in the shoe and presses the metatarsals into the toe of the shoe. High heels may thus be a cause of metatarsalgia. Most women are best off with a medium heel (about 1½ inches), commonly called a Cuban or an Oxford heel.

**The Adult Everted Flat-foot or Pes Planus.** Such a foot may do all that its owner normally demands of it, but it is liable to foot strain after long standing, or, in women, after a change from a medium to a low heel. The symptoms are aching and pain after exercise and long standing, and the pain is often felt up the leg. Evening swelling of the foot may also



FIG. 597. Unilateral pes cavus, secondary to poliomyelitis.

be noted. The foot is tender at various sites, and passive movement may produce pain. In late cases, there may be arthritic changes in the mid-tarsal region, but usually radiographs show no abnormality. Symptoms may be provoked by : (a) Change to an occupation demanding more time on the feet ; (b) pregnancy ; (c) a change in footwear ; (d) general ill-health and decreased muscle tone ; and (e) too early activity on the feet following an illness.

Treatment is determined by the causative condition. In all cases, the acute phase can be cleared up by a course of radiant heat, massage and exercise, combined with faradism to the leg muscles. In cases with underlying mid-tarsal arthritis or postural deformities, a recurrence is likely unless some attempt is made to balance the pressures on the foot. This may consist of altering the footwear, e.g. providing a valgus insole to support the longitudinal arch, and above all adjusting the height of the heel. The rigidity of the arch-support is adjusted to the weight of



the metatarsophalangeal joint ; it is then sewn to the abductor of the fifth toe (Lapidus operation).

The terminal interphalangeal joints are frequently malformed, and this may lead to an early hammer-toe defect of one of these joints. Fusion of this joint is difficult at an early age ; therefore correction of the deformity should be postponed till the toe is of a reasonable size (about the age of twelve years). The terminal joint is then excised, and the toe temporarily fixed straight by an intramedullary Kirschner wire which is removed after three weeks.

**Postural Defects.** There are many variations in the normal posture of the foot consistent with sound function. The mobility of the tarsus is an inborn condition, and though it may be improved with exercises, as it is in ballet dancers, it remains throughout life a characteristic of the particular foot. Thus an elderly lady may well have a more mobile foot than an athletic young man.

Many children have a valgus stance giving the appearance of a flat foot, and it may remain till adolescence when it disappears spontaneously. Sometimes the condition persists into adult life, but function is often satisfactory. In these cases, the mothers often complain that the children turn their feet in when walking or trip easily. There is little to be done but to reassure them. The more marked cases may benefit from internal wedging of the heel ( $\frac{1}{4}$  or  $\frac{1}{2}$  inch), and the heel may be prolonged on the same side to support the arch. The provision of shoes with a firm arch which is a little raised, e.g. Inneraze shoes, is often a guarantee of better foot posture and more even wear of the shoes.

Tibial torsion is an occasional cause of internal rotation of the feet. With the feet held in the normal position, it can be seen that the patellæ look outwards ("squinting" patellæ). This deformity decreases as a rule with growth, but may require correction between the ages of six and eight years by a rotational osteotomy. Built-in valgus supports and wedges aid correction in the actively growing period.

The height of the arch is very variable. A severe unilateral pes cavus is usually the result of muscular deficiency in the calf and may result from poliomyelitis (Fig. 597). Bilateral pes cavus may be a congenital abnormality, and varies very much in degree. Severe or even moderate pes cavus may lead to difficulty in obtaining shoes which lace up satisfactorily. As the patient grows older, there is excessive pressure on the metatarsal heads, with prolapse of the transverse arch and clawing of the toes, and possibly the development of hammer toes and bursæ over them. Comfort can usually be obtained by suitably made footwear. The essential features of this are a valgus insole and metatarsal pad, a rise in the height of the heel so that more weight is taken on the heel, and soft toe caps if the toes are clawed.

There is a prevailing impression that flat heels are sensible and essential in correcting foot complaints. This is erroneous. The patient with a short

tendo Achilles, or the woman used to high heels or with pes cavus is uncomfortable with low heels. Raising the heel results in more pressure being borne by the heel until the rise is such that the foot slips in the shoe and presses the metatarsals into the toe of the shoe. High heels may thus be a cause of metatarsalgia. Most women are best off with a medium heel (about 1½ inches), commonly called a Cuban or an Oxford heel.

**The Adult Everted Flat-foot or Pes Planus.** Such a foot may do all that its owner normally demands of it, but it is liable to foot strain after long standing, or, in women, after a change from a medium to a low heel. The symptoms are aching and pain after exercise and long standing, and the pain is often felt up the leg. Evening swelling of the foot may also



FIG. 597. Unilateral pes cavus, secondary to poliomyelitis.

be noted. The foot is tender at various sites, and passive movement may produce pain. In late cases, there may be arthritic changes in the mid-tarsal region, but usually radiographs show no abnormality. Symptoms may be provoked by : (a) Change to an occupation demanding more time on the feet ; (b) pregnancy ; (c) a change in footwear ; (d) general ill-health and decreased muscle tone ; and (e) too early activity on the feet following an illness.

Treatment is determined by the causative condition. In all cases, the acute phase can be cleared up by a course of radiant heat, massage and exercise, combined with faradism to the leg muscles. In cases with underlying mid-tarsal arthritis or postural deformities, a recurrence is likely unless some attempt is made to balance the pressures on the foot. This may consist of altering the footwear, e.g. providing a valgus insole to support the longitudinal arch, and above all adjusting the height of the heel. The rigidity of the arch support is adjusted to the weight of

the metatarsophalangeal joint ; it is then sewn to the abductor of the fifth toe (Lapidus operation).

The terminal interphalangeal joints are frequently malformed, and this may lead to an early hammer-toe defect of one of these joints. Fusion of this joint is difficult at an early age ; therefore correction of the deformity should be postponed till the toe is of a reasonable size (about the age of twelve years). The terminal joint is then excised, and the toe temporarily fixed straight by an intramedullary Kirschner wire which is removed after three weeks.

**Postural Defects.** There are many variations in the normal posture of the foot consistent with sound function. The mobility of the tarsus is an inborn condition, and though it may be improved with exercises, as it is in ballet dancers, it remains throughout life a characteristic of the particular foot. Thus an elderly lady may well have a more mobile foot than an athletic young man.

Many children have a valgus stance giving the appearance of a flat foot, and it may remain till adolescence when it disappears spontaneously. Sometimes the condition persists into adult life, but function is often satisfactory. In these cases, the mothers often complain that the children turn their feet in when walking or trip easily. There is little to be done but to reassure them. The more marked cases may benefit from internal wedging of the heel ( $\frac{1}{4}$  or  $\frac{1}{2}$  inch), and the heel may be prolonged on the same side to support the arch. The provision of shoes with a firm arch which is a little raised, e.g. Inneraze shoes, is often a guarantee of better foot posture and more even wear of the shoes.

Tibial torsion is an occasional cause of internal rotation of the feet. With the feet held in the normal position, it can be seen that the patellæ look outwards ("squinting" patellæ). This deformity decreases as a rule with growth, but may require correction between the ages of six and eight years by a rotational osteotomy. Built in valgus supports and wedges aid correction in the actively growing period.

The height of the arch is very variable. A severe unilateral pes cavus is usually the result of muscular deficiency in the calf and may result from poliomyelitis (Fig. 597). Bilateral pes cavus may be a congenital abnormality, and varies very much in degree. Severe or even moderate pes cavus may lead to difficulty in obtaining shoes which lace up satisfactorily. As the patient grows older, there is excessive pressure on the metatarsal heads, with prolapse of the transverse arch and clawing of the toes, and possibly the development of hammer toes and bursæ over them. Comfort can usually be obtained by suitably made footwear. The essential features of this are a valgus insole and metatarsal pad, a rise in the height of the heel so that more weight is taken on the heel, and soft toe caps if the toes are clawed.

There is a prevailing impression that flat heels are sensible and essential in correcting foot complaints. This is erroneous. The patient with a short

pressure. Because of the rigidity of the epidermis, separation may occur between the dermis and epidermis with the formation of a hæmorrhagic blister, and this may at times be very painful. Treatment consists of balancing the foot by wedges or arch supports to relieve pressure over the affected area, and shaving off the thickened epidermis. The area is continually covered with an air-tight zinc strapping. Each time it is renewed, it removes some softened epithelium from the surface. The thickened skin can be completely removed in this way.

Plantar callosities are often confused with plantar warts. Because of the pressure on a plantar wart, it is driven into the sole of the foot, and thus becomes very painful. Being of infective origin, warts are likely to be multiple and bilateral. Areas of skin, not usually subject to excessive pressure are affected. A plantar wart assumes a more localised rounded form than a callus, and warts may occur in groups. Distinction in doubtful cases can usually be made by carefully paring the thickened skin with a razor blade. In a callosity, an evenly pink dermis is exposed, unless there has been epidermal separation. In a wart, a small circle with a whitened periphery is exposed, and this appearance is produced by dermal tissue around the margins of the in-driven wart. A plantar wart may be successfully eradicated by application of glacial acetic acid twice a day with a match-stick. Podophyllin powder or corn plasters may also succeed, but they are not always successful. The most satisfactory method for an established wart is curettage followed by carbolicisation of the base. Even this is not always successful, for recurrences may occur or small invisible warts may later enlarge.

The reaction of the foot to abnormal pressure is well demonstrated by the common *bunion*. The defence against pressure and friction first consists of thickening of the epidermis; there is then a thickening of subcutaneous fibrous tissue and the development in this of a distended lymphatic, which eventually becomes lined with secreting cells of a synovial type and forms an adventitious bursa. Deep to this there is more thickened fibrous tissue, forming in the case of a bunion part of the joint capsule, and then the metatarsal head or bony exostosis.

The bursa, communicating with lymphatics as it does, is liable to infection. When this occurs and pus discharges, a bursal fistula is likely to follow. The opening is liable to blockage which may be followed by recurrent inflammation. This story is particularly common with bunions. Mechanical irritation of a bursa is apt to be followed by an effusion, which eventually subsides. The appearance of the swelling is thus very variable. Pain can be relieved by stretching the shoes over the prominence, by appliances designed to relieve the pressure, or changed footwear, but the only really effective and permanent relief is provided by removal of the redundant skin, the bursa and the underlying cause of pressure. This operation is discussed in more detail later.

Other common prominences in the foot are : *Hammer Toes*.—These

the patient and to the degree of flexion it is wished to avoid at the mid-tarsal region. The height of the support varies with the shape of the foot. In a few chronic cases and in those with mid-tarsal arthritis, manipulation of the foot may be carried out as a prelude to a course of physiotherapy.

**Peroneal Spasm (Spasmodic Flat-foot or Pes Planus).** The characteristic feature of this condition is a flat everted painful foot with pain behind the fibular malleolus where the peroneal tendons can be seen and felt to be tense and tender. The condition shows considerable variation in its intensity, and remissions are common. It may be very painful and have

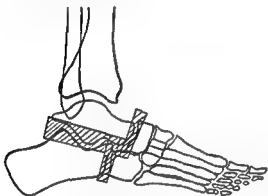


FIG. 598. The bone removed in performing triple arthrodesis of the foot.

an acute onset. It is most commonly encountered in adolescence. Two groups of cases may be separated by radiological examination. In the first, the radiograph shows the presence of a calcaneo-navicular bar, or of a talo-calcaneal synchondrosis or synostosis in the region of the sustentaculum tali. These cases are resistant to treatment, and local resection of the ankylosing bar of bone has not proved effective. If persistently troublesome, a triple arthrodesis

of the subtaloid, talo-navicular and calcaneo-cuboid joints is necessary (Fig. 598).

In the second group no radiological abnormality can be discovered. In acute cases, immobilisation in plaster in the neutral position and avoidance of weight-bearing are necessary. In the less acute cases, a plaster overshoe may be worn and weight-bearing permitted. If the pain slowly subsides, physiotherapy may be begun. Spontaneous remissions and recurrences occur. Persistent pain may demand triple arthrodesis. The cause is unknown and the cause of the reflex spasm is uncertain, but it may be due to irritation of the interosseous talo-calcaneal ligament, for procaine infiltration of this area sometimes relieves the spasm.

**Pressure Effects and Bony Swellings in the Foot.** The reaction of the foot to pressure does not differ from that of the hand, but the continued pressure of the body weight on footwear produces a more severe and more obvious reaction, and any irregularity of bone becomes accentuated by the soft tissue reaction over it. The plantar skin is normally thickened over the weight-bearing areas of the sole, and thus abnormalities of weight distribution are shown on the sole by abnormal thickening of the skin, e.g. under the head of the second metatarsal in prolapse of the transverse arch. Such areas of skin are large; they gradually tail off into thinner skin and are related to obvious points of

pressure. Because of the rigidity of the epidermis, separation may occur between the dermis and epidermis with the formation of a hæmorrhagic blister, and this may at times be very painful. Treatment consists of balancing the foot by wedges or arch supports to relieve pressure over the affected area, and shaving off the thickened epidermis. The area is continually covered with an air-tight zinc strapping. Each time it is renewed, it removes some softened epithelium from the surface. The thickened skin can be completely removed in this way.

*Plantar callosities* are often confused with *plantar warts*. Because of the pressure on a plantar wart, it is driven into the sole of the foot, and thus becomes very painful. Being of infective origin, warts are likely to be multiple and bilateral. Areas of skin, not usually subject to excessive pressure are affected. A plantar wart assumes a more localised rounded form than a callus, and warts may occur in groups. Distinction in doubtful cases can usually be made by carefully paring the thickened skin with a razor blade. In a callosity, an evenly pink dermis is exposed, unless there has been epidermal separation. In a wart, a small circle with a whitened periphery is exposed, and this appearance is produced by dermal tissue around the margins of the in-driven wart. A plantar wart may be successfully eradicated by application of glacial acetic acid twice a day with a match-stick. Podophyllin powder or corn plasters may also succeed, but they are not always successful. The most satisfactory method for an established wart is eurette followed by carbolic acid of the base. Even this is not always successful, for recurrences may occur or small invisible warts may later enlarge.

The reaction of the foot to abnormal pressure is well demonstrated by the common *bunion*. The defence against pressure and friction first consists of thickening of the epidermis; there is then a thickening of subcutaneous fibrous tissue and the development in this of a distended lymphatic, which eventually becomes lined with secreting cells of a synovial type and forms an adventitious bursa. Deep to this there is more thickened fibrous tissue, forming in the case of a bunion part of the joint capsule, and then the metatarsal head or bony exostosis.

The bursa, communicating with lymphatics as it does, is liable to infection. When this occurs and pus discharges, a bursal fistula is likely to follow. The opening is liable to blockage which may be followed by recurrent inflammation. This story is particularly common with bunions. Mechanical irritation of a bursa is apt to be followed by an effusion, which eventually subsides. The appearance of the swelling is thus very variable. Pain can be relieved by stretching the shoes over the prominence, by appliances designed to relieve the pressure, or changed footwear, but the only really effective and permanent relief is provided by removal of the redundant skin, the bursa and the underlying cause of pressure. This operation is discussed in more detail later.

Other common prominences in the foot are : *Hammer Toes*.—These

may be congenital, due to malformation of the terminal interphalangeal joints, or acquired, when the proximal interphalangeal joints are more commonly affected. A bursa forms over the hyperflexed joint. *Enlarged Calcaneal Tuberosity.*—The lateral calcaneal tuberosity may be enlarged, particularly in girls, in whom irritation by excessively high-heeled shoes may play a part. A bursa forms over the prominence, and the overlying skin is liable to vascular changes. The tuberosity should be removed through a transverse incision; the insertion of the tendo Achilles may need partial reflection for removal to be complete. *Enlarged Base of the Fifth Metatarsal.*—This may be unduly large, or the irregularity may follow a fracture or epiphyseal injury. A seam in the shoe may cross it and irritate it. *The "Over-bone."*—This term is applied to a swelling on the dorsum of the foot over the articulation between the first metatarsal and the first cuneiform. It is due to bony hypertrophy resulting from excessive joint pressure. It may be seen either in *pes planus* or *pes cavus*. In removing it, the articulation is inevitably opened. *The Navicular Tubercle.*—This may ossify from a separate centre, and then is larger than normal. It may also be unduly tender. The swelling may be noted in late childhood. Operation is not indicated, and the provision of a valgus insole is usually sufficient to relieve pressure and pain. It may be more prominent than normal in a flat everted foot. *The Fifth Toe.*—This is commonly the site of tender callosities or corns. A congenital deformity in which the fifth toe overlies the fourth may contribute to the pressure on it. In severe cases, amputation may be tempting, but care must be taken that the point of pressure is not thereby transferred to the head of the fifth metatarsal; as a precaution this should always be shortened. Often a modified Keller's operation on the fifth toe is satisfactory (see under *hallux valgus*). A bursa on the dorsum of the metatarsophalangeal joint of the great toe is a sign of *hallux rigidus*. The toe being unable to bend in the take off in walking, is pressed against the seam of the toe cap. *Exostoses* may also appear in this region.

**Pain in the Hind-foot.** The causes of pain in this region are: (1) Enlargement and inflammation of a bursa over the lateral calcaneal tuberosity. (2) Subtaloid arthritis. (3) Plantar fasciitis. (4) Osteochondritis of the calcaneal epiphysis (*Serer's disease*). (5) Panniculitis of the heel pad. (6) Recurrent inversion sprain of the ankle due to rupture of the fibular collateral ligament of the ankle. (7) Tenosynovitis of the tendo Achilles. (8) Rupture of the tendo Achilles.

**Subtaloid Arthritis.** This follows injury to the subtaloid joint, most commonly after a fracture of the calcaneus. It may occur in a club-foot which has been difficult to correct, or in a foot deformity due to injury or poliomyelitis. The joint can be palpated below the fibular malleolus, where swelling and tenderness may be noted. Inversion and eversion of the heel are painful and restricted. Walking, particularly over rough ground, aggravates the condition. In mild cases the discomfort may be

relieved by wearing boots instead of shoes. Usually steady degeneration takes place, and arthrodesis of the joint becomes necessary. Because of the close association between movements at the subtaloid and mid-tarsal joints, mid-tarsal arthritis commonly accompanies the condition, and then a triple arthrodesis becomes necessary.

**Plantar Fasciitis.** Pain in the heel of obscure origin may occur in middle-aged people. In half the cases it is due to plantar fasciitis. The attachment of the plantar fascia to the calcaneus is tender. Radiologically localised rarefaction of the calcaneus or ossification of the fascial attachment may be noted. The formation of a plantar spur is common, although a spur alone, unless unusually large, is not by itself a cause of pain. Comparison with the radiograph of the opposite foot is often of interest, for a painless spur of similar size may be found. Treatment consists of short-wave diathermy, faradic foot baths, massage and exercises. This quickly clears up a large number of cases. If the pain is disabling and fails to respond, it can always be relieved by division of the plantar fascia at its attachment to the calcaneus.

In the remaining half of the cases of painful heel the pain is diffuse. The fatty plantar pad is tender both below and on the sides of the heel. The cause of the pain is obscure, and the term "panniculitis" which is applied to it is probably inaccurate. The response to treatment is uncertain. A sponge-rubber heel-pad may give relief, but the heel of the shoe should be lowered, otherwise the pad may increase pressure on the heel. The physiotherapeutic measures outlined for plantar fasciitis should be tried. Recovery occurs in several weeks or months.

**Rupture of the Tendo Achilles.** Rupture may be complete or partial. The usual cause is a sudden strain of the calf in a middle-aged man. He is seized with pain in the calf as though he had been struck with a stick, and then loses power in the leg. The rupture may occur at the musculo-tendinous junction, or in the lower part of the tendon. It is irregular, with a certain amount of fraying of the tendon (Fig. 599). Partial ruptures occur at the musculo-tendinous junction. If the patient is seen early, the gap between the ends of the tendon can be palpated, but it soon fills with blood, and if the patient is seen a week or ten days after the accident, it may be difficult to determine the lack of continuity, though thickening is usually present. Soft tissue radiography may be a help in showing the interruption of the tendon shadow. The loss of power in the calf, and the increase in dorsiflexion of the foot, combined with the local bruising and swelling, should make the diagnosis certain. Partial ruptures may be more difficult to diagnose, but power recovers rapidly and there is no lengthening of the tendon. Immediate operation and repair is the only satisfactory treatment, for any increase in length of the tendon impairs the power of the calf muscles.

**Pulled Calf Muscle.** This accident produces a feeling of being sharply struck in the calf. It is due to rupture of fibres of the soleus, usually on



the lateral side, and is followed by local tenderness and induration and later a hæmatoma at the lateral border of the muscle and down the leg. Treatment consists of a firmly applied supporting bandage, radiant heat, massage and exercises, with or without weight-bearing, according to the amount of pain produced.

**Pain in the Mid-tarsal Region.** This may be due to : (1) Tenosynovitis of the extensor tendons. (2) An inflamed bursa over an "overbone." (3) An enlarged and tender secondary centre for the navicular tubercle.



FIG. 599. Rupture of the tendo Achillis. The area of blood clot, lying between the frayed ends of the tendon, is well shown.

(4) Osteochondritis of the navicular bone in children of three to ten years of age. (5) Spasmodic flat-foot. (6) Mid-tarsal arthritis. (7) Foot strain.

**Osteochondritis of the Tarsal Navicular (Köhler's disease).** This is occasionally bilateral. It is most commonly encountered around the age of six years. The child complains of pain in the foot and walks with a slight limp. Local tenderness is found and radiologically there is flattening and increased density of the navicular, which is sometimes fragmented (Fig. 600). As in other cases of osteochondritis juvenilis, the disease runs a chronic course, and slow restitution of the normal bone outlines occurs in one to two years. There is no specific treatment.

**Mid-tarsal Arthritis.** This may follow injury or abnormal strains on

the joint. Inverting the fore-foot with one hand while steadying the heel with the other hand is painful. Local edema on the dorsum of the foot or palpable irregularity may be present. Minor cases are relieved by a valgus insole which takes the strain off the dorsum of the foot. In a few cases the support of a boot is advisable. In severe cases, or in those accompanied by subtaloid arthritis, arthrodesis is required. In acute episodes of pain, physiotherapy may give some relief.

Defective foot posture, getting up after an illness, or unusual activity on the feet may cause pain in the mid-tarsal region. The gradual onset of the pain, the history of a precipitating cause, and the commonly



FIG. 600. Osteochondritis juvenilis of the tarsal navicular.

associated loss of general muscular and physical tone, suggest the diagnosis. Response to adequate physiotherapy (described under metatarsalgia) confirms the diagnosis. Recurrent attacks suggest the need to correct the foot posture by altering the footwear, or by providing valgus insoles.

**Pain in the Fore-foot.** This is the most common site of pain in the foot and there are many causes. Amongst the most common are : (1) Anterior metatarsalgia. (2) Morton's metatarsalgia. (3) Osteo-arthritis of the metatarsophalangeal joint of the great toe associated with hallux rigidus, or hallux valgus and bunion. (4) March fracture (Chap. 105). (5) Sesamoid bruising and perisemoiditis. (6) Dorsal subluxation of the proximal phalanx (second and third toes). (7) Freiberg's disease of the second metatarsal head. (8) Corns, callosities and plantar warts.

**Anterior Metatarsalgia.** This may be encountered in an acute form in a foot with normal appearance, or more commonly in a chronic form with exacerbations, in patients with defective foot posture. It is more common in women than men. The most frequently associated defect is prolapse of the transverse arch, shown by : (a) Clawing of the toes (possibly an

the lateral side, and is followed by local tenderness and induration and later a hæmatoma at the lateral border of the muscle and down the leg. Treatment consists of a firmly applied supporting bandage, radiant heat, massage and exercises, with or without weight-bearing, according to the amount of pain produced.

**Pain in the Mid-tarsal Region.** This may be due to : (1) Tenosynovitis of the extensor tendons. (2) An inflamed bursa over an "overbone." (3) An enlarged and tender secondary centre for the navicular tubercle.



FIG. 599. Rupture of the tendo Achilles. The area of blood clot, lying between the frayed ends of the tendon, is well shown.

(4) Osteochondritis of the navicular bone in children of three to ten years of age. (5) Spasmodic flat-foot. (6) Mid-tarsal arthritis. (7) Foot strain.

**Osteochondritis of the Tarsal Navicular (Köhler's disease).** This is occasionally bilateral. It is most commonly encountered around the age of six years. The child complains of pain in the foot and walks with a slight limp. Local tenderness is found and radiologically there is flattening and increased density of the navicular, which is sometimes fragmented (Fig. 600). As in other cases of osteochondritis juvenilis, the disease runs a chronic course, and slow restitution of the normal bone outlines occurs in one to two years. There is no specific treatment.

**Mid-tarsal Arthritis.** This may follow injury or abnormal strains on

or sometimes by bunching the metatarsals together. The only curative treatment is removal of the neuroma through a short incision in the sole.

**Hallux Rigidus.** It is characteristic of hallux rigidus that the big toe is straight. If the toe were deviated as in hallux valgus it would not be forced into dorsiflexion on "taking off" in walking. It is the forcing of the rigid toe to bend that produces the pain. Pressure on the toe cap produces a callosity and often a bursa over the dorsum of the first metatarsophalangeal joint (Fig. 602). The thickening at this site is diagnostic of the condition. It is due to osteo-arthritis of the first metatarsophalangeal joint, resulting in flattening of the articular surfaces and limitation of movement. The condition may be seen at varying stages in development, and it may produce symptoms long before the toe is actually rigid.

FIG. 602. The classical pressure area on the dorsum of the head of the first metatarsal in hallux rigidus.



Radiologically there is narrowing of the joint space, sclerosis of the bone ends, and a varying degree of marginal exostosis. Complete relief is given by an arthroplasty of the Keller type (see under hallux valgus). Temporary relief may be given in milder cases by reducing the degree of dorsiflexion of the toe in walking; this is done by placing a rounded leather rocker in between the two layers of the sole of the shoe, just behind the line of the metatarsal heads, so that the fore-part of the foot can roll over the rocker for a few degrees before the toes are forced into dorsiflexion.

**Hallux Valgus.** The primary cause of hallux valgus remains unknown. Although hallux valgus is less common in the unshod races, shoes have little to do with its causation, but they may aggravate a natural tendency for the toe to deviate. Heredity plays a part. The condition is more commonly seen in women than men. The great toe is displaced *laterally*, often with a certain amount of rotation (Fig. 603). The first metatarsal is displaced *medially* (metatarsus primus varus), and the first intermetatarsal "space" increased in width. The prominence of the metatarsal head produces a pressure point which is protected by the formation of a bursa and thickening of the tissues—commonly described as a bunion. The sesamoids are dragged laterally by their soft

attempt to lift up the metatarsal head) ; (b) loss of the normal fatty pad under the metatarsal heads, which are consequently readily palpable ; and (c) callosities, which are usually situated under the heads of the second and third metatarsals. The whole of the forepart of the foot is tender, and it is painful when manipulated. Movement of the toes on the metatarsal heads may be very uncomfortable. Oedema over the metatarsals is common. Rest relieves the pain, and activity aggravates it. Changes of footwear may relieve or accentuate it.

Acute metatarsalgia with normal foot posture is relieved rapidly by a course of faradic foot baths, massage and exercises. Chronic metatarsalgia with an associated deformity is often equally relieved, but relapses occur. An attempt to correct the foot posture and to balance the pressures on the fore-foot should be made. Among the simplest of these measures is a metatarsal pad, which should lie behind the metatarsal



FIG. 601. Typical neuroma removed in Morton's metatarsalgia.

heads to take the pressure off them. It may be made of a piece of zinc oxide felt, or consist of a pad attached to a garter. When it is necessary to correct the position of the longitudinal arch as well, the metatarsal pad may be combined with a valgus insole. Particular attention should be paid to the footwear. No appliances are effective if they are not based on sound leather-soled lace-up shoes. The height of the heel is important in women. If the patient is used to low heels, raising the heel will take pressure off the fore-part of the foot. The heel must not, however, be raised so high that the foot slips forward on to the metatarsals ; the average height used is that of the Oxford or Cuban heel, namely,  $1\frac{1}{2}$  inches.

**Morton's Metatarsalgia.** This comparatively uncommon form of metatarsalgia is distinguished by the sudden and severe pain which arrests the patient. After a few minutes, walking can be resumed, but another attack is likely to follow. Spontaneous night pain may also occur. The pain is related to the interdigital cleft and the adjacent surfaces of the third and fourth toes. Examination shows impairment of sensation. It is due to the formation of a neuroma on the corresponding plantar digital nerve supplying this area (Fig. 601). Thrombosis of the accompanying plantar vessels occurs, and may be the primary cause of the disorder. The pain can be reproduced by pressure over the neuroma,

or sometimes by bunching the metatarsals together. The only curative treatment is removal of the neuroma through a short incision in the sole.

**Hallux Rigidus.** It is characteristic of hallux rigidus that the big toe is straight. If the toe were deviated as in hallux valgus it would not be forced into dorsiflexion on "taking off" in walking. It is the forcing of the rigid toe to bend that produces the pain. Pressure on the toe cap produces a callosity and often a bursa over the dorsum of the first metatarsophalangeal joint (Fig. 602). The thickening at this site is diagnostic of the condition. It is due to osteo-arthritis of the first metatarsophalangeal joint, resulting in flattening of the articular surfaces and limitation of movement. The condition may be seen at varying stages in development, and it may produce symptoms long before the toe is actually rigid.

FIG. 602. The classical pressure area on the dorsum of the head of the first metatarsal in hallux rigidus.



Radiologically there is narrowing of the joint space, sclerosis of the bone ends, and a varying degree of marginal exostosis. Complete relief is given by an arthroplasty of the Keller type (see under hallux valgus). Temporary relief may be given in milder cases by reducing the degree of dorsiflexion of the toe in walking; this is done by placing a rounded leather rocker in between the two layers of the sole of the shoe, just behind the line of the metatarsal heads, so that the fore-part of the foot can roll over the rocker for a few degrees before the toes are forced into dorsiflexion.

**Hallux Valgus.** The primary cause of hallux valgus remains unknown. Although hallux valgus is less common in the unshod races, shoes have little to do with its causation, but they may aggravate a natural tendency for the toe to deviate. Heredity plays a part. The condition is more commonly seen in women than men. The great toe is displaced *laterally*, often with a certain amount of rotation (Fig. 603). The first metatarsal is displaced *medially* (metatarsus primus varus), and the first intermetatarsal "space" increased in width. The prominence of the metatarsal head produces a pressure point which is protected by the formation of a bursa and thickening of the tissues—commonly described as a bunion. The sesamoids are dragged laterally by their soft

tissue attachments; at least one comes to lie in the intermetatarsal "space" (Fig. 604). Osteo-arthritic changes occur in the joint. A line forms on the head of the metatarsal indicating the area in contact with the base of the phalanx. Marginal exostoses form. The line of pull of the tendons is altered and their action tends to increase the deformity. This may be so marked that the toe lies at right angles to the metatarsal; secondary pressure effects then arise on the other toes.

The patient presents with pain. From the practical point of view it is due to: (a) Pressure over the bunion and consequent inflammation; joint movements are free and painless; or (b) arthritic changes in the joint, accompanied by a bunion, which itself may, however, be responsible



FIG. 603. Severe bilateral hallux valgus and bunions.

for some of the pain. Exclusion of the joint as a major contributory cause of pain has an important bearing on treatment.

*Treatment.* In mild cases it may be useful in acute exacerbations to pad the bunion with a ring of felt. For long-term support a valgus and metatarsal pad, with a prolongation lying along the inner border of the foot above the bunion is more efficient. This appliance gives relief for many years. When conservative methods fail, operation is indicated. If the bunion only is the cause of pain, it is removed together with the bursa, exostoses and the prominent part of the metatarsal head. When the predominant cause of pain is the arthritis, the alternatives are *arthrodesis* or *arthroplasty*. Although the position of the toe is improved by arthroplasty which shortens the toe it will not be much improved, and therefore if the big toe is overlying the others, or the patient expresses a wish for a straight toe, arthrodesis is indicated. There is a choice of two methods of arthroplasty. In *Küller's operation*, the proximal half of the phalanx and the prominent portion of the metatarsal head are excised (Fig. 604), together with the exostoses. A false joint forms. Although it takes time for the flexor tendons to shorten and to function again, the patient can walk on the heel in a slipper a few days after the operation. In *Mayo's operation* the head of the metatarsal is excised, but this requires care, for if too much is excised, the sesamoids

are deprived of support and pain may return. A longer convalescence is also required after this operation.

If *arthrodia* of the toe is to be carried out it must be fixed in 35 degrees of dorsiflexion and a slight abduction. After removing the joint cartilage and exostoses, the desired position of the toe is maintained by transfixion with a screw or Kirschner wire. Support is needed for six weeks when fusion is firm and is best provided by a walking plaster and overboot.

**Sesamoid Lesions.** Fractures of the sesamoids may occur and lead later to arthritic changes; these fractures are liable to be confused with the occasional double centre of ossification for the medial sesamoid. Pain may occur in the area of the sesamoids after excessive use of the feet (e.g. walking on soft sand), or after landing heavily on the feet. Spontaneous pain in the sesamoids in young people is encountered, accompanied by local swelling and tenderness; the cause is obscure, but the condition clears up with rest. The use of a metatarsal pad and garter is of value in relieving pressure under the toe.

#### **Dorsal Dislocation of the Second Toe.**

In deformed feet resulting from severe hallux valgus, or following injury or rheumatic infection, prolapse of the second metatarsal head commonly occurs and is indicated by the flattened transverse arch, the palpable metatarsal head and the overlying callosity. Eventually dorsal subluxation of the phalanx may take place with the development of increased pain. The prominent base of the phalanx may be



FIG. 604. Keller's operation. The line "A" indicates the level of section of the proximal phalanx and the line "B" the amount of the prominent metatarsal head removed. Note the coexistent subungual exostosis.

palpable on the dorsum of the foot. Reduction is unstable. The best treatment, therefore, is excision of the base of the phalanx, and correction of the footwear to balance the pressure on the foot.

**Freiberg's Disease of the Second Metatarsal Head.** This is an uncommon variety of osteochondritis juvenilis which affects the epiphysis of the metatarsal head and results in its becoming flattened. There is consequent interference with movement of the joint which eventually develops arthritis. In a few cases, a fragment of the head separates as in osteochondritis dissecans, and in such cases it may be difficult to tell which is the primary condition. When pain is unrelieved by physiotherapy, or when there is great enlargement of the metatarsal head, the latter should be excised.



**Painful Lesions of the Toes. Hammer Toe.**—In the common variety the proximal interphalangeal joint is hyperflexed, and the distal one extended. A double lesion may, however, occur with flexion of both interphalangeal joints; pressure occurs on both joints with the development of a painful double callosity. Prolapse of the transverse arch; pes cavus; excessively long toes; injury; or congenital malformations of the interphalangeal joints, particularly of the terminal joint, may be the causal factor. Treatment consists of excision of the bursa through a transverse elliptical incision and fixation of the joint in extension by a hole and spike arthrodesis. Fixation of the distal interphalangeal joint is less satisfactory; firm fibrous union after excision may be sufficient, but if bony arthrodesis is the goal, the joint should be transfixed temporarily by a Kirschner wire.

**Subungual Exostosis** (Fig. 604). This lifts up the nail and may become ulcerated. It is painful, and the patient can only be relieved by removal of the nail and the exostosis. Usually there is sufficient nail bed to prolapse over the gap, or to suture over it.

**Onychogryphosis.** In this condition the nail becomes thickened and difficult to trim, so that it may eventually grow to resemble a horn. The etiology is obscure. If persistent irritation were the only cause the condition might be expected to be limited to the great toe, but although it is most frequently encountered here, all the toes may be affected (Fig. 605). Recurrence follows removal of the nail, and although this may allow the new nail to be kept in reasonable shape by soaking and trimming with bone forceps, the most satisfactory treatment is excision of the nail bed (*vide infra*).

**Ingrowing Toe Nail.** The primary cause of this condition is the abnormal relation of the nail margin to the nail fold, so that the nail presses into the fold and produces ulceration, which, if followed by infection, produces a lateral *paronychia*. This may become chronic with granulomatous proliferation. The cause of the nail deformity is variable. Hypercurved nails lead to it, and are congenital in origin. It may follow an acute lateral *paronychia*. Deformities of the pulp of the toe and excessively long toes may be factors in its occurrence. Defective foot hygiene and tight footwear help to cause acute attacks of infection.

The infection should first be cleared up, and this usually demands removal of the nail, in order to obtain effective drainage. After one attack of inflammation it is reasonable to wait for a second attack before excision of the nail bed is recommended, unless there is a severe nail deformity, when immediate excision of the nail bed may be undertaken. The nail bed extends from the insertion of the extensor tendon to the edge of the lunula, and passes down on the lateral margins of the phalanx for a short distance. It is essential to remove these deep corners if there is not to be regrowth of small troublesome isolated fragments of the nail bed. A tourniquet is applied and the nail is removed; two oblique incisions

are made from the angles of the nail bed, and the quick of the nail is reflected back. The nail bed is then excised down to bone. When this has been done, one of two procedures may be carried out. The lateral incisions may be closed with sutures and the quick allowed to lie in contact with the subungual tissue. Healing occurs, leaving a "blind" nail bed which gradually epithelialises. A toe of normal appearance is thus preserved. If it is desired to shorten the toe and to avoid leaving any nail bed, the distal part of the terminal phalanx is nibbled away to permit approximation of the "quick" and the skin of the pulp. This flattens the end of the toe, and thus produces an ugly "finish."



FIG. 605. Severe onychogryphosis.

**Severe Foot Deformities.** These may be the result of congenital malformations, infective arthritis, injury, paralysis, or rheumatoid arthritis. In such cases it is only exceptionally that corrective surgery is indicated to obtain a plantigrade foot. If the foot is already plantigrade, removal of prominences which are bound to rub any kind of footwear is justified, e.g. amputation of overlying toes, or excision of a bunion. In general, the only satisfactory treatment is the provision of orthopædic shoes or boots. In these, the insole is shaped to fit the foot, and covered with a resilient material, such as sponge rubber. The height of the heel is regulated to suit the balance of pressures. The toecap should be soft to avoid rubbing. The tongue of the shoe may be padded to avoid pressure on the dorsum of the foot after lacing the shoe. Boots may be used instead of shoes when support of the ankle or mid-tarsus is needed. The

**Painful Lesions of the Toes. Hammer Toe.**—In the common variety the proximal interphalangeal joint is hyperflexed, and the distal one extended. A double lesion may, however, occur with flexion of both interphalangeal joints; pressure occurs on both joints with the development of a painful double callosity. Prolapse of the transverse arch; pes cavus; excessively long toes; injury; or congenital malformations of the interphalangeal joints, particularly of the terminal joint, may be the causal factor. Treatment consists of excision of the bursa through a transverse elliptical incision and fixation of the joint in extension by a hole and spike arthrodesis. Fixation of the distal interphalangeal joint is less satisfactory; firm fibrous union after excision may be sufficient, but if bony arthrodesis is the goal, the joint should be transfixed temporarily by a Kirschner wire.

**Subungual Exostosis (Fig. 604).** This lifts up the nail and may become ulcerated. It is painful, and the patient can only be relieved by removal of the nail and the exostosis. Usually there is sufficient nail bed to prolapse over the gap, or to suture over it.

**Onychogryphosis.** In this condition the nail becomes thickened and difficult to trim, so that it may eventually grow to resemble a horn. The aetiology is obscure. If persistent irritation were the only cause the condition might be expected to be limited to the great toe, but although it is most frequently encountered here, all the toes may be affected (Fig. 605). Recurrence follows removal of the nail, and although this may allow the new nail to be kept in reasonable shape by soaking and trimming with bone forceps, the most satisfactory treatment is excision of the nail bed (*vide infra*).

**Ingrowing Toe Nail.** The primary cause of this condition is the abnormal relation of the nail margin to the nail fold, so that the nail presses into the fold and produces ulceration, which, if followed by infection, produces a lateral paronychia. This may become chronic with granulomatous proliferation. The cause of the nail deformity is variable. Hypercurved nails lead to it, and are congenital in origin. It may follow an acute lateral paronychia. Deformities of the pulp of the toe and excessively long toes may be factors in its occurrence. Defective foot hygiene and tight footwear help to cause acute attacks of infection.

The infection should first be cleared up, and this usually demands removal of the nail, in order to obtain effective drainage. After one attack of inflammation it is reasonable to wait for a second attack before excision of the nail bed is recommended, unless there is a severe nail deformity, when immediate excision of the nail bed may be undertaken. The nail bed extends from the insertion of the extensor tendon to the edge of the lunula, and passes down on the lateral margins of the phalanx for a short distance. It is essential to remove these deep corners if there is not to be regrowth of small troublesome isolated fragments of the nail bed. A tourniquet is applied and the nail is removed; two oblique incisions

## SECTION XII

### INJURIES TO JOINTS AND BONES

THE HAND  
by  
J. G. BONNIN

BURNS  
by  
VALENTINE A. J. SWAIN

#### CHAPTER 91

#### INJURIES TO JOINTS

JOINTS may appear to be weaker than bones, but in fact they are usually stronger, for they may be protected by bony overhang, as in the hip or the elbow, or by strong ligaments as in the knee, or a combination of both as in the ankle. The shoulder, however, relies on its muscular cuff (the short rotator cuff) for stability and mobility, and as the cuff and the relatively weak ligaments are easily torn or stretched, the shoulder is the most commonly dislocated joint. Dislocations are grouped as follows : (1) *Traumatic Dislocation*.—This results from sudden severe force acting through the bone, and producing persistent displacement of the joint surfaces relative to each other. This may lead to : (2) *Recurrent Dislocation*.—The capsule of the joint is so stretched that a relatively trivial injury or unusual movement produces recurrence of the dislocation. This in turn may lead to : (3) *Habitual Dislocation*.—Dislocation occurs readily with certain movements, and can usually be reduced by another special movement which the patient soon learns.

Severe strains of joints may produce dislocation (luxation), subluxation or injury of individual ligaments, of various degrees of severity. In subluxation, the capsule of the joint is stretched or torn, but not sufficiently to permit persistent displacement of the bone surfaces on each other, and although the capsular damage may be severe in subluxation, there is less risk of recurrent dislocation than after complete luxation; the physical signs of the joint injury may resemble those of dislocation, but the radiograph is usually normal. Similar findings, however, characterise a dislocation which has become reduced spontaneously. Thus there

use of an iron and T-strap may be required to correct an inversion or eversion deformity.

**Gout (Podagra).** This is now a rare disease. It is almost confined to men, and shows hereditary tendencies. Crystals of sodium biurate are deposited around the small joints of the toes ; the metatarsophalangeal joint of the big toe is the most commonly affected. Pain comes on suddenly and is intense ; jarring gives excruciating spasms of pain. The toe becomes hot, red, swollen and tense. A general febrile disturbance may accompany the local manifestations. Attacks last one to two weeks, and then slowly subside. Recurrent attacks result in disorganisation of the joint. The treatment is medical.

swelling has had time to appear. Methods are employed to reduce the swelling ; the first is elevation of the part, and the second application of a firm bandage which should provide even but not constrictive pressure. A crepe bandage applied over cotton wool is satisfactory, although a Robert Jones' bandage (a layer of cotton wool and then of bandage, another of cotton wool and a further of bandage) is better. The two bandages increase the support of the dressing considerably, so that it acts as a soft splint, and the two layers of cotton wool, if the first layer is not too compressed leave room for expansion. Thirdly, an ice bag may be used. This simple remedy is not to be despised as a method of reducing pain and swelling. Use powdered ice in water to avoid frost-bite. After twenty-four hours, the second stage of the local reaction to the injury occurs and is characterised by vasoconstriction, which results in persistence of the hæmatoma and swelling. Treatment should now be changed to methods which produce vasodilatation and thus hasten reduction of the swelling. Heat may be applied in one of many forms, e.g. radiant heat, electrical pads, or hot compresses. Massage, beginning with effleurage, and gentle exercise of muscles or tendons crossing the joint aid in reducing the swelling. At this stage, an injection of procaine may be used to produce vasodilatation, and to relieve pain so that massage and movement may be made more comfortable. It may be used as an emergency measure to permit activity on a sprained joint, but increased swelling occurs later. A supporting bandage of an elastic type is needed during active treatment and for some days afterwards. Partial rupture of a ligament produces more severe effects, including a larger hæmatoma, and should be treated in the same way, but progress is slower, and treatment more prolonged. In certain situations, such as the ankle, and especially if the injury is associated with other ligamentous injuries or with a fracture, a short period of rest in plaster during the phase of absorption or recovery may be necessary.



FIG. 607. Strain radiograph of a partial rupture of the fibular collateral ligament of the ankle. The talus is tilted 25 degrees due to tearing of the calcaneo-fibular part of the ligament.

Complete rupture of a ligament renders the joint unstable and is usually best treated by suture of the ligament. This is always necessary in such injuries of the knee, and often necessary in those of the ankle

is a danger of being misled by a normal X-ray. Careful clinical examination is necessary, often with the aid of general anaesthesia which both facilitates the examination and permits check radiography to be carried out under strain (*vide infra*).

Injuries of individual ligaments produce less severe signs of joint damage; they also are usually associated with a normal radiograph. Movement of the joint is affected in one particular direction rather than in all directions, and one particular ligament is tender, and painful on being stretched. Injuries of ligaments are classified as follows: (1) *Strain*.—The ligament is overstretched, but not beyond its capacity to

return to its normal length. There is local oedema and tenderness, perhaps associated with a slight effusion into the joint and a little stiffness. (2) *Sprain*.—The ligament is stretched beyond its capacity to return immediately to normal. There is rupture of intra-ligamentous fibres, but the peri-ligamentous tissues are relatively intact (Fig. 606). There is severe local swelling and oedema, and some bruising. The ligament is tender, and painful when stretched, but the stability of the

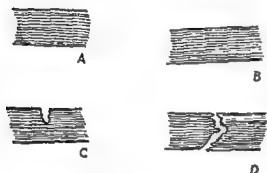


FIG. 606. Ligamentous injury. A. The normal ligament. B. A sprain of a ligament showing intraligamentous rupture of fibres. C. Partial rupture of a ligament. D. Complete rupture of a ligament.

joint is unaffected, though there is a small effusion and limitation of mobility. (3) *Partial Rupture of a Ligament* (often called a "severe sprain").—Some fibres of the ligament are torn, together with periligamentous tissues, and in consequence there is severe bruising and a blood-stained synovial effusion. All the signs of a sprain are accentuated. The joint is still stable on clinical examination. (4) *Complete Rupture of a Ligament*.—There are signs of hæmorrhage, often sufficient to suggest a fracture; there is a hæmorrhagic effusion into the joint and the joint is unstable, due to the rupture of the ligament. This can be proved by "strain" radiography (Fig. 607).

**Treatment.** Simple measures are all that are needed for a strain. Local pain may be relieved by application of a compress of *lotio plumbi cum opio*, or inunction of a liniment (e.g. *linimentum terebinthinæ*). Rest and a supporting bandage are occasionally necessary for a few days. The reaction to a sprain occurs in two stages. In the first, there is vasodilatation and swelling with increasing pain due to increased tissue tension. Application of heat is inadvisable as it increases the swelling and therefore the pain. Firm strapping is also inadvisable, as it may not relax sufficiently with increasing swelling and may thus cause increased pain. Furthermore, adhesive plaster should not be applied before

humerus is inverted between the anterior and posterior parts of the capsule, and muscle spasm holds it against the glenoid with the arm fixed above the head (very rare).

*Physical Signs.* (1) The function of the shoulder is gravely impaired ; the patient usually holds the wrist of the affected side with the other hand. (2) Flattening of the deltoid contour. (3) The head of the humerus may be felt in an abnormal position, and the fact that it is the head may be confirmed by rotating the humerus. (4) There is a feeling of emptiness below the acromion. (5) The patient cannot place the elbow against the ribs (Dugas' sign). (6) Hamilton's ruler test : A ruler can be laid against the lateral epicondyle and the acromion ; this is normally impossible due to the bulge of the humeral head. (7) Altered length of the arm, e.g. increased in subglenoid dislocation. (8) When the examiner moves the shoulder the normal feeling of easy gliding is replaced by a sensation of elastic mobility and pain is produced.

*Treatment.* The chief difficulty in dislocation of the shoulder is to overcome muscle spasm. The complicated classical manoeuvres were developed in order to exert sufficient force to do this, in the days before general anaesthesia. Nowadays, with the patient completely relaxed it is usually sufficient to pull gently downwards on the arm and manipulate the head of the humerus back through the tear in the inferior part of the joint capsule with the fingers of the other hand. Other methods involving the use of force are dangerous ; this is particularly true of Kocher's method which may cause damage to the brachial plexus (*vide infra*). Because of this risk, it is essential that a neurological examination be made before manipulation, for otherwise damage to the plexus may be erroneously attributed to the reduction. The nerve most often injured by the dislocation is the axillary (circumflex) nerve, with resultant paralysis of the deltoid muscle whereas this is the nerve least likely to be injured by manipulation. The medial and posterior cords of the plexus are more likely to suffer. *The Hippocratic Method.*—The unshod heel of the surgeon is placed in the axilla and used for counter-traction and as a fulcrum over which the head of the humerus can be levered outwards by adducting the arm. *Kocher's Manoeuvre.*—This makes use of the forearm as a powerful lever. After a short period of traction on the shoulder, the forearm is externally rotated with the elbow at the side to stretch the subcapularis. The elbow is then adducted and the hand circumducted across the patient's face to the opposite side, when reduction usually occurs.

*After-treatment.* It is questionable whether the extent of the period of after-treatment affects the risk of recurrent dislocation. If the capsule is widely torn and the head of the humerus dented, recurrent dislocation will probably occur regardless of the length of the original immobilisation. A week in a sling with the arm held to the side and then the gradual use of the shoulder, discarding the sling in the third week, is sufficient.



joint, and in rupture of the rotator cuff of the shoulder. In some instances, such as rupture of the orbicular ligament of the head of the radius, repair is very seldom required.

**Chronic Sprain.** Sometimes, especially in partial rupture of a ligament when the injury has been inadequately rested and treated, persistent pain and swelling may occur, and is sometimes accompanied by vasomotor changes over the tender ligament. Such cases are slow to respond to the usual massage, radiant heat and exercises, but may be helped by short-wave diathermy, or local infiltration with hydrocortisone or procaine. Occasionally manipulation may be needed.

**Complications of Dislocations.** (1) *Fracture.*—(a) Of the shaft of the bone. This renders reduction of the dislocation difficult. The temporary insertion of a spike into the displaced end of the bone while manipulation is undertaken may help. Open operation may, however, be needed. (b) Of parts of the bones in or close to the joint. These may consist of marginal depressed or avulsed fragments. They are usually reduced at the same time as the dislocation, but occasionally require open fixation. (c) Through the cartilaginous surface of the joint. The fragment may become detached rapidly as a loose body in the joint, or it may take a long time before the hinge of cartilage attaching the fragment is broken (see osteochondritis dissecans). (2) *Rupture of Muscular Attachments.*—This is commonly seen at the shoulder where partial or complete rupture of the short rotator cuff seriously affects joint function. (3) *Rupture of ligaments with joint instability*, e.g. the cruciate ligaments of the knee, or the fibular collateral ligament of the ankle. (4) *Involvement of Blood Vessels.*—(a) Pressure interfering with the circulation. (b) Actual rupture with hæmatoma formation. (5) *Injury to Nervous Structures.*—E.g. damage to the ulnar nerve in dislocation of the elbow, or to the spinal cord in dislocations of vertebræ. (6) Recurrent and habitual dislocation (*vide supra*).

**Dislocation of the Shoulder.** In the shoulder, stability is sacrificed for mobility, and the head of the humerus, supported only by a muscular cuff, is easily displaced from the shallow glenoid cavity. The shoulder is the most frequently dislocated joint. Abduction must normally be accompanied by rotation of the humerus; thus when leverage is applied to the arm so rapidly that this cannot occur dislocation results. Most dislocations of the shoulder usually begin with leverage of the head out of the glenoid cavity through the lower weak part of the joint capsule, and this is followed by a varying degree of further displacement in other directions (*vide infra*). Muscle spasm also plays a part, for the condition is particularly common in epileptics who fall. The final positions into which the head of the humerus is displaced are: (1) *Subglenoid.*—This is rare for the position is unstable; the head of the humerus quickly becomes subcoracoid. (2) *Subcoracoid.*—This is the most common position encountered. (3) *Posterior.* (4) *Luxatio Erecta.*—The head of the

particularly if early movement is not insisted on. There is limitation of all movements of the shoulder; sometimes very little movement remains. Pain may be troublesome. In serious cases, the pain eventually subsides, but movements are permanently limited.

**Posterior Dislocation.** Though this is a rare accident, it merits mention, as it is so commonly overlooked in the first instance. This unfortunate error is due to the absence of severe deformity of the shoulder in fat people, and the close resemblance of the anteroposterior X-ray to the normal (Fig. 608). The important physical signs are: (a) Inability to get the elbow to the side; (b) complete loss of external rotation; and (c) the forearm is held across the chest. The diagnosis is confirmed by a lateral X-ray (Fig. 609). Reduction is easy under general anaesthesia, but retention is difficult and dislocation tends to recur. The secret of effective retention is to maintain the shoulder in the abducted and externally rotated position for the first weeks and then gradually to bring it to the side. This may be done on an abduction splint, but is more safely carried out in bed with the hand suspended vertically.

**Recurrent Dislocation of the Shoulder.** Whether anterior or posterior, the recurrence is due to stretching and tearing of the capsular attachments; thus anteriorly, the joint cavity becomes continuous with the subscapular bursa. The ease with which the head slips out of the glenoid cavity is greatly increased if there is a dent in the humeral head. The logical method of treatment is to reattach the capsule to the rim of the glenoid cavity (Bankart's operation). Technically, this is difficult, and, furthermore, the sutures inserted through the margin of the glenoid cavity encroach on the joint surface. Fibrosis and a reduction in capsular laxity may be produced anteriorly by plicating the tendon of subscapularis (Putti-Platt operation). This method is effective, but it causes some loss of external rotation at the shoulder. The simplest and most effective operation is the



FIG. 610. The position for the anterior bone-block inserted for recurrent anterior dislocation of the shoulder. A posterior bone-block may be used for posterior recurrent dislocations.

insertion of a bone block on the neck of the scapula just medial to the glenoid rim (labrum glenoidale), so deepening the socket (Fig. 610). The presence of the subscapular bursa soon results in freedom of movement over the bone block and restoration of full external rotation.

**Dislocations of the Clavicle. Acromial End.**—Minor strains of the acromioclavicular joint may occur, producing local tenderness and pain on elevating the shoulder. The strength of the articulation depends on the

Paralysed muscles are treated according to the principles enunciated under *The Peripheral Nerves* (Chap. 61). If the shoulder has remained dislocated for four weeks, closed methods of reduction are not likely to succeed and open operation will be needed.

**Complications.** (1) Groove fracture of the humeral head. In either anterior or posterior dislocations, the head of the humerus is liable to be deeply dented by the pressure of the glenoid margin. Such a depression facilitates the development of recurrent dislocation of the shoulder. (2) Rupture of the short rotator cuff. If complete, this produces a totally unstable shoulder which very readily redislocates. More commonly, the rupture is partial, and small ruptures, though at first interfering with abduction, may recover. Larger ruptures prevent stabilisation of the humeral head in the glenoid cavity on abducting the arm, and abduction is therefore accompanied by rotation of the scapula; the deltoid is unable to assist. Thus only about 45 degrees of active abduction can be achieved. Full passive abduction can be obtained (c.f. degenerative tendinitis and subacromial bursitis in which passive abduction is limited) and often when obtained can be maintained by the patient. Operative repair of the torn capsule is required. (3) Degenerative tendinitis, and subacromial bursitis. In elderly people, degenerative changes in the short rotator cuff are common. These may be produced or aggravated by injury,



FIG. 608.



FIG. 609.

FIG. 608. Anteroposterior radiograph of a posterior dislocation of the shoulder. The appearance closely resembles the normal, but the glenoid overlap is slightly increased and the humeral head is over-rotated internally, so that its normal hemispherical appearance is lost. This injury is thus easily overlooked.

FIG. 609. The lateral view of the shoulder in abduction in posterior dislocation shows the head of humerus caught on the posterior margin of the glenoid cavity.

particularly if early movement is not insisted on. There is limitation of all movements of the shoulder; sometimes very little movement remains. Pain may be troublesome. In serious cases, the pain eventually subsides, but movements are permanently limited.

**Posterior Dislocation.** Though this is a rare accident, it merits mention, as it is so commonly overlooked in the first instance. This unfortunate error is due to the absence of severe deformity of the shoulder in fat people, and the close resemblance of the anteroposterior X-ray to the normal (Fig. 608). The important physical signs are: (a) Inability to get the elbow to the side; (b) complete loss of external rotation; and (c) the forearm is held across the chest. The diagnosis is confirmed by a lateral X-ray (Fig. 609). Reduction is easy under general anaesthesia, but retention is difficult and dislocation tends to recur. The secret of effective retention is to maintain the shoulder in the abducted and externally rotated position for the first weeks and then gradually to bring it to the side. This may be done on an abduction splint, but is more safely carried out in bed with the hand suspended vertically.

**Recurrent Dislocation of the Shoulder.** Whether anterior or posterior, the recurrence is due to stretching and tearing of the capsular attachments; thus anteriorly, the joint cavity becomes continuous with the subscapular bursa. The ease with which the head slips out of the glenoid cavity is greatly increased if there is a dent in the humeral head. The logical method of treatment is to reattach the capsule to the rim of the glenoid cavity (Bankart's operation). Technically, this is difficult, and, furthermore, the sutures inserted through the margin of the glenoid cavity encroach on the joint surface. Fibrosis and a reduction in capsular laxity may be produced anteriorly by plicating the tendon of subscapularis (Putti-Plattoperation). This method is effective, but it causes some loss of external rotation at the shoulder. The simplest and most effective operation is the

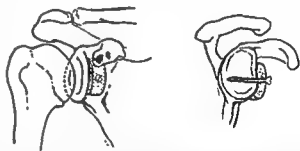


FIG. 610. The position for the anterior bone-block inserted for recurrent anterior dislocation of the shoulder. A posterior bone-block may be used for posterior recurrent dislocations.

insertion of a bone block on the neck of the scapula just medial to the glenoid rim (labrum glenoidale), so deepening the socket (Fig. 610). The presence of the subscapular bursa soon results in freedom of movement over the bone block and restoration of full external rotation.

**Dislocations of the Clavicle.** *Acromial End.*—Minor strains of the acromioclavicular joint may occur, producing local tenderness and pain on elevating the shoulder. The strength of the articulation depends on the

conoid and trapezoid ligaments attaching the bone to the coracoid process. Injuries which cause a little upward shift of the acromial end of the clavicle are due to rupture of the joint capsule only and are called subluxations. Injuries which cause wide displacement of the clavicle are due to rupture of the conoid and trapezoid ligaments and are true dislocations (Fig. 611). Maintenance of reduction is extremely difficult without some form of internal fixation. Fortunately there is little disability following an unreduced subluxation; and even following complete dislocation, function may be good though the cosmetic appearance is poor. The best form of



Fig. 611. Complete dislocation of the acromioclavicular joint, always accompanied by rupture of the conoid and trapezoid ligaments.

immediate retention is provided by strapping which is designed to take the weight of the arm off the outer end of the clavicle. A pad is placed over the root of the neck and inner end of the clavicle, leaving the acromioclavicular joint free. A second pad is placed under the olecranon. The application of strapping is begun on the back, then continued over the neck pad, under the flexed elbow, over the neck pad again, and ends over the front of the chest. The region of the acromioclavicular joint is left uncovered so that it can be examined. Careful supervision is necessary and re-tightening of the strapping by further application of strapping over the original band is

carried out when required. Reduction must be maintained in this way for two weeks in subluxation and for four weeks in complete dislocation. This treatment is apt to be followed by stiffness of the shoulder. Therefore, open reduction is favoured, and is accomplished by inserting a wide-thread screw through the outer third of the clavicle into the base of the coracoid process. If the hole in the clavicle is deliberately made larger than the width of the screw and the screw head guarded by a washer, some activity of the shoulder may be permitted and stiffness is thus avoided. The screw is usually removed after two to three months.

**Sternal End.** The anterior end of the first rib, lying below the sternal end of the clavicle, prevents downward dislocation of the sternal end of the clavicle, and the strong rhomboid ligament makes dislocation in other directions uncommon. Anterior subluxation is most commonly encountered, and, as it causes little disability and is impossible to retain, is best left untreated. Posterior dislocation is rare, but is of interest for it may cause alarming pressure on the trachea and great vessels. Upward

dislocation produces an unsightly cosmetic defect. Reduction of both these dislocations may be accomplished by drawing the patient's shoulders backward against the bent knee with the patient sitting. Reduction is difficult to maintain and is therefore liable to remain incomplete. Either a figure-of-eight bandage, as for clavicular fractures, may be used, or the patient may lie with a sandbag between the shoulders.

**Dislocation of the Mandible.** This may be unilateral or bilateral, and if it results from injury may be complicated by a fracture of the condyle or rami of the jaw (it may occur on yawning). Movement of the jaw is restricted and painful. Deviation of the chin to the side indicates unilateral dislocation, which is confirmed by absence of the condyle from its normal place in front of the ear. In bilateral dislocations the jaw is fixed open. Reduction is achieved by grasping the lower jaw with both hands. The thumbs are protected by a towel, and placed as far back on the occlusive surface of the molars as possible. Downward pressure with a slight backward tilt is all that is required. General anæsthesia is required, except in recurrent dislocations. A four-tail bandage under the chin gives the patient some comfort afterwards.

**Dislocations of the Elbow.** Dislocation of the elbow may be complicated by separation of the upper radio-ulnar joint. Dislocation of the head of the radius alone is often accompanied by fracture. Dislocation of the radius and ulna may be anterior (inevitably accompanied by fracture of the sigmoid notch at the level of the head of the radius), posterior (the most common), lateral or medial. *Posterior Dislocations.*—These occur from falls on the hand with the elbow flexed. The radius usually remains attached to the ulna, but the coronoid process is usually fractured. Rapid swelling may obscure the bony landmarks of the elbow in any dislocation. If seen early, the condition is easily recognised by the shortening of the forearm, the prominent olecranon, and the broadening of the elbow. The lateral angle of the joint is about 135 degrees. The hand on the affected side is supported by the opposite hand. Radiological examination is essential to confirm the diagnosis, and to detect any associated fracture (*vide infra*).

*Reduction* under general anæsthesia is simple. Downward pressure is applied on the flexed elbow with both hands and the humerus is pushed backwards with the thumbs. The elbow is kept at right angles in a simple sling. Exercises are begun at the end of the first week, and the sling is discarded at the end of the third week. Medial and lateral dislocations are equally easy to reduce and retain. Anterior dislocations are accompanied by fracture of the sigmoid notch. Often this is an oblique fracture through the base of the coronoid process. This is easily fixed by a screw passing at right angles across it, and this stabilises the reduced dislocation.

*Complications.* *Nerve Injury.*—The ulnar nerve is the most susceptible to trauma, particularly in lateral dislocations. The deep branch of the radial nerve (posterior interosseous nerve) may be injured when the radial

head is displaced. The median nerve, being protected by soft tissues, is rarely affected. *Fractures.*—The same variety of fractures of the radius may occur as without dislocation. The coronoid process may be fractured in posterior dislocations, and the medial epicondyle may be avulsed and even occasionally caught in the joint in abduction dislocations (Fig. 612). The ulnar nerve is usually injured at the same time. If overlooked, the union of the fragment in the joint may damage the sigmoid notch. The fragment can readily be withdrawn from the joint by manipulation,



FIG. 612. After reduction of a posterior dislocation of the elbow, the medial epicondyle is found to be displaced and caught between the ulna and the lower end of the humerus.

faradic stimulation of the flexor muscles, or open operation. Bony union with some descent of the fragment, or even fibrous union is consistent with good function. The capitellar surface of the humerus may be damaged, leading to osteochondritis dissecans, or the production of a loose body. Fractures of the shafts of the bones around the elbow may also be associated with dislocation. Fracture of the ulna at the base of the sigmoid notch always occurs with anterior dislocations. Fracture of the shaft of the ulna may coexist with dislocation of the head of the radius (Monteggia fracture q.v.). Fracture of the neck or shaft of the radius may also be found with dislocation of the elbow. Other com-

plications are myositis ossificans, and ossifying hæmatoma in the brachialis anticus. Injury to the joint surface, disturbance of growth of the epiphyses, or deformity may lead to osteo-arthritis in later life.

**Dislocation of the Lower Radio-ulnar Joint.** This injury is usually associated with a fracture of the lower quarter of the radius. It may also occur with a Colles' fracture. When the radius is fractured special steps have to be taken to control the fracture (Chap. 96). In Colles' fractures, the lower radio-ulnar joint is often damaged. The consequent loss of alignment may later cause mild post-traumatic osteo-arthritis. A wrist strap may relieve pain for a time, but severe arthritis may require excision of a small segment of the ulna so that a false joint is formed above the wrist. Repair of relaxed ligaments by open operation is unsatisfactory.

**Dislocations of the Wrist.** Dislocation of the radiocarpal joint may occur alone, or combined with a carpal fracture or a fracture of the styloid process of the radius. The lunate is the most susceptible bone to dislocation alone and may be dislocated on the volar surface of the wrist

without displacement of the remainder of the carpus (Fig. 613). The displacement of the bone among the flexor tendons is variable. Reduction can usually be carried out by manipulation if the radiocarpal joint is opened by skeletal traction. If this fails, open operation through an incision on the ulnar side of the wrist is necessary. Later, sclerosis may occur in the bone due to avascular necrosis and be followed by osteo-arthritis of the wrist. When avascular necrosis has occurred, excision of the bone should be carried out before osteo-arthritis develops. The operation is carried out through an incision on the dorsum of the wrist.

Volar dislocation of the carpal bones usually includes the whole of the proximal carpal row, whereas backward dislocation may leave the lunate in position, or be complete.

The line of separation may run through a fracture of the waist of the navicular, leaving the proximal pole in place against the lunate, or the line may pass through a fracture of the radial styloid. Both the lunate and the navicular may be left attached to the radius. There are thus several possible types of carpal dislocation. Reduction with the aid of skeletal traction and manipulation is usually easy. After reduction the wrist should be immobilised



FIG. 613. Volar dislocation of the carpal lunate.

in a forearm plaster of the Colles' type, with the hand in the functional position. Immobilisation should be maintained for four weeks, unless there is an associated fracture which would take longer to unite, e.g. a fracture of the navicular. After removal of the plaster a firm bandage is applied and gentle exercises are begun.

**Dislocations of the Metacarpals and Phalanges.** Carpometacarpal dislocations are rare except in the thumb. There is seldom much displacement and this can be reduced by digital pressure. In the thumb, the dislocation is associated with a fracture (Bennett's stove fracture) which, by reducing the grip of the saddle of the metacarpal on the multangulum majus (trapezium) makes subluxation inevitable after reduction, unless special steps are taken to prevent this (Chap. 96). Dislocations of the metacarpophalangeal joints are usually simple to reduce, but owing to the interposition of tendon, or parts of the capsule, dislocation of the metacarpophalangeal joint of the thumb may be impossible to reduce by manipulation. Open operation through an anterolateral incision is necessary. Leverage, or division of interposing tissue, reduces the dislocation. The thumb should be retained in the flexed position for



three weeks by bandages or plaster. Interphalangeal dislocations may be accompanied by fractures of the saddle-shaped base of the more distal phalanx rendering reduction unstable, e.g. the dorsal marginal fracture associated with one variety of mallet finger. If the reduced dislocation cannot be retained in perfect position by plaster of Paris or a finger



FIG. 614. Posterior dislocation of the hip. Note the flexion, adduction and internal rotation.

wire, the insertion of a fine Kirschner wire across the joint in the line of the finger may be indicated. Avulsion fracture of the phalanges produced by traction of a collateral ligament may occur. The sesamoids present in the flexor apparatus, sometimes opposite each joint, must not be mistaken for fractures.

**Dislocation of the Hip.** This dislocation is rare, for very severe violence is necessary to produce it. The hip may be dislocated anteriorly in abduction, but posterior dislocation in adduction is much more common, and is particularly liable to occur in motor accidents in which the knee comes into violent contact with the dashboard. Partial abduction of the hip results in the posterior part of the acetabulum receiving greater force. This accounts for the frequent occurrence of fracture of the posterior rim of the acetabulum. Three types of dislocation are described: (1) *Posterior*.—Iliac and sciatic. (2) *Anterior*.—Pubic and obturator. (3) *Central*, in which the head is driven through the floor of the acetabulum. In posterior dislocations, the head of the femur may pass upwards on the lateral surface of the ilium, or downwards towards the sciatic notch; during the latter movement, the

sciatic nerve may be injured. (N.B.—Examine for paralysis before reduction.) There is much muscle spasm, and loss of movement at the hip. The hip is held in flexion, adduction and internal rotation (Fig. 614). The leg appears shorter. The diagnosis is confirmed by radiography.

**Reduction of Posterior Dislocations.** There are two methods in common use. The oldest is to place the patient on the floor, under general anaesthesia (Fig. 615). The leg on the affected side is flexed to a right angle at the knee and hip, the pelvis is steadied by an assistant, and the leg is then lifted upwards in some abduction and the head of the femur pulled forwards into the acetabulum. A more recent method is to carry out a similar manoeuvre with the patient face downwards on the operating

table, but with the lower extremities overhanging one end. The surgeon then steadies the patient's foot on the affected side between his own knees and presses hard downward behind the knee, feeling the buttock with his free hand to ascertain when the head of the femur enters the acetabulum. After-treatment consists of a fortnight in bed, followed by the wearing of a short plaster hip spica for a further two or three weeks in order to rest the joint and thus avoid the development of new bone around the hip joint, or avascular necrosis of the femoral head. These are rare complications, but they are more common in young patients owing to the great force necessary to dislocate the hip. If there is an associated fracture of the acetabular rim, one of three conditions may be found:



FIG. 615. Reduction of a posterior dislocation of the hip.

(1) The hip is stable on reduction and the fragment is in good position. Nothing more need be done. (2) The hip is unstable on reduction. The fragment must be replaced through a posterior approach and fixed with a screw. (3) The fragment has slipped into the joint as a loose body. It must be removed.

*Anterior Dislocations.* The Y-shaped ligament remains intact and holds the leg in abduction, slight flexion, and external rotation. There is a feeling of elastic rigidity of the hip. The anteriorly displaced femoral head may be palpated. There may be lengthening of the limb. Reduction is carried out by placing the anaesthetised patient on the operating table, and applying traction to the abducted and flexed (45 degrees) leg. Reduction is facilitated by downward and outward pressure by an assistant over the adductors.

In the so-called *central dislocation*, the head of the femur is driven

through the acetabular floor. It is treated by lateral traction applied by a Kirschner wire in the great trochanter, and traction in the line of the limb.

**Dislocation of the Knee.** The knee relies for its strength on its ligaments, reinforced by muscular insertions. Rupture of the medial collateral ligament of the knee joint may occur with excessive abduction, or rupture of the fibular collateral ligament with adduction. It is not customary to describe this as a dislocation unless the tibial plateau has been shifted as a whole to one or other side, indicating widespread tearing of the capsule and rupture of the cruciate ligaments. Such an injury demands the same treatment as rupture of a collateral ligament, namely, open repair of the ligament and the torn quadriceps expansion. This should be followed by fixation in plaster of Paris for six weeks. Repair of the cruciate ligaments is unsatisfactory and difficult. If effective repair of the collateral ligament is obtained, the knee can function well, in spite of a torn cruciate ligament. Only open operative repair can produce a strong collateral ligament. The term dislocation of the knee is more commonly applied to anterior and posterior displacements of the tibial plateau in relation to the femoral condyles. Posterior dislocation is produced with the knee flexed, whereas anterior dislocation is produced by blows behind the flexed or extended knee. The diagnosis is obvious. The popliteal artery may suffer in posterior dislocations. Manipulative reduction is straightforward. The knee should be immobilised in extension for six to ten weeks in plaster of Paris. In spite of the considerable displacement, there is often a surprisingly good return of function, for the collateral ligaments, though detached from the bone at the time of the injury, are restored to their normal position by reduction, and adhere firmly, thus tending to counteract any loss of stability caused by rupture of the cruciate ligaments. Quadriceps exercises are begun with the knee still in plaster, as the stability of the knee is largely dependent on the efficiency of this muscle. The exercises must be continued with active flexion of the knee, after removal of the plaster.

**Dislocations of the Patella.** Dislocation is always lateral owing to the angle of pull of the quadriceps, and is commonly facilitated by some congenital defect (e.g. failure of development of the lateral femoral condyle or the patella, a lax patellar retinaculum or knock-knee). Full extension of the knee readily reduces the dislocation. If there is an underlying abnormality, the dislocation may become recurrent. In view of this risk, it is best to immobilise the knee for a time after the first incident. During the period of swelling, rest and firm bandaging must be maintained, together with aspiration of the joint if there is a large effusion. When this has subsided, the knee is fixed in plaster of Paris for two or three weeks, leaving the foot free. Quadriceps exercises are maintained throughout. Recurrent dislocations may be treated by transplantation of the tibial tuberosity medially, plication of the vastus

medialis, or tendon transfer. The object is to produce a medial pull on the patella. If there is subpatellar osteo-arthritis the patella should be excised.

**Dislocation of the Fibula.** Either the upper or lower tibiofibular joints may be dislocated. Separation of the lower tibiofibular joint is called diastasis of the joint,\* and is discussed elsewhere as a complication of ankle injury. Dislocation of the upper end of the fibula is rare, but may be mistaken for a fracture of the tibial plateau, or a rupture of the fibular collateral ligament. Careful inspection of radiographs, and a comparative radiograph of the opposite knee may be needed. Reduction and retention with the knee in slight flexion are needed.

**Dislocations of the Foot.** The talus may be considered as the key to the many complex dislocations affecting the posterior part of the foot. It takes part in the formation of three joints, the ankle, the talocalcaneal joint, and the talocalcaneonavicular joint. It may become separated from all three (total or triple dislocation of the talus). The bone may come to lie in a variety of positions between the lower end of the tibia and the upper surface of the calcaneus and navicular. Manipulative reduction may be difficult, and therefore open operation is often needed. There is a risk of necrosis of the skin where it is stretched over the displaced bone. Therefore early reduction is important. Occasionally avascular necrosis of the bone follows dislocation.

The talus may be dislocated at the talocalcaneal and talonavicular joints, maintaining its normal position at the ankle. This is termed a complete subtaloid or double dislocation and may be either medial or lateral according to the direction in which the foot was displaced at the time of injury. It can easily be reduced by manipulation. A plaster is worn below the knee, at first without weight-bearing for four weeks, and then with weight-bearing for a further four weeks.

Dislocation of the talus at the ankle joint is usually combined with a fracture of the malleoli, or diastasis of the inferior tibiofibular articulation, and is discussed, more appropriately, with fractures of the ankle. Complex displacements occur if there is a fracture of the neck of the talus, or the body of the talus, and are more likely to be followed by avascular necrosis.

Mid-tarsal sprain, because of the proximity of the swelling and pain to the ankle joint, is liable to be mistaken for a sprain of the ankle, but careful examination shows the ankle to be freely and painlessly mobile, and the pain to be localised around the talonavicular and calcaneocuboid joints and to be increased by inversion or eversion of the forefoot. A short period of rest and a supporting bandage are required.

Metatarsals may occasionally be dislocated in crushing and compound injuries of the foot, and the dislocations may have to be reduced by open operation. Dislocations of metatarsophalangeal joints and interphalangeal joints resemble similar lesions in the hand.

\* Diastasis = separation of bones without fracture.

## CHAPTER 92

# FRACTURES AND THEIR TREATMENT

### GENERAL PRINCIPLES

INJURY to bone should be regarded as a complication of injury to the soft parts, for union of the bone is valueless if the soft parts are unable to endow the part with sensation and movement. A fracture is liable to be accompanied by severe soft tissue injury, produced directly by the injuring force and also by the movement of the ends of the broken bone; furthermore, the presence of a fracture restricts physiotherapy which is so necessary in restoring function. The injury to the soft tissues is obvious enough in open (compound) fractures in which the fragments are in communication with the exterior, but is equally serious in closed fractures. In fractures of the skull, the associated injury of the intracranial contents is much more important than the bony injury, and it is therefore more logical to discuss these fractures as a complication of intracranial damage than the other way about (Chap. 50). Fractures may be complicated by injuries of the skin (open or compound fractures), nerves, vessels, tendons and tendon sheaths, or internal organs.

**Injuries of Growing Bones.** The softness and elasticity of growing bones makes them liable to minor injuries, and the presence of epiphyseal plates provides weak places where separation may occur. *Greenstick Fractures.*—When a growing bone is bent, the side subject to tension breaks first, and the compressed side remains unbroken or becomes impacted. The deformity may be small, and the fracture being incomplete permits some function. Bruising and swelling may be slight. If the deformity is negligible, it may be left to be corrected by growth; the limb needs merely to be supported by plaster of Paris. If the deformity is severe, reduction is needed. Often this turns the fracture into a complete one, but as the soft tissues are not seriously damaged, a good position is usually maintained. *Infraction Fractures.*—These are fractures of cancellous bone produced by buckling under stress. The bone is only slightly weakened, and good function is usually preserved. A local ridge or tender area may be found in the bone. This is seen on X-rays to be due to a ridge pushed out on the surface of the bone, like a bulge on bamboo (Fig. 616). No treatment other than support by a splint or plaster for ten to fourteen days is needed. *Epiphyseal Separations.*—Although the surface of the epiphyseal plate is pitted with depressions interlocking with colliculi on the end of the metaphysis, the junction is weaker than neighbouring bone. Displacement of the epiphysis to one side as the result of injury is common, and it may be completely separated.

As the epiphysis slips, it always carries a fragment of metaphyseal bone with it, and this is always on the side to which the epiphysis is pushed (Fig. 617). In reduction, the epiphysis is pushed in the opposite direction, and the attached fragment of bone provides an effective stop against over-reduction. Epiphyseal injuries have to be reduced and retained in the same manner as fractures, but as union occurs rapidly, reduction should be made before the end of the first week. Support is only needed for three weeks. Interference with the growth of the epiphysis is rare, and occurs only when the epiphyseal plate itself is fractured: even then normal growth is usual.

#### THE CHARACTERISTICS OF FRACTURES

A fracture may consist of a simple fissure with slight disturbance of surrounding tissue, or a complicated crushing injury with frag-

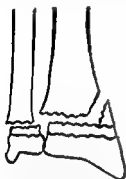


FIG. 617. Epiphyseal separation at the lower ends of the radius and ulna. Note the separation is on the metaphyseal side of the epiphysis, and a fragment of the metaphysis remains attached to the epiphysis. This fragment is always on the side towards which the epiphysis is displaced.



FIG. 616. An infraction fracture of the lower end of the femur. Note the slight ridge on the cortex on the lateral aspect of the femur above the epiphysis. When this ridge extends around the bone, the term "bamboo" fracture is sometimes employed.

mentation of bone and extensive soft tissue damage. Immediate treatment is dictated first of all by the patient's general condition, and the need to combat any shock. Secondly, treatment is determined by the nature of the soft tissue injuries, the type of bone injury, and the time interval after injury.

**Signs and Symptoms of Fracture.** (1) *Pain*.—This varies greatly. There is numbness of the part for a short time after injury, when the patient may not appreciate the severity of the injury. During this period reduction without an anæsthetic is sometimes possible. A patient under the influence of alcohol may feel little or no pain. (2) *Deformity*.—This also is very variable, depending on the site of fracture and whether it is complete or not. Fracture of the shaft of a long bone produces obvious angulation or shortening, whereas fracture of the pelvis or

## CHAPTER 92

# FRACTURES AND THEIR TREATMENT

### GENERAL PRINCIPLES

**INJURY** to bone should be regarded as a complication of injury to the soft parts, for union of the bone is valueless if the soft parts are unable to endow the part with sensation and movement. A fracture is liable to be accompanied by severe soft tissue injury, produced directly by the injuring force and also by the movement of the ends of the broken bone; furthermore, the presence of a fracture restricts physiotherapy which is so necessary in restoring function. The injury to the soft tissues is obvious enough in open (compound) fractures in which the fragments are in communication with the exterior, but is equally serious in closed fractures. In fractures of the skull, the associated injury of the intracranial contents is much more important than the bony injury, and it is therefore more logical to discuss these fractures as a complication of intracranial damage than the other way about (Chap. 50). Fractures may be complicated by injuries of the skin (open or compound fractures), nerves, vessels, tendons and tendon sheaths, or internal organs.

**Injuries of Growing Bones.** The softness and elasticity of growing bones makes them liable to minor injuries, and the presence of epiphyseal plates provides weak places where separation may occur. *Greenstick Fractures.*—When a growing bone is bent, the side subject to tension breaks first, and the compressed side remains unbroken or becomes impacted. The deformity may be small, and the fracture being incomplete permits some function. Bruising and swelling may be slight. If the deformity is negligible, it may be left to be corrected by growth; the limb needs merely to be supported by plaster of Paris. If the deformity is severe, reduction is needed. Often this turns the fracture into a complete one, but as the soft tissues are not seriously damaged, a good position is usually maintained. *Infraction Fractures.*—These are fractures of cancellous bone produced by buckling under stress. The bone is only slightly weakened, and good function is usually preserved. A local ridge or tender area may be found in the bone. This is seen on X-rays to be due to a ridge pushed out on the surface of the bone, like a bulge on bamboo (Fig. 616). No treatment other than support by a splint or plaster for ten to fourteen days is needed. *Epiphyseal Separations.*—Although the surface of the epiphyseal plate is pitted with depressions interlocking with colliculi on the end of the metaphysis, the junction is weaker than neighbouring bone. Displacement of the epiphysis to one side as the result of injury is common, and it may be completely separated.

impacted fracture. Misinterpretation of radiographs may occur and have legal as well as therapeutic importance. Good quality X-rays at odd angles may be necessary, but care must be taken not to be misled by the distortion produced. A carefully chosen oblique view may give important information, e.g. in fracture of the carpal navicular. Comparative views of the opposite side are especially useful in doubtful epiphyseal lesions in children. Radiography a week or two later may show a previously invisible fracture after some absorption of bone has occurred along the bony edges. Certain conditions may simulate a fresh fracture, e.g. persistence of an old fracture line in the skull, last traces of epiphyseal lines, foramina of nutrient arteries, old ununited fractures, small fragments of bone near osteo-arthritic joints, sesamoid and accessory bones, or old collapse fractures of the vertebrae.

Difficulty may arise when there is clinical suspicion of a fracture and radiography shows normal bone. This should not be taken as proof that no serious damage has occurred, for dislocations may become reduced spontaneously, and complete rupture of a ligament produces features clinically indistinguishable from avulsion of its bony attachment; if no injury to bone is seen, injury to the joint must be excluded by careful testing of ligaments and by radiography under strain (Fig. 607). A general anæsthetic is necessary for adequate testing of ligaments and "strain" radiography, though information may be obtained with local anæsthesia.

**Displacement of Fractures.** Whatever the site of fracture and however complicated the injury, the displacement of the ends of the fractured bone relative to each other may be analysed into four possible movements: (1) *Shortening*.—This is produced by muscle spasm, which causes over-riding of the fragments or impaction. If the bone ends can be interlocked, shortening can be prevented. If they cannot be interlocked, a force equal to that producing the shortening must be used to overcome the spasm, e.g. some form of skeletal fixation or traction. Permanent shortening is serious in the lower limb, but much less serious in the upper limb. (2) *Angulation*.—Muscle spasm may be a factor in producing this, e.g. the flexion of the upper fragment of the femur in fractures below the insertion of iliopsoas. Gravity may also play a part in producing angulation, but it is most commonly due to inaccurate reduction of the fracture; it is, however, one of the easiest deformities to correct. (3) *Side-to-side Displacement*.—Provided the axes of the two fragments of a fractured long bone are parallel, overlap of the bone ends not exceeding half their width is unimportant. Displacement may be persistent due to interposition of soft tissue or bone fragments. (4) *Rotation*.—This is corrected by aligning the limb. Small errors in large bones may be ignored. Perfect correction is best carried out by open reduction and interlocking of the bone ends. In the fingers, it is most important to avoid rotation, for malunion with rotation causes the finger to deviate from its normal line on flexion—a defect particularly important to musicians and manual workers.



of the calcaneus may produce little visible deformity. (3) *Abnormal Mobility*.—The nearer a fracture is to a joint the more difficult it is to detect abnormal mobility. Pain and muscle spasm increase the difficulties of examination. (4) *Loss of Transmitted Movement*.—Thus if the elbow is rotated and the head of the humerus fails to rotate with it, the shaft of the humerus is fractured. (5) *Swelling*.—This again may be extremely variable, depending on the amount of soft tissue damage, the size of any damaged vessels, and the opportunity or otherwise for blood to escape through an open wound. Swelling is important, for failure to allow for it under splints or plaster of Paris may lead to interference with the circulation and consequent loss of a limb. Swelling increases steadily for the first twenty-four hours, remains stationary for a time, and then after three days begins to subside as vasoconstriction and absorption replace the vasodilatation and œdema. The greater the swelling, the more difficult it is to appreciate the position of the fracture by touch, and the more the soft tissues obstruct reduction. (6) *Bruising*.—Skin discoloration, due to breakdown of blood, appears early if a fracture communicates with the subcutaneous tissues. It may appear later in less severe injuries, or it may track along tissue planes to appear some distance from the fracture, e.g. at the lower border of the deltoid in shoulder injuries. The direction of its movement is affected by the arrangement of the tissue planes, and the influence of gravity. (7) *Crepitus*.—This was the first recognised sign of fracture. Eliciting crepitus produces pain; therefore it is seldom deliberately invoked, though it may be accidentally felt. Crepitus is absent in incomplete or impacted fractures.

Gentleness is essential in the examination of a fracture to avoid pain and further damage to soft parts. Attention should always be paid to the circulation in the distal part of a fractured limb, and the nerve supply, before any manipulation is carried out. Temporary splinting may be needed to facilitate transport of patients to hospital. Straight splints serve for the forearm and leg. The injured leg may be bandaged to the uninjured for support. The arm is supported by a sling with a bandage over it and the arm. The Thomas's splint if available is the best first-aid splint for the lower limb. Splinting a fracture is important in combating shock as well as in preventing further damage to the soft tissues. Pain should be relieved by morphia. Hæmorrhage is usually controlled by the pressure of a firmly bandaged dressing. A tourniquet is rarely needed; loosely applied it increases bleeding by compressing the veins; applied tightly enough to stop arterial bleeding it may cause gangrene and lead to subsequent amputation.

**Radiology.** An X-ray is essential when the history or physical signs suggest the possibility of a fracture. Thus an elderly woman who falls, and then complains of pain in the hip, even though she may have got up and walked to hospital, cannot be excluded from having a fracture of the femoral neck until carefully X-rayed, for it is possible to walk on an

**The Forces Producing Fracture.** All fractures are the result of some force applied to the body. The force may be recognisably greater than the skeleton could be expected to bear, or it may be so slight (e.g. the influence of gravity) that the fracture is termed "spontaneous." Such a fracture indicates an abnormal state of the bone, and is better referred to as a *pathological fracture*. External force may be applied directly, breaking the bone under the point of impact, or indirectly, causing a fracture at some distance from the point of application of the force, e.g. fracture of the femoral neck in tripping over a mat. Direct violence tends to produce bending fractures, with fragmentation of the bone (*comminution*). The skin may be broken over the bone, producing a direct open (*compound*) fracture. Indirect violence often produces a spiral fracture and sharp spicules penetrating the skin render the fracture compound from within (*indirect compound fracture*). Muscular violence may cause a fracture, especially in a diseased bone, but also in a normal bone; e.g. the patella may be snapped across the femoral condyles and ribs may be fractured by coughing. Bone is also susceptible to fatigue; thus multiple repeated minor strains eventually destroy the molecular structure of the bone; reparative processes begin before fracture actually occurs; a fracture line may therefore be accompanied by evidence of repair when the patient is first seen. The commonest example of a *fatigue fracture* is "march" fracture of a metatarsal (Chap. 105).

**The Natural History and Repair of Fractures.** It is the surgeon's objective to achieve union of fractures in good anatomical position, in the minimum possible time, and without complications, and also to preserve the function of the soft tissues. In this pursuit, though nature is a powerful ally, there are liable to be many unexpected checks to the attainment of these ideals. The aim in all injuries is to give nature the best possible chance to repair the damage, and, properly assisted, the natural powers of recovery are remarkable, particularly in infection. Even in pathological fractures an attempt at repair is made and is often successful, e.g. when in neoplasms of bone, growth of the tumour is not more rapid than new bone formation. Attention is too often concentrated on the local factors affecting union, but there are important constitutional factors which must not be overlooked. The chief of these is the age of the patient.

The younger the patient, the more rapid is the repair, and consequently the shorter is the time available for accurate reposition of the parts before callus formation interferes with manipulation. Thus in children the final position of the fracture must be obtained in not later than ten days. In elderly people union is slower. The time for union also varies with the particular bone affected and the type of fracture. It is impossible to fix a definite time in which all fractures should unite, but experience gives a rough idea of the average time taken for the various fractures. Thus a fracture of both bones of the leg (i.e. tibia and fibula) in an adult is firm

**The Shape of Fractures.** The pattern of the fracture is determined by the causal stress, bending, torsion or compression. These forces do not always operate singly, but may be combined in varying degrees with each other. It is, however, usually possible to recognise the nature of the dominant force. Though this in itself may be of little practical value, the shape of the fracture it produces is of great importance, for it not only influences the deformity, but also the choice of treatment needed to overcome it. Thus a bending stress may produce a transverse fracture, but there is a tendency for fissures to run out from it at an angle of 45 degrees, and the line of fracture may follow one of these fissures (Fig. 618). If the fracture follows the lines of shearing in both directions

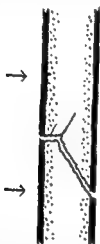


FIG. 618. A fracture produced by bending stress, partly transverse and partly oblique. The fine fissures, due to shearing stress, may become complete fractures and result in the separation of a triangular or "butterfly" fragment.

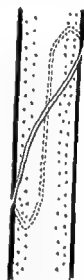


FIG. 619. A spiral fracture. Note the long straight element joined by a markedly sinuous section.

(i.e. proximally and distally) separation of a "butterfly" fragment occurs. With young elastic bones, the fissure may be incomplete, for bone yields readily to tension, but is resistant to pressure; the greenstick type of fracture is thus produced. A torsional stress produces a spiral fracture. It consists of two parts, a vertical or near vertical straight component joined by a doubly curved component (Fig. 619). The fractured ends are fluted and sharp and tend to penetrate soft tissues including the skin, thus producing a compound fracture indirectly. The fragments also tend to slide past each other causing shortening. Compression may be caused by the weight of the body and increased by the acceleration of falling. Acting on a curved bone, compression may produce a bending fracture. Acting directly along the shaft of a bone, it tends to produce an impacted fracture in which the expanded cancellous end of the bone is driven over the compact bone of the shaft. Acting on short cancellous bones such as the calcaneus or a vertebral body, it crushes and impacts them.

preferably by local pressure, or, if unavoidable, by clamping and/or ligature of larger vessels. Serious blood loss must be made good by transfusion as soon as possible. Shock is discussed under *Reactions to Injury*, Chap. 108, and *Blood Transfusion*, Chap. 109.

Hæmorrhage is the immediate cause of swelling around a fracture ; to this is added local œdema and fluid exudates, which reach a maximum in about twenty-four hours. This swelling can be dangerous, for it produces increasing tension when it cannot escape through tissue planes, e.g. as in the upper end of the posterior tibial compartment, where swelling may compress the posterior tibial artery ; or if the limb has been fixed in splints or plaster of Paris, expansion may be impossible and serious compression develops. Preliminary fixation must therefore always make allowance for subsequent swelling. This may be done by use of : (a) Plaster slabs, leaving one-third of the circumference of the limb open ; (b) padding ; or (c) skeletal traction, leaving the fracture site free of any constriction. Because of the later reduction in diameter of the limb caused by subsidence of the swelling and disuse atrophy of the limb, there are very few fractures which can be effectively treated by a single plaster cast. A second cast is needed at the end of the first seven to fourteen days when swelling has subsided. The cast may have to be changed several times.

After about three days the exudate is widely distributed in the tissue planes of the limb, destroying the elasticity of the soft tissues, and rendering manipulation more and more difficult as organisation of exudate proceeds. The appearance of widespread ecchymosis at this stage is an indication of this process. Reduction should therefore be achieved if possible before this time. Occasionally, if the hæmatoma around a fracture is very large, it should be evacuated to reduce the time of resorption and the subsequent fibrosis. This cannot be done by aspiration ; the clots have to be expressed through an incision and drainage is necessary for forty-eight hours. When a hæmorrhage has occurred into a joint, the tension in the joint may be very painful. Aspiration of the joint is then of value.

**The Repair of a Fracture.** This may be divided into stages, but in a cross-section of a healing fracture all the changes mentioned may be taking place at the same time in different parts of the section. A hæmatoma surrounds the fracture and is bounded by the surrounding soft tissues. The periosteum is almost invariably torn, but this is of little significance except in the very young. Invasion of the hæmatoma by granulation tissue begins where the blood supply is best and least disturbed by the injury, i.e. from muscle and undisturbed periosteum. The centre of the hæmatoma remains a mixture of fluid and clot until replaced by granulation tissue derived from the periphery of the organising hæmatoma and from the medullary cavity. While this is proceeding, organisation of the first formed granulation tissue occurs. Increasing numbers of fibro-

in twelve weeks. An undisplaced fracture of the navicular unites in seven weeks, and an undisplaced Colles' fracture is firm in three to four weeks.

The patient's general condition also exerts an effect on the rate of union. Poor general health, e.g. from renal or gastro-intestinal disease, may delay repair. Active syphilis, or even treated syphilis may be a bar to union, or union may be delayed till antisyphilitic treatment is instituted. Few other systemic diseases, apart from the hereditary bone dystrophy, osteogenesis imperfecta, prevent union, and even in the latter disease union is usually only delayed. Paget's disease, though rendering the bones more brittle and liable to transverse fractures, does not affect the time of repair. Senile bony atrophy predisposes to collapse fractures of the vertebral bodies and long bones, but these fractures slowly unite. Bony atrophy of this type is often accompanied, particularly after the post-menopausal endocrine changes in women, by the development of excessive subcutaneous fat. This not only increases the weight, but also the risk of fracture (e.g. of the wrist) on falling, and may make immobilisation of an excessively fat limb difficult.

Local diseases are usually much more important in their influence. In muscular paralysis, whatever the cause, the nourishment of the limb is impaired, and the repair of the fracture is consequently retarded. The common local bone diseases which weaken bone and may lead to pathological fracture are :

(a) *Inflammatory Diseases.* (1) *Acute.*—Osteomyelitis. This risk is nowadays reduced, but still exists in spite of antibiotic therapy. (2) *Chronic.*—Tuberculosis of bone. Brodie's abscess. Gummas.

(b) *Neoplastic Diseases.* (1) *Innocent.*—Adolescent bone cysts. Chondromas. (2) *Malignant.*—Primary : sarcoma. Secondary : carcinoma, particularly from the breast, prostate and thyroid. (3) *Bone Marrow Tumours.*—Multiple myeloma. Though these diseases may delay or prevent union, the treatment of the fractures themselves is usually the same as that for traumatic fractures. Special additional treatment is only required for tumours susceptible to endocrine therapy, or producing excessive pain, and also in established non-union.

**Immediate Effects of Fractures.** Immediately following a fracture there is local numbness which, associated with the mental stress of the accident, may prevent the patient realising the seriousness of the injury, or enable the patient to carry on in an unexpectedly normal manner. Pain and swelling, however, eventually develop, being aggravated by movement of the part. Relief of the pain by local splinting and bandaging is important in counteracting shock. This may be supplemented by injection of 2 per cent. procaine into the fracture hæmatoma and administration of morphia. Loss of blood may be visible in an open wound, or may occur diffusely into soft tissues, or into a body cavity. The degree of oligæmic shock is directly proportional to the blood loss, and is progressive if the loss continues. Hæmorrhage must therefore be stopped,

preferably by local pressure, or, if unavoidable, by clamping and/or ligature of larger vessels. Serious blood loss must be made good by transfusion as soon as possible. Shock is discussed under *Reactions to Injury*, Chap. 108, and *Blood Transfusion*, Chap. 109.

Hæmorrhage is the immediate cause of swelling around a fracture ; to this is added local œdema and fluid exudates, which reach a maximum in about twenty-four hours. This swelling can be dangerous, for it produces increasing tension when it cannot escape through tissue planes, e.g. as in the upper end of the posterior tibial compartment, where swelling may compress the posterior tibial artery ; or if the limb has been fixed in splints or plaster of Paris, expansion may be impossible and serious compression develop. Preliminary fixation must therefore always make allowance for subsequent swelling. This may be done by use of : (a) Plaster slabs, leaving one-third of the circumference of the limb open ; (b) padding ; or (c) skeletal traction, leaving the fracture site free of any constriction. Because of the later reduction in diameter of the limb caused by subsidence of the swelling and disuse atrophy of the limb, there are very few fractures which can be effectively treated by a single plaster cast. A second cast is needed at the end of the first seven to fourteen days when swelling has subsided. The cast may have to be changed several times.

After about three days the exudate is widely distributed in the tissue planes of the limb, destroying the elasticity of the soft tissues, and rendering manipulation more and more difficult as organisation of exudate proceeds. The appearance of widespread ecchymosis at this stage is an indication of this process. Reduction should therefore be achieved if possible before this time. Occasionally, if the hæmatoma around a fracture is very large, it should be evacuated to reduce the time of resorption and the subsequent fibrosis. This cannot be done by aspiration ; the clots have to be expressed through an incision and drainage is necessary for forty-eight hours. When a hæmorrhage has occurred into a joint, the tension in the joint may be very painful. Aspiration of the joint is then of value.

**The Repair of a Fracture.** This may be divided into stages, but in a cross-section of a healing fracture all the changes mentioned may be taking place at the same time in different parts of the section. A hæmatoma surrounds the fracture and is bounded by the surrounding soft tissues. The periosteum is almost invariably torn, but this is of little significance except in the very young. Invasion of the hæmatoma by granulation tissue begins where the blood supply is best and least disturbed by the injury, i.e. from muscle and undisturbed periosteum. The centre of the hæmatoma remains a mixture of fluid and clot until replaced by granulation tissue derived from the periphery of the organising hæmatoma and from the medullary cavity. While this is proceeding, organisation of the first formed granulation tissue occurs. Increasing numbers of fibro-

blasts appear, and slowly assume the characteristics of osteoblasts ; they lay down collagen fibrils in their exoplasm and between these fibrils bone salts are eventually deposited, forming new bone. This process is most advanced at the junction of the undisturbed periosteum and the hæmatoma. It follows that the earliest signs of reparative callus appear in this part of the hæmatoma. Some cartilage cells are seen among the osteoblasts ; their number is variable, but seems to be increased by movement at the fracture site. This may be one factor in the development of a false joint at the site of a fracture.

While osteoblasts are forming new bone, *osteoclasts* are absorbing dead cells on the surface of the bone ends and any loose fragments of bone. The primary stimulus to new bone formation is not known, although it may be this local concentration of the breakdown products of bone. Callus is converted into bone at the point where it first appears. Excess callus often develops and while it is absorbed at the periphery, organisation of centrally formed bone proceeds. This reorganisation of trabecular bone in the callus takes place in accordance with the stresses to which the callus is subjected. Compression is the stimulus to the formation of denser bone, and eventually leads to the formation of compact bone joining the compact bone of the fracture ; finer cancellous bone fills the medullary cavity. In young people, considerable remodelling of bone is possible and thus both angulation and side-to-side deformity may be spontaneously corrected, so that it becomes difficult to detect the nature of any pre-existing deformity.

#### Factors Influencing the Union of Bones.

(1) *Infection.* Direct infection of the bone ends, as may occur in a compound fracture, is the most serious complication threatening union. Callus formation is arrested, and the bone ends, already partially devitalised by the injury, become surrounded by infected granulation tissue which causes absorption of bone, and thus increases the space between the ends which become sealed with inactive fibrous tissue. Larger fragments of bone may be lost as sequestra. Union may eventually take place in spite of infection, but multiple operations may have to be performed and much delay may occur. Fibrosis and consequent adhesion of soft tissues develop and later interfere with function. Prevention of infection and treatment of infected fractures are discussed under compound fractures.

(2) *Avascular Bone Necrosis.* If a piece of bone is cut off from its blood supply, only the surface cells can obtain nourishment and the rest of the bone dies. If a large single fragment, or multiple small fragments as in a comminuted fracture, lose their blood supply, bony union may be delayed or prevented. When the fragment on one side of a fracture is cut off from its blood supply (e.g. in fracture of the carpal navicular) non-union is inevitable. Lesser degrees of avascular necrosis exert a retarding influence on healing. In many fractures, irrespective of whether

they are open or closed, there is such wide displacement of the bones and stripping of the soft tissues around them that death of a surface layer of bone cells occurs, resulting in diminished proliferative power of the bone ends. This mechanism accounts for many cases of atrophic non-union in young and healthy people. A fragment of bone having been cut off from its blood supply is at least temporarily prevented from undergoing further change. How then is avascular necrosis recognised? Around the avascular fragment, changes occur as the result of injury; they include hyperæmic decalcification and disuse atrophy of bone with a normal blood supply, whereas an avascular fragment retains its normal density and thus in X-rays stands out in contrast with surrounding bone although it would not be noticed next to normal bone. This change may be suspected at any time after the fourth week, and is usually obvious by



FIG. 620. Bone density changes in avascular necrosis of the femoral head. A. Normal bone density throughout. B. The shaft of the bone with a good blood supply undergoes traumatic osteoporosis and disuse atrophy, rendering the necrotic head apparently denser. C. With the passage of time and use of the limb, the density of the shaft returns to normal, and the deposit of calcium salts in the necrotic head makes it denser than normal bone. The fracture, of course, remains ununited.

about the eighth week. Much later in the history of the necrotic fragment there is an absolute increase in its density compared with that of normal bone due to deposition of calcium salts in the fragment—a foreign body reaction (Fig. 620).

(3) *Movement*. It is obvious that fractured ribs which are in constant slight movement unite well, and that movement is no bar to union. Constant and excessive movement may prevent union, or produce a false joint. Few forms of splinting produce absolute immobilisation, though it may be achieved by mechanical methods.

(4) *Compression*. Bone was built to withstand compression, and it responds to compression by hypertrophy. Early weight-bearing in a plaster cast may thus act as a *stimulus* to bony union, as well as being a method of treatment which permits graduated exercises.

(5) *Interposition of Soft Parts*. The interposition of muscle or the impaction of a plate of compact bone across the open ends of the medullary cavity may, by interrupting the continuity of the hæmatoma, produce a layer of fibrous tissue which permanently separates the bone ends. Wide displacement may serve the same end by allowing soft tissue to prolapse



between the fragments. The presence of soft tissue may be appreciated during manipulation by the absence of crepitus. If wide separation is confirmed by X-ray, open operation is indicated to prevent non-union.

(6) *Separation of the Bone Ends.* This may be produced by loss of bone, over-traction on the bones, or the bracing effect of an unfractured or already united neighbouring bone. Over-traction is a risk with skeletal traction which places great power in the hands of the surgeon. Skeletal traction should thus be minimal, and be used for the minimal time. Fixed distraction of the fragments by transfixion pins should be forbidden, and fixed separation by plates screwed on to the bone must be avoided.

**The Union of Bones.** Two separate though related standards have to be considered : (1) Clinical union ; (2) radiological union. Clinical union always precedes radiological union, often being many weeks in advance of it. If a fracture has been reduced by open operation and accurately plated, it may be difficult to see any radiological signs of union. Therefore, if the patient is not to be kept an unnecessarily long time in plaster, it follows that clinical union must be the standard on which the time for increasing activity is based. The *strength* of union may, however, best be judged on radiological evidence, and the radiological signs of *non-union* are of great importance. Clinical union is determined by : (a) Absence of abnormal mobility ; (b) absence of pain at the fracture site on straining it, when testing for abnormal mobility ; (c) loss of tenderness at the fracture site. Clinical union may be difficult to determine due to pain arising from other lesions, or to the propinquity of a joint which interferes with the examination. The findings should then be checked with the radiographs, and if the presence of union is still uncertain, two courses are open : (1) The limb may be exercised in bed for a week and then re-examined. It is surprising how often doubts are resolved at the end of this period. (2) The limb may be replastered and weight-bearing or non-weight-bearing exercises begun. At the end of three to four weeks the plaster is removed and the situation reassessed.

Radiological evidence of union is provided by the appearance of organised bone between the fracture surfaces and the marginal organisation of the callus. Later on, compact bone appears, but decisions regarding treatment cannot wait for this. The thicker the soft tissues, the more difficult is it to judge the strength of the callus, particularly as this is not very dense in the early stages of repair. Long oblique or spiral fractures, having a longer surface area for union, are stronger at an early stage than a transverse fracture in which union must be firmer if bending at the fracture site is not to result from muscular pulls or weight bearing. The signs of non-union and delayed union are discussed in a later chapter.

### GENERAL PRINCIPLES OF TREATMENT

The object is to restore the part not only to its pre-accident appearance, but to its pre-accident functional capacity. This object cannot always be

achieved, but it must always be pursued, and is carried out in a series of steps, the three R's of fracture treatment : (1) *Reduction*.—Useless unless the position achieved is retained. (2) *Retention*.—A necessary evil which may delay re-education. (3) *Re-education or Rehabilitation*.—The art of obtaining the early return of function and the best use of remaining function.

**Reduction.** The object of reduction is to restore the bones to their normal alignment and anatomical shape. It is opposed by : (1) Muscle spasm which can now be readily and completely overcome by anaesthesia and muscle relaxants ; (2) interposition of bone fragments and soft tissues at the fracture—it cannot always be overcome without resort to open operation and inspection of the bone ends ; and (3) swelling and fibrosis of the soft parts, preventing restoration of the limb to its normal length and resisting the reposition of the bone fragments.

Early reduction takes advantage of the favourable condition of the soft parts ; by reducing the deformity, it reduces the risk of pressure damage to skin, blood vessels and other soft tissues, and it relieves pain. Immediate reduction may be accomplished by any of the following methods, but the choice of the correct method in each individual fracture is one of the arts of fracture treatment. (1) *Manipulation*. (2) *Continuous Traction*.—This combines some retention with slow reduction. (3) *Operation*.—(a) Open operation : inspection, reduction and internal fixation ; (b) Limited operations, e.g. bone transfixion. Later reduction of an angular deformity may be carried out by wedging the plaster (Chap. 103).

The deformities to be overcome are : (1) *Shortening*.—This is overcome by traction (manual, skin or skeletal). (2) *Angulation*.—Overcome by manual reposition or wedging the plaster. (3) *Side-to-side Shift*.—Corrected by manual replacement. (4) *Rotation*.—Corrected by aligning the limb or the bone ends when these are seen at open operation. Reduction should be carried out as soon as the general condition of the patient permits ; sometimes an immediate temporary reduction, with perhaps inadequate retention, may be necessary to avoid local complications while shock is being treated. Definitive treatment may thus have to be postponed. Nevertheless, it is a fundamental principle of fracture treatment, and indeed of the treatment of all injuries, that the first opportunity to treat an injury is the best opportunity, and that this treatment should if possible be definitive ; it may not necessarily be the final treatment, but should form part of a well-planned scheme of treatment. It follows from this that, ideally, the first person to treat an injury should be capable of visualising the final treatment. The ground lost by an error of judgement in the early stages of treatment may never be regained.

**Retention.** The minimal retention necessary to maintain the reduced fragments in good position should be used. All retention interferes with function ; therefore if this is to be preserved, fixation should be designed

between the fragments. The presence of soft tissue may be appreciated during manipulation by the absence of crepitus. If wide separation is confirmed by X-ray, open operation is indicated to prevent non-union.

(6) *Separation of the Bone Ends.* This may be produced by loss of bone, over-traction on the bones, or the bracing effect of an unfractured or already united neighbouring bone. Over-traction is a risk with skeletal traction which places great power in the hands of the surgeon. Skeletal traction should thus be minimal, and be used for the minimal time. Fixed distraction of the fragments by transfixion pins should be forbidden, and fixed separation by plates screwed on to the bone must be avoided.

**The Union of Bones.** Two separate though related standards have to be considered : (1) Clinical union ; (2) radiological union. Clinical union always precedes radiological union, often being many weeks in advance of it. If a fracture has been reduced by open operation and accurately plated, it may be difficult to see any radiological signs of union. Therefore, if the patient is not to be kept an unnecessarily long time in plaster, it follows that clinical union must be the standard on which the time for increasing activity is based. The *strength* of union may, however, best be judged on radiological evidence, and the radiological signs of *non-union* are of great importance. Clinical union is determined by : (a) Absence of abnormal mobility ; (b) absence of pain at the fracture site on straining it, when testing for abnormal mobility ; (c) loss of tenderness at the fracture site. Clinical union may be difficult to determine due to pain arising from other lesions, or to the propinquity of a joint which interferes with the examination. The findings should then be checked with the radiographs, and if the presence of union is still uncertain, two courses are open : (1) The limb may be exercised in bed for a week and then re-examined. It is surprising how often doubts are resolved at the end of this period. (2) The limb may be replastered and weight-bearing or non-weight-bearing exercises begun. At the end of three to four weeks the plaster is removed and the situation reassessed.

Radiological evidence of union is provided by the appearance of organised bone between the fracture surfaces and the marginal organisation of the callus. Later on, compact bone appears, but decisions regarding treatment cannot wait for this. The thicker the soft tissues, the more difficult is it to judge the strength of the callus, particularly as this is not very dense in the early stages of repair. Long oblique or spiral fractures, having a longer surface area for union, are stronger at an early stage than a transverse fracture in which union must be firmer if bending at the fracture site is not to result from muscular pulls or weight bearing. The signs of non-union and delayed union are discussed in a later chapter.

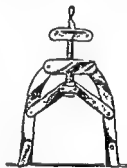
#### GENERAL PRINCIPLES OF TREATMENT

The object is to restore the part not only to its pre-accident appearance, but to its pre-accident functional capacity. This object cannot always be

The shape of the fracture greatly influences the choice of retentive apparatus, whether external or internal. Transverse fractures, if they can be reduced end to end, do not give rise to shortening. Plaster of Paris is a satisfactory medium for retention, for by this means angulation and rotation can be checked and controlled. If transverse fractures are to be controlled by open operation, a plate or an intramedullary pin is employed. Oblique and spiral fractures tend to slip and so produce shortening; therefore some form of distraction, either continuous or fixed, must be employed to prevent shortening. Plaster alone is unable to prevent shortening, for as swelling subsides the fragments tend to slip. By operation a stable and satisfactory reduction can be achieved by fixing an oblique or spiral fracture with two transverse screws, which can be inserted with little soft tissue disturbance. Comminuted fractures are the most difficult to stabilise, requiring a long plate to bridge the fragments; during its insertion considerable soft tissue damage is inevitable. A combination of skeletal fixation and plaster may give adequate stability and prevent shortening, and may sometimes be preferable.

Traction may be either skin traction or skeletal traction. Skin traction cannot be used for the application of great force; therefore its use is confined to obtaining the lesser degrees of traction needed to stabilise a limb (except in children in whom the larger area of skin in proportion to the body weight enables it to be used satisfactorily for distraction).

FIG. 623. Apparatus for use with Kirschner's wire. The wire is drilled through skin and bone. The stirrup is attached to the wire and the screws are tightened. Tension is given to the wire by turning the winged screw. (Courtesy London Splint Co. Ltd.)



Skeletal traction is carried out by transfixion of certain recognised bony points, such as the tibial tuberosity, calcaneum, olecranon, etc., with a Kirschner wire (Fig. 623). Its sole disadvantage is that there is a tendency for a wire to cut out in children or in osteoporotic bones (a Steinmann's pin is used in these circumstances—Fig. 624). Skeletal traction is very efficient; it helps to control rotation and is very comfortable. When much traction has to be applied it is the method of choice. There is a risk of infection of the entry holes in the skin. Skin infection around the metal can be prevented by avoiding movement of the skin relative to the pin or wire. These must therefore be free from the rotation of the stirrup and prevented from slipping from side to side; furthermore, the skin

to permit the greatest possible freedom of movement consistent with maintaining the fracture in good position. Every fracture does not need retention by apparatus. Sometimes the fracture is stable, but protection may be needed to reduce pain. The equation, fracture = plaster, is manifestly untrue. The choice of retention apparatus must be made from : (1) *Splints*.—(a) Prepared. Useful to rest a part, but too unadaptable for permanent fixation. (b) Plaster of Paris. This enables perfectly fitting splints to be made on the spot, and is so adaptable that a wide



FIG. 621. The Thomas's splint. (Courtesy of Doun Bros. Ltd.)

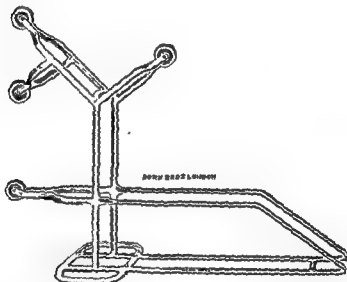


FIG. 622. The Böhler-Braun splint. This is a useful "skeleton" splint for temporary support of the leg, which is elevated with hip and knee in flexion. (Courtesy of Doun Bros. Ltd.)

range of fractures may be treated by this means. (2) *Continuous Traction*.—This does not alone provide the stable conditions and perfect soft tissue rest necessary in severe injuries, but may be combined with splints to give adequate fixation. (3) *Internal Fixation*.—(a) Completely covered by skin, e.g., plates and screws. (b) Penetrating the skin : transfixion wires attached to splints or incorporated in plaster. Supplementary "skeleton" splints, such as the Thomas's splint (Fig. 621) or the Böhler-Braun splint (Fig. 622) may be used to obtain elevation of the limb to reduce swelling. The Thomas's splint permits the limb to lie free from the bed, and this increases the patient's comfort, for when he moves the splint can also move, and so less strain is placed on the fracture.

old job, or stay away. Often, in these circumstances, it is better for the patient to attend a rehabilitation centre daily where exercises and occupations designed to fit him for his work are carried out and lunch is provided. A much earlier return to work is achieved with this regime than with irregular out-patient supervision. For a few patients, the residential rehabilitation centre provides more concentrated specific rehabilitation and facilitates assessment of patients' disabilities ; when travel is restricted by the disability residence at a centre may become a necessity.

The return to work may be hindered by social problems arising from long absence from employment, or the need, due to the severity of the injury, to take up a new occupation. Many channels of assistance are available to obtain new employment for the severely injured or to train them for new occupations. When the problem is only one of adapting the patient's disabilities to a particular machine tool, the "sheltered" workshop in which the patient is encouraged to try out the machine free from the pressure of the factory and in company with other disabled men, is of the greatest assistance, though not always available.

Though the surgeon may not take a direct part in rehabilitation, he should, to complete his task properly and in deference to the timid ignorance of many patients, act as co-ordinator of treatment from the time of injury to the return to work. Few patients are happy unemployed, and therefore idleness should be reduced to a minimum.

around the wire should be supported by firmly packed gauze or plaster. If these precautions are taken, infection of the pin track is rare, and if it occurs is usually superficial. The risk must, however, be borne in mind and therefore no skeletal pin or wire should transfix a fracture hæmatoma.



FIG. 624. A Steinmann's pin and stirrup. Pins are made in various lengths and diameters. They may be tapped through bone with a hammer, or drilled through by means of a handle fitting the blunt end of the pin. (Courtesy London Splint Co. Ltd.)

**Re-education.** This should be a continuous process from the completion of reduction to the return to work. Fixation should be designed to permit the maximum use of the injured part. Exercises should be arranged under supervision for all patients except those with the most trivial injuries. There are two possible exceptions to this rule. Children suffer little from immobilisation, if it is not too prolonged, and they are so eager to move that they use every free muscle. Only when there is a complicated injury or an associated muscular paralysis do they need much re-education. Active patients in a walking plaster usually perform sufficient exercises getting about and therefore need little in the way of specific exercises. Re-education is needed in elderly patients because they become stiff if joints are fixed; muscular wasting is often rapid, and they often lack the mental energy to take regular exercises and have therefore to be stimulated by the energy of others.

Re-education begins in the ward or physiotherapy department, and consists of specific exercises directed to the use of the unfixed muscles and joints. Thus in a Colles' fracture, the fingers, elbow and shoulder are put through a series of exercises. Such exercises are often best carried out in a class, which provides interest and competition. Specific exercises may be done against a background of general bodily exercises designed to improve the patient's general condition, respiration, and appetite. Diversional therapy may also be employed, not only to interest patients, but to exercise special muscles, e.g. those of the shoulder or fingers by weaving. When patients are ambulatory, the use of crutches provides a means of obtaining general exercises; these may be supplemented by special exercises. When a fracture is firm enough, weight-bearing in a plaster cast or in a special splint, such as a walking caliper, gives additional exercises to the affected part and stimulates callus formation.

Finally the patient's return to work must be considered. Light work is usually a doctor's dream. The patient must return to work fit for his

old job, or stay away. Often, in these circumstances, it is better for the patient to attend a rehabilitation centre daily where exercises and occupations designed to fit him for his work are carried out and lunch is provided. A much earlier return to work is achieved with this regime than with irregular out-patient supervision. For a few patients, the residential rehabilitation centre provides more concentrated specific rehabilitation and facilitates assessment of patients' disabilities ; when travel is restricted by the disability residence at a centre may become a necessity.

The return to work may be hindered by social problems arising from long absence from employment, or the need, due to the severity of the injury, to take up a new occupation. Many channels of assistance are available to obtain new employment for the severely injured or to train them for new occupations. When the problem is only one of adapting the patient's disabilities to a particular machine tool, the "sheltered" workshop in which the patient is encouraged to try out the machine free from the pressure of the factory and in company with other disabled men, is of the greatest assistance, though not always available.

Though the surgeon may not take a direct part in rehabilitation, he should, to complete his task properly and in deference to the timid ignorance of many patients, act as co-ordinator of treatment from the time of injury to the return to work. Few patients are happy unemployed, and therefore idleness should be reduced to a minimum.



## CHAPTER 93

### COMPOUND FRACTURES AND THEIR TREATMENT

A FRACTURE is best regarded as a complication of soft tissue injury, altering in no way the general principles of the treatment of wounds, though demanding additional treatment to retain the bone fragments in position. The object of the treatment of a compound (open) fracture is to convert it into a closed one and thereby to prevent the entry of infection. In compound fractures, without skin loss, there are three objects to be attained: (1) To prevent infection—the essential aim. (2) To suture immediately, under suitable conditions, such damaged tissues in need of repair, notably ligaments, tendons and nerves. (3) To utilise the opportunity provided by the open wound to reduce and fix the fracture.

**Prevention of Infection.** The aim is to put nature in the best position to defend herself, and then give her extra assistance with antibiotics. The battle is against infecting organisms and if fought on a clean field of living cells is usually won by nature in the opening stages. Only when the field of battle is dirty or continually shifting (*vide infra*) do the organisms gain the upper hand. Bacterial multiplication is encouraged by: (1) Dead tissue and blood clot which provide abundant feeding material. (2) Moisture which encourages organisms in superficial wounds, although in closed wounds tissue fluids may contain antibodies. (3) Foreign bodies and dirt may not only convey infection, but upset local defence mechanisms. (4) Failure of antibodies and leucocytes to reach the organisms, due to defective blood supply, dead tissue, or fibrous barriers. (5) Movement of tissue planes which prevents the sealing off of infected areas.

It follows that the steps to be taken in encouraging the maximum defensive reaction of the body to injury are: (1) Prevention or treatment of shock. (2) Excision of all dead tissue until a living wall of cells is left, with a good circulation. (3) Removal of all foreign bodies. (4) Hæmostasis. If the latter cannot be achieved, drainage must be instituted to avoid leaving clot which is a nutriment medium for organisms, and may produce necrosis by causing pressure in closed tissue planes. (5) Support for the tissues—not only for the bones—so that œdema is opposed by light pressure and tissue planes do not move on each other. (6) Provision of adequate skin cover to prevent infection entering the deeper layers, and maintenance of the skin surface in a dry condition. (7) Good surgical technique, including impeccable asepsis. If these rules are followed, the defensive reactions of the body are usually effective. With the assistance of antibiotics, correct surgical treatment has greatly reduced the incidence

of infection of open wounds. The risks of immediate infection are now far less than the risk of the skin sloughing which may permit later infection.

**Established Infection.** A wide range of organisms may be found in infected fractures; they fall into two groups, pathogenic and saprophytic. The pathogenic organisms are responsible for cellulitis, osteomyelitis, lymphangitis, adenitis, bacteraemia and septicæmia. These serious complications have been very greatly reduced by adequate surgery and the use of antibiotics, but infection with organisms resistant to antibiotics (some strains of *Staphylococcus pyogenes*) are becoming increasingly frequent. The use of antibiotics is discussed under *Antibiotic Therapy* (Chap. 110).

**Types of Compound Fracture.** Compound (open) fractures are divided into two kinds: (a) *Direct Compound Fractures*.—The bone and the skin are broken at the site of the blow, and there is a much greater chance of foreign bodies, dirt and fragments of skin being driven into the depths of the wound. (b) *Indirect Compound Fractures*.—The bone breaks away from the site of the acting force, and the deformity following this causes a sharp spicule of bone to penetrate through the skin. The risk of infection is much less, and the best treatment is to remove the soiled spicule if small, or if large to clean the fragment through a small incision without deep exposure, and then to treat the fracture as if it were closed. An indirect compound fracture is no bar to internal fixation when indicated.

**Direct Compound Fractures.** Effective surgery demands good access, and the wound must therefore be adequately enlarged without impairing the vitality of the wound edges and without jeopardising subsequent suturing. Systematic debridement of the wound is then carried out, but each tissue calls for slightly different handling. Skin is very resistant to infection. Therefore the minimum of excision is practised; in a clean-edged laceration skin excision may be omitted altogether. Wounds with crushed edges must be excised back to a bleeding edge, and for the sake of the subsequent scar small irregularities may be excised to obtain a flowing line. Fat is not provided with such a good defence and must therefore be excised until firmly supported clean tissue is exposed. Loose tags of fibrous tissue are excised. Dead muscle is a good nutrient medium for organisms and must be freely excised back to living tissue. Living muscle contracts on being pinched with forceps and has a good colour. Dirty bone may be scraped clean or the affected part chiselled off. Loose fragments of bone without a blood supply may present a problem. If small they should be removed. If large, their removal might invite non-union; therefore the risk of infection incurred by leaving them as a foreign body must be balanced against their value as a bone graft.

Ligaments forming part of the protection of a joint should be repaired as one of the steps to isolate the joint and prevent its infection. Where the capsule cannot be closed, the gap may be filled by living muscle,

turned into the gap by means of suitably placed incisions. Divided tendons need repairing, but whether this should be done early or late depends on the estimated risk of infection and the amount and quality of the skin cover. When clean healing can be anticipated immediate repair is best. The same conditions hold for several nerves. Division of a large blood vessel when there is an adequate undamaged collateral circulation, e.g. the brachial artery, poses no problems, but when a vessel such as the popliteal artery or the common femoral is divided, arterial grafting may save the limb (see Chap. 74).

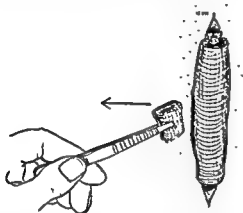


FIG. 625. The wound should be packed firmly with a gauze swab to prevent dirt being washed into it when the skin is being cleaned. The arrow is to indicate that the skin is swabbed away from the skin edge.

**Surgical Routine in Compound Fractures.** In the casualty department, attention is given to hæmostasis, wound cover, and temporary splinting. Resuscitation, if the patient is shocked, is begun there, or the patient is taken immediately to a theatre or special room where facilities for transfusion, oxygenation, and general supervision are available so that the wards are not disturbed. When the patient is fit for surgery, the preparation is carried out in the theatre under anæsthesia, otherwise the preparation is apt to be painful and inadequate. A separate

preparation trolley is required with forceps, swabs, scissors, soap and water, and iodine or spirit. The surgeon washes his hands thoroughly, but does not put a gown over his apron. The wounds if large or bleeding are packed with gauze swabs, to prevent the introduction of dirt (Fig. 625). The skin is then washed or scrubbed clean with soap and water, dried, and painted with spirit or iodine. The limb is then wrapped in a sterile towel. If a tourniquet is required to facilitate inspection of the wound it is now applied. The surgeon now scrubs up and the operation proceeds.

The skin is cleaned with spirit again, and the swabs are removed from the wounds. The operation field is towelled and the debridement of the wound is carried out meticulously. Consideration of the nature of the wound, the possible risk of infection, the shape of the fracture, and the age of the patient determines whether any internal fixation is to be employed. With or without internal fixation, the wound is closed with the minimum of suturing in the deeper layers, and careful hæmostasis is obtained, but if there is doubt about it the wound is drained for twenty-four hours with a small rubber drain. The skin is sutured carefully without tension.

The fracture must now be supported. If internal fixation has been

employed, the external fixation is lighter and designed to control and lightly compress the soft tissues. If the fracture has been controlled during the operation with skeletal traction and attachment to an orthopaedic table, the wires are incorporated in the plaster of Paris surrounding the limb. If no transfixion wires are used, the limb is set manually and immobilised in a plaster cast. A compound fracture usually swells less than a closed one, for blood is able to escape through the wound, but, nevertheless, some steps to permit swelling must be taken (padding the plaster, splitting the plaster or using only a plaster backslab). The limb should be elevated when the patient goes to the ward (the arm on pillows; the leg on a Braun's or Thomas's splint, or by raising the foot of the bed).

Treatment with antibiotics should begin in the preoperative phase so that the blood level of the drug is adequate by the time of operation. Effective reduction and fixation of the fracture should render the patient fairly comfortable. Serious pain indicates a complication (infective or vascular) or ineffective support and demands investigation. The patient's general condition is the best guide to progress. If the patient is alert, interested and active, and eats and sleeps well, the fracture is usually free from complications.

**Compound Fractures with Skin Loss.** Unclosed skin defects over fractures lead inevitably to infection of bone; therefore the wounds must be closed with living skin before infection occurs. It may prove impossible to approximate the skin edges due to: (1) Skin loss; (2) severe swelling of the part; (3) defective circulation in the remaining skin. Immediate closure is always the ideal. Bare bone and tendon (unless the area of exposed tendon is very small) cannot be successfully covered with a split-skin (Thiersch) graft; a full-thickness skin graft is therefore necessary. Occasionally muscle can be drawn together over exposed bone and used as a base for a split-skin graft which is the easiest type of graft for inexperienced hands to apply.

Skin cover may be obtained by the following manœuvres: (1) *Advancement Flaps*.—Wide undercutting of the skin may allow the skin edges to be approximated. This method is only applicable when there is a small area of skin loss. Tension at the suture line must be avoided, otherwise marginal necrosis of the skin occurs. (2) *Rotation Flaps*.—A rotation flap is fashioned by curved incisions extending from the wound. After undercutting the full-thickness skin flap is advanced and rotated over the area of the exposed bone or tendons. Inevitably a bare area is left on the other side of the flap. This is covered with a split-skin graft. (3) *Split-skin Grafting* (Fig. 626).—The Thiersch graft requires a vascular bed for it to "take." It is therefore primarily of use in closing defects over soft tissue, e.g. the amputated tip of the finger pulp, but it is a valuable supplement to full thickness methods of obtaining skin cover (*vide supra*). (4) *Pedicle Flaps*.—There is no time to tube a pedicle and advance it; the flap must be a direct one. Around the hand small flaps may be obtained from the

palmar skin or the dorsum of the fingers. Distant sites may also be used, e.g. the thigh. Larger flaps for the wrist may be obtained from the abdominal wall. Cross leg flaps are technically difficult and are best entrusted to the plastic surgeon.

**Special Infections. Gas Gangrene.**—The organisms responsible for this infection, *Clostridium welchii*, *Clostridium oedematiens* and *Clostridium septicum*, are spore-forming and anaerobic. They are unable to grow in living tissue and only cause infection when they contaminate tissue devitalised or destroyed by open injury. Spread of infection is due to diffusion of toxins and local thrombosis so that a continuous source of new dead tissue is available to the organisms. The best defence is put up by living tissue with a good blood supply, so that efficient excision of wounds is the most important step in preventing infection, and excision of dead tissue with minimum exposure of damaged tissue surfaces is the best treatment of established infection. The condition is most likely to be encountered in crushing injuries, or in inadequately treated wounds, and may limit itself to: (1) A small area around a wound; (2) a muscle belly or group of muscles; or (3) a segment of a limb.



FIG. 626. Extensive Thiersch grafts applied over an area of skin loss in a compound fracture of the elbow.

of temperature. The wound is pale and oedematous with a thin foetid discharge. Crepitation may be found in the wound due to gas although diagnosis and treatment should not await this late sign. Discoloration of the skin at some distance from the wound may be noted due to spread of necrosis in deeper tissues. Fulminating septicæmic forms may be seen. Confirmation of the infection can rapidly be obtained by examining a smear from the wound for clostridia, although the diagnosis should be made on the clinical evidence.

**Treatment.** Localised infections involving the subcutaneous tissues can be dealt with by wide excision. Muscle bellies or groups of muscles

The presence of gas gangrene is indicated by toxæmia with a rapid pulse out of proportion to the rise

need to be excised until healthy tissue with a good blood supply is exposed. When a limb segment is involved, amputation is needed and this may be best carried out through a joint to avoid leaving muscle surfaces exposed to infection. Patients with wounds in which there is widespread muscle damage, or in which treatment has been delayed, or in which there is established gas gangrene, should be given polyvalent gas gangrene antitoxin (9,000 units of *Cl. welchii* antitoxin, 4,500 units of *Cl. septicum* antitoxin and 3,000 units of *Cl. edematiens* antitoxin). More important, however, is the administration of an antibiotic (e.g. Aureomycin to which the clostridia are susceptible), which also controls the invariably associated coccidial infection. A severe hæmolytic anaemia usually develops and must be treated by blood transfusions. *Prognosis.*—This varies with the general condition of the patient and the degree of invasion by the organism (i.e. with the time the diagnosis is made). The more localised the infection the more susceptible it is to surgery and the better the outlook.

*Tetanus.* The clinical features of tetanus are due to absorption of exotoxin from a wound infected with the tetanus bacillus (*Cl. tetani*). The route of absorption is along motor nerves to anterior horn cells of the spinal cord where the toxin exerts its effect, producing reflex spasm of the muscles supplied. Tetanus organisms are found especially in manured and cultivated soil. The conditions in which a wound is inflicted are of importance in assessing the risk of developing tetanus. Infection by other organisms greatly increases the multiplication of tetanus bacilli. The severity of the muscle spasm and its spread are dependent on the amount of toxin absorbed. Experience has shown that when symptoms appear before the end of the first week after injury tetanus is usually fatal and after that period the chance of survival progressively increases. Spasm may appear first in a group of limb muscles or the jaw muscles, or it may be generalised.

The chief defence against tetanus is careful debridement of wounds and consequent aseptic healing. Antibiotics, although inactive against tetanus itself, nevertheless play an important role in its prevention by suppressing infection with other organisms which enhance the growth of tetanus bacilli. Prophylactic administration of tetanus antitoxin is still "legally" desirable for deep penetrating wounds or those sustained where animal excreta are prevalent or when old wounds are reopened, although its value is uncertain, and it carries risks: They are: (a) *Anaphylactic shock*, which may occur within a few minutes, or, less severely, in up to two hours; (b) *serum sickness*, which is characterised by pyrexia, rashes, cedema and painful joints, and usually occurs seven to twelve days after injection in people who have had serum before; (c) *thermal reaction*, a sudden pyrexia with rigors following intravenous injection and probably due to "pyrogens" which are present in some batches of serum.

Whenever serum is given, irrespective of the route of administration, a solution of 1 : 1,000 adrenaline should always be immediately available

for hypodermic injection in case anaphylaxis occurs. In giving tetanus antitoxin one of the following procedures should be carried out : (1) *No History of Asthma or Infantile Eczema.*—Give the injection of antitoxin intramuscularly and keep the patient under close observation for half an hour. (2) *History of a Previous Injection of Serum.*—Give a trial dose of 0.2 ml. subcutaneously. If no reaction occurs within half an hour give the rest of the antitoxin intramuscularly. (3) *History of Asthma or Eczema.*—Reduce the trial dose to 0.2 ml. of 1 : 10 dilution subcutaneously. If there is no reaction, follow this in half an hour by 0.2 ml. of undiluted serum subcutaneously. After another half-hour, give the rest of the antitoxin intramuscularly and keep the patient under observation for a further half-hour.

The prophylactic dose of antitoxin is 1,500 International Units (1950, i.e. = twice the potency of the old I.U. = one U.S.A. Unit), subcutaneously or intramuscularly, repeated weekly for three weeks. The therapeutic dose is 100,000 I.U. intravenously followed by 25,000 I.U. weekly as long as required. Before the first intravenous injection, it is most important to take the precautions described above and never to give an intravenous injection of antitoxin before a preliminary intramuscular dose has been tolerated for half an hour. Intravenous injections should be given slowly with the serum at room temperature. The antitoxin must never be given intrathecally.

In the past the deaths from tetanus have occurred from respiratory obstruction due to spasm or retained secretions, and exhaustion. The abolition of spasm by means of muscle relaxants (e.g. Mephenesin) has brought new hope in the treatment of tetanus. The management is summarised as follows : (1) Excise and clean the wound to prevent further absorption of toxin. (2) Administer antitoxin intravenously and intramuscularly (*vide supra*). (3) Give sedatives and control the spasm with a muscle relaxant. (4) Control respirations with a tracheotomy and positive pressure apparatus. (5) Control pulmonary infection by tracheal toilet and antibiotics. (6) Maintain the patient's general condition by control of fluid balance and feeding through a gastric tube.

*Active immunisation with tetanus toxoid* is most strongly recommended for everybody as the most effective means of prevention.

**Infected Compound Fractures.** Infection may be seen early due to lack of treatment, or to skin necrosis. Complete debridement at this stage with primary closure is a very risky procedure. A thorough debridement, with the minimal opening of fresh tissue planes is indicated. The wound is not closed, but packed lightly with vaseline gauze, and enclosed in plaster (the "closed plaster" treatment). Some local infection is almost inevitable, but may be readily controlled by administration of appropriate antibiotics. As soon as infection is eliminated, secondary suture of the wound may be carried out. When there is osteomyelitis, successful closure over the bone is unlikely, but even if the granulating area is re-

duced by suture healing occurs more quickly. When infection has become established and there is frank osteomyelitis, a long period of treatment is necessary, and there is no guarantee of success. The combination of adequate surgery and antibiotics has ruled out the serious complications of cellulitis and septicæmia, but may leave a wide area of skin loss and extensive osteomyelitis.

When X-rays show or suggest the presence of dead fragments of bone the wound is explored, any dead bone is removed, and a closed plaster is applied. This may have to be done on several occasions before a cleanly granulating wound is obtained with no sinus communicating with bone. The time is then ripe for secondary closure of the wound. Careful skin toilet and the application of a skin paste (e.g. silicone ointment) to the area around the wound is necessary to avoid dermatitis. The method of skin closure is important, for it is probable that the amount of bone lost together with the infection may prevent union, and therefore a bone graft or other open operation may later be necessary. For a safe secondary operation full thickness skin cover is necessary and is achieved by the methods already discussed under *Compound Fractures with Skin Loss*. With satisfactory skin closure there is a chance for the fracture to unite. Rarely, in comminuted and incomplete fractures, union will have occurred in spite of infection. The chances of the fracture uniting depend on the amount of bone loss, the degree of contact of the bone ends, and their vitality. When the signs are encouraging, the closed plaster treatment may be continued and weight-bearing, which often acts as an extra stimulus to union, permitted. Repeated radiological examination is needed to decide whether union is occurring, or if there is established non-union. In any case some time must be allowed to elapse, for immediate operation would probably be followed by recurrence of infection. This risk is always present in interfering with a previously infected fracture, but the chances of success are increased by waiting three to six months after all evidence of infection has disappeared.

The treatment necessary for a severe compound fracture of the leg with skin loss and infection may cover a period of two or more years, and even at the end of this time success may not be achieved. The knee and the foot become stiff, and the circulation and skin nourishment are likely to be deficient. The health of elderly patients deteriorates during a long course of treatment and therefore amputation may have to be advised. In younger patients circumstances dictate the course, but immediate judgement can usually be postponed. A long period away from work, with the inevitable uncertainties and the need to learn a new occupation, may produce such mental and moral deterioration that early amputation followed by the fitting of an artificial limb may be preferable. However, amputation is the last resort and should not be done without obtaining a second opinion.



for hypodermic injection in case anaphylaxis occurs. In giving tetanus antitoxin one of the following procedures should be carried out : (1) *No History of Asthma or Infantile Eczema*.—Give the injection of antitoxin intramuscularly and keep the patient under close observation for half an hour. (2) *History of a Previous Injection of Serum*.—Give a trial dose of 0.2 ml. subcutaneously. If no reaction occurs within half an hour give the rest of the antitoxin intramuscularly. (3) *History of Asthma or Eczema*.—Reduce the trial dose to 0.2 ml. of 1 : 10 dilution subcutaneously. If there is no reaction, follow this in half an hour by 0.2 ml. of undiluted serum subcutaneously. After another half-hour, give the rest of the antitoxin intramuscularly and keep the patient under observation for a further half-hour.

The prophylactic dose of antitoxin is 1,500 International Units (1950, i.e. = twice the potency of the old I.U. = one U.S.A. Unit), subcutaneously or intramuscularly, repeated weekly for three weeks. The therapeutic dose is 100,000 I.U. intravenously followed by 25,000 I.U. weekly as long as required. Before the first intravenous injection, it is most important to take the precautions described above and never to give an intravenous injection of antitoxin before a preliminary intramuscular dose has been tolerated for half an hour. Intravenous injections should be given slowly with the serum at room temperature. The antitoxin must never be given intrathecally.

In the past the deaths from tetanus have occurred from respiratory obstruction due to spasm or retained secretions, and exhaustion. The abolition of spasm by means of muscle relaxants (e.g. Mephenesin) has brought new hope in the treatment of tetanus. The management is summarised as follows : (1) Excise and clean the wound to prevent further absorption of toxin. (2) Administer antitoxin intravenously and intramuscularly (*vide supra*). (3) Give sedatives and control the spasm with a muscle relaxant. (4) Control respirations with a tracheotomy and positive pressure apparatus. (5) Control pulmonary infection by tracheal toilet and antibiotics. (6) Maintain the patient's general condition by control of fluid balance and feeding through a gastric tube.

Active immunisation with tetanus toxoid is most strongly recommended for everybody as the most effective means of prevention.

**Infected Compound Fractures.** Infection may be seen early due to lack of treatment, or to skin necrosis. Complete debridement at this stage with primary closure is a very risky procedure. A thorough debridement, with the minimal opening of fresh tissue planes is indicated. The wound is not closed, but packed lightly with vaseline gauze, and enclosed in plaster (the "closed plaster" treatment). Some local infection is almost inevitable, but may be readily controlled by administration of appropriate antibiotics. As soon as infection is eliminated, secondary suture of the wound may be carried out. When there is osteomyelitis, successful closure over the bone is unlikely, but even if the granulating area is re-

A pin may be used as a temporary lever and then withdrawn, or it may be incorporated in the plaster as in fractures of the calcaneus to help maintain reduction. These methods, even with the use of special appliances and radiological control, are to a large extent "blind," and as obstruction to reduction is often caused by tissues interposed between the fragments, there are some failures which would be obviated by open reduction and internal fixation.

Internal fixation is unnecessary in the young, for the growth of the bone aligns the majority of reasonably reduced fractures; there are exceptions such as in slipping of the upper femoral epiphysis. Often open reduction may be needed in the young, but reduction is stable enough without internal fixation. Internal fixation is unwise in infected fractures, as the opening of new tissue planes spreads infection.

#### The Choice of Methods of Internal Fixation. (1) Wire.—

This was the first material used (for fracture of the patella). It does not easily produce perfect apposition, and is liable to distort or break. It has largely fallen out of use, though stainless steel wire may, if used through chosen drill holes, be the best method of retention in fractures of the fingers. (2) Bands.—Metallic bands (Parham's band) passed around the bone, and tightened by a special clamp, are only effective in spiral or oblique fractures and have gone out of fashion. (3) Screws.—Where these can be used alone they produce strong fixation; they have the advantage that a small amount of metal is inserted. They are of particular use in oblique and spiral fractures, in which two screws inserted across the fracture line produce firm fixation. There is a choice of screws with a fine thread and of machine screw pattern (Sherman screws) suitable for firm compact bone, or screws with a coarse thread and of wood screw pattern (Venable screws) suitable for cancellous bone. The latter are the more generally useful, and can be adapted to all purposes. To obtain a good grip, screws should be inserted into drill holes  $\frac{1}{16}$  inch smaller in diameter than the screws. For dense bone the difference should be  $\frac{1}{32}$  inch to avoid splitting the bone. (4) Plates.—These are combined with screws. There have been many designs. Lane's original plates had the holes in alignment, and were thereby unduly weak. New designs to improve the strength of the plate and to enable the screws to be inserted at an angle to the cortex have been made. Among them the slotted plates (Egger's plates) are particularly



FIG. 627. A small form of the Roger Anderson cross-pin appliance used for fractures of the mandible.

## CHAPTER 94

### INTERNAL FIXATION OF FRACTURES

INTERNAL fixation of fractures was primarily designed to produce perfect anatomical reposition ; it was, however, found to have two important secondary advantages ; thus it reduced the need for external support, and made exercise of the part possible much earlier. These side-effects are regarded as being so important that they are now the indication for internal fixation of many fractures—notably those of the upper end of the femur. What amounts to the conversion of a closed fracture into an open fracture, even under strictly aseptic conditions, is not to be undertaken lightly, for though the risk of infection is nowadays slight, there remains the possibility of interference with the blood supply of the bone or skin necrosis. Internal fixation is the best method of treatment in the following circumstances : (1) When a fracture is known to be unstable when reduced by closed methods. (2) When manipulative and other methods have failed. (3) When restoration of a perfect anatomical position is essential for good function, e.g. fractures of both bones of the forearm. (4) When early ambulation or use of the part is essential to return of function or to preservation of health, e.g. fracture of the upper end of the femur in the elderly. (5) In established non-union.

Many methods of internal fixation have been devised, and new ones are continually appearing ; this shows that no single method has proved ideal for all cases. In practice it is found that certain methods suit certain bones and certain fractures. The chief problem is the choice of the best modification of a given method ; all carry the risk of technical error. Therefore familiarity with a technique may favour its choice.

There are certain halfway stages along the road to internal fixation to be considered first, namely, fixation with pins, spikes, threaded nails, or transfixion by wire. These enable the fragments to be controlled and the fracture to be reduced either by the sense of touch or by radiological control ; they may be retained or withdrawn after any retentive apparatus, e.g. plaster of Paris, is applied, or they may be incorporated in the plaster. The most important of these methods is the cross-pin technique of Roger Anderson. The bone fragments are transfixed with two pins on each side of the fracture ; the pins are angled in relation to each other and subsequently bolted to a rigid bar which maintains the position of the fragments. This is still the best method of fixation in many fractures of the mandible, and is useful in other situations (Fig. 627). It is unsuitable in the thigh, for the pins have to transfix muscle, and the movement of the muscle around the pins produces infection at their points of entry.

Excessive tissue reaction occurred around the metals used in the past, but the two metals now in general use, Vitallium, an alloy, which can be moulded, but is a little brittle, and En 58 J stainless steel (18.8 S Mo) cause little or no tissue reaction, and may therefore be left permanently *in situ*. Removal of fixative agents may be rendered necessary by infection, atrophic non-union, or extrusion of the metal externally or on the surface of a joint due to bone absorption, e.g. as may occur with a Smith-Petersen pin. Occasionally a screw may be inserted to restrict movement, but may have to be removed to permit full joint function, e.g. in fractures of the fibular malleolus, or dislocations of the acromioclavicular joint. Patients often demand the removal of palpable screws, though they may cause no pain.

Operative fixation of a fracture may often be undertaken immediately, for it is the method of choice. There is much to gain in comfort, and repeated manipulation is avoided. Sometimes when manipulation is likely to be successful, and retention in splints or by skeletal traction satisfactory, the decision regarding operation may be delayed to see if manipulative reduction achieves the desired result. If it fails open operation may then be recommended. In some cases the degree of shock may enjoin some delay. Late operation is necessary when retention has failed to check the appearance of deformity, or when it is evident that non-union will occur.

Scrupulous asepsis is vital. Preparation of the part may be done in the ward, or in the theatre; the latter is chosen if there is difficulty in handling the limb, or much pain is likely to be inflicted. Operations on limbs may be facilitated by application of a tourniquet. At the end of operation, hæmostasis must be ensured to avoid the risk of tension on the skin edges. In a few smaller procedures such as screwing the medial malleolus, the tourniquet may be left on till the wound is dressed, but the skin suturing must never be blood-tight. When there is doubt about hæmostasis, the wound is drained for twenty-four hours. The soft tissues should be supported and protected by even bandaging and the use of a plaster cast, or splint, even if the limb is already stable. When any post-operative swelling has subsided, splints may be removed if the fracture needs no external support. Limbs should be elevated to reduce swelling. The arm may be raised on pillows, and the leg on splints; although raising the foot of the bed may suffice for the leg.

The use of post-operative antibiotics should not become a matter of routine (see *Antibiotic Therapy*, Chap. 110), but they are given to patients with compound fractures and when infection is suspected. Major operations in which there is long exposure in the theatre or in which there is much soft tissue handling are an indication for antibiotics. Small clean surgical procedures do not need them.



FIG. 628. Slotted Egger's plates. (Courtesy London Splint Co. Ltd.)

useful in permitting a range of screw positions, and enabling the bone ends to be pushed together before the screws are tightened (Fig. 628). Plates are essential where there is a gap to be bridged, or in transverse and near transverse fractures.

#### (5) Intramedullary

**Nails.**—The tri-fin nail of Smith-Petersen was the first variety of intramedullary nail, designed particularly for use in the femoral neck (q.v.). Since the work of Küntscher longer nails for use in the shafts of long bones have been introduced (Fig. 629). They are straight or curved according to the bone for which they are designed. They are inserted into the medullary cavity at chosen sites, e.g. the tibial tuberosity, or the great trochanter, and thence passed across the fracture line. They are intended to get a firm grip on the cancellous bone inside the shaft; nails of correct diameter are therefore carefully chosen, otherwise they may jam or be loose and thus ineffective. A double exposure of bone is required, once at the site of injury for reduction, and once at the site of introduction of the nail. Correctly inserted Küntscher nails produce a very stable limb capable of early use, and they are of particular value in fractures of the femur. (6) *Bone grafts* are a form of internal fixation. They are usually fixed with screws like plates, but they are often partially buried in the medullary cavity.

The main factors influencing the choice of the method of fixation are :

(1) The particular fracture. Special apparatus has been designed for some fractures, notably the Smith-Petersen pin for the fractured femoral neck.

(2) The shape of the fracture.

- |                |                       |
|----------------|-----------------------|
| (a) Transverse | } Plate and screws or |
| (b) Comminuted |                       |
| (c) Spiral     | } Two                 |
| (d) Oblique    |                       |
- screws.

(3) The object of the operation, e.g. approximation of bone ends, early ambulation, or treatment of non-union.



FIG. 629. Types of Küntscher nails, for intramedullary fixation. (Courtesy London Splint Co. Ltd.)

Excessive tissue reaction occurred around the metals used in the past, but the two metals now in general use, Vitallium, an alloy, which can be moulded, but is a little brittle, and En 58 J stainless steel (18-8 S Mo) cause little or no tissue reaction, and may therefore be left permanently *in situ*. Removal of fixative agents may be rendered necessary by infection, atrophic non-union, or extrusion of the metal externally or on the surface of a joint due to bone absorption, e.g. as may occur with a Smith-Petersen pin. Occasionally a screw may be inserted to restrict movement, but may have to be removed to permit full joint function, e.g. in fractures of the fibular malleolus, or dislocations of the acromioclavicular joint. Patients often demand the removal of palpable screws, though they may cause no pain.

Operative fixation of a fracture may often be undertaken immediately, for it is the method of choice. There is much to gain in comfort, and repeated manipulation is avoided. Sometimes when manipulation is likely to be successful, and retention in splints or by skeletal traction satisfactory, the decision regarding operation may be delayed to see if manipulative reduction achieves the desired result. If it fails open operation may then be recommended. In some cases the degree of shock may enjoin some delay. Late operation is necessary when retention has failed to check the appearance of deformity, or when it is evident that non-union will occur.

Scrupulous asepsis is vital. Preparation of the part may be done in the ward, or in the theatre; the latter is chosen if there is difficulty in handling the limb, or much pain is likely to be inflicted. Operations on limbs may be facilitated by application of a tourniquet. At the end of operation, hæmostasis must be ensured to avoid the risk of tension on the skin edges. In a few smaller procedures such as screwing the medial malleolus, the tourniquet may be left on till the wound is dressed, but the skin suturing must never be blood-tight. When there is doubt about hæmostasis, the wound is drained for twenty-four hours. The soft tissues should be supported and protected by even bandaging and the use of a plaster cast, or splint, even if the limb is already stable. When any post-operative swelling has subsided, splints may be removed if the fracture needs no external support. Limbs should be elevated to reduce swelling. The arm may be raised on pillows, and the leg on splints; although raising the foot of the bed may suffice for the leg.

The use of post-operative antibiotics should not become a matter of routine (see *Antibiotic Therapy*, Chap. 110), but they are given to patients with compound fractures and when infection is suspected. Major operations in which there is long exposure in the theatre or in which there is much soft tissue handling are an indication for antibiotics. Small clean surgical procedures do not need them.

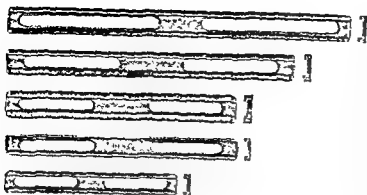


FIG. 628. Slotted Egger's plates. (Courtesy London Splint Co. Ltd.)

useful in permitting a range of screw positions, and enabling the bone ends to be pushed together before the screws are tightened (Fig. 628). Plates are essential where there is a gap to be bridged, or in transverse and near transverse fractures. (5) *Intramedullary*

**Nails.**—The tri-fin nail of Smith-Petersen was the first variety of intramedullary nail, designed particularly for use in the femoral neck (q.v.). Since the work of Küntscher longer nails for use in the shafts of long bones have been introduced (Fig. 629). They are straight or curved according to the bone for which they are designed. They are inserted into the medullary cavity at chosen sites, e.g. the tibial tuberosity, or the great trochanter, and thence passed across the fracture line. They are intended to get a firm grip on the cancellous bone inside the shaft; nails of correct diameter are therefore carefully chosen, otherwise they may jam or be loose and thus ineffective. A double exposure of bone is required, once at the site of injury for reduction, and once at the site of introduction of the nail. Correctly inserted Küntscher nails produce a very stable limb capable of early use, and they are of particular value in fractures of the femur. (6) *Bone grafts* are a form of internal fixation. They are usually fixed with screws like plates, but they are often partially buried in the medullary cavity.

The main factors influencing the choice of the method of fixation are :

(1) The particular fracture. Special apparatus has been designed for some fractures, notably the Smith-Petersen pin for the fractured femoral neck.

(2) The shape of the fracture.

- |                |                       |
|----------------|-----------------------|
| (a) Transverse | } Plate and screws or |
| (b) Comminuted |                       |
| (c) Spiral     | } Two                 |
| (d) Oblique    |                       |
- screws.

(3) The object of the operation, e.g. approximation of bone ends, early ambulation, or treatment of non-union.



FIG. 629. Types of Küntscher nails, for intramedullary fixation. (Courtesy London Splint Co. Ltd.)

Excessive tissue reaction occurred around the metals used in the past, but the two metals now in general use, Vitallium, an alloy, which can be moulded, but is a little brittle, and En 58 J stainless steel (18-8 S Mo) cause little or no tissue reaction, and may therefore be left permanently *in situ*. Removal of fixative agents may be rendered necessary by infection, atrophic non-union, or extrusion of the metal externally or on the surface of a joint due to bone absorption, e.g. as may occur with a Smith-Petersen pin. Occasionally a screw may be inserted to restrict movement, but may have to be removed to permit full joint function, e.g. in fractures of the fibular malleolus, or dislocations of the acromioclavicular joint. Patients often demand the removal of palpable screws, though they may cause no pain.

Operative fixation of a fracture may often be undertaken immediately, for it is the method of choice. There is much to gain in comfort, and repeated manipulation is avoided. Sometimes when manipulation is likely to be successful, and retention in splints or by skeletal traction satisfactory, the decision regarding operation may be delayed to see if manipulative reduction achieves the desired result. If it fails open operation may then be recommended. In some cases the degree of shock may enjoin some delay. Late operation is necessary when retention has failed to check the appearance of deformity, or when it is evident that non-union will occur.

Scrupulous asepsis is vital. Preparation of the part may be done in the ward, or in the theatre; the latter is chosen if there is difficulty in handling the limb, or much pain is likely to be inflicted. Operations on limbs may be facilitated by application of a tourniquet. At the end of operation, hæmostasis must be ensured to avoid the risk of tension on the skin edges. In a few smaller procedures such as screwing the medial malleolus, the tourniquet may be left on till the wound is dressed, but the skin suturing must never be blood-tight. When there is doubt about hæmostasis, the wound is drained for twenty-four hours. The soft tissues should be supported and protected by even bandaging and the use of a plaster cast, or splint, even if the limb is already stable. When any post-operative swelling has subsided, splints may be removed if the fracture needs no external support. Limbs should be elevated to reduce swelling. The arm may be raised on pillows, and the leg on splints; although raising the foot of the bed may suffice for the leg.

The use of post-operative antibiotics should not become a matter of routine (see *Antibiotic Therapy*, Chap. 110), but they are given to patients with compound fractures and when infection is suspected. Major operations in which there is long exposure in the theatre or in which there is much soft tissue handling are an indication for antibiotics. Small clean surgical procedures do not need them.



## CHAPTER 95

# THE COMPLICATIONS OF BONY INJURIES

### IMMEDIATE COMPLICATIONS

**Injury to Nerves** (see Chap. 61).

**Injury to Blood Vessels** (see under *Peripheral Vascular Surgery*).

**Injuries to Viscera.** They may occur independently of fractures or be associated with them, e.g. rupture of the spleen and fracture of the ribs on the left side. Visceral injuries may affect the patient's general condition, and may be one cause of failure of recovery from shock, or of the blood pressure falling in spite of apparently adequate transfusion. On the other hand, they may merely be a complication of the local condition, e.g. rupture of the urethra in fracture of the pelvis.

**Injuries to Joints.** A fracture into a joint, however carefully reduced, usually produces damage to the articular surface. Only in children can repair of articular surfaces occur, and even then it is often incomplete. The gliding movement of the joint is impeded to a variable degree, and eventually osteo-arthritis develops. The possible *immediate* complications of fractures into joints are : (1) *Hæmarthrosis*.—This may need aspiration to relieve pain. (2) Separation of a *loose body* into the joint. This requires removal. (3) *Displacement of an epiphysis into the joint*, e.g. the medial epicondyle of the elbow. This needs replacement. (4) Damage to the *articular cartilage*. It may not become apparent till later, e.g. traumatic chondromalacia of the patella. (Open wounds of joints and ligamentous injuries are described under *compound fractures*.)

**Injuries to Tendons.** All divided tendons should be repaired by suture with stainless steel wire or fine silk. Whether they can be safely repaired at once depends on whether the wound is likely to have been contaminated and the effectiveness of the skin cover. When both are in doubt, in crushing injuries of the hand, tendon repair should be delayed (secondary suture).

### DELAYED COMPLICATIONS

**Infection.** The problems of infection including that caused by tetanus and gas gangrene are discussed under *compound fractures*. Following an injury associated with a large hæmatoma, there may be a rise of temperature of as much as 2° on the first two or three evenings, but the patient's general condition remains good. This should not necessarily cause suspicion of infection.

**Myositis Ossificans and Ossifying Hæmatoma.** True myositis ossificans is very rare ; there are changes in the intramuscular connective tissue,

leading to new bone formation. It is accompanied by severe swelling, local heat, and sometimes pyrexia. After a few days, deposition of calcium may be seen in the muscles in the direction of the muscle fibres (Fig. 630). The brachialis is the muscle most often affected. An ossifying hematoma is much more commonly seen, and may be continuous with the fracture site or separated from it by soft tissue (Fig. 631). It is a natural but uncontrolled extension of the normal process of repair. It is of no importance provided it does not interfere with function. At first, the ossific deposit has radiologically soft edges due to active tissue changes at its periphery; interference at this stage increases tissue activity and produces more widespread new bone formation. Later, when the edges of the new bone are dense and well defined, the fragment may be removed if it interferes with function.



FIG. 630. Myositis ossificans. Note that the calcification is laid down along the lines of the muscle fibres. (From "A Complete Outline of Fractures," by J. G. Bonnin, by courtesy of William Heinemann Ltd.)

**Fat Embolism.** Fat, in microscopic drops, gains entry into the circulation, but whether this is due to mechanical causes, or to some failure of the fat-carrying mechanism of the blood, or a combination of both, is not known. The result, however, is the appearance of drops of fat in the sputum or the urine, and the development of a syndrome, in which the dominant features may be cerebral or pulmonary. Fat embolism occurs on the first to the third day after injury and is often confused with shock. Many mild cases recover. No satisfactory treatment exists.

**Myositis Fibrosa (Volkmann's Contracture).** This condition is distinguished from other types of fibrosis in muscle by the contracture which follows, and, when severe, by the consequent limitation of movement of joints. It occurs most commonly in the flexor muscles of the forearm, although it may occur in the leg where it tends to be unrecognised. The condition is always associated with injury, and often with vascular and nervous phenomena in the affected limb. There is no doubt of its association with tight splinting, and its rare occurrence nowadays is largely due to recognition of this fact. Three stages in the disease may be

recognised : (1) The patient complains of severe pain in the limb within eight to forty-eight hours of injury, or after manipulation of an injury or change of treatment ; the hand is swollen and engorged ; and there is limitation of finger movement, which is painful. The radial pulse may be absent or diminished. Sensory disturbance of the hand may be found, and may sometimes mask the pain. Recovery from this stage may be complete, or localised contracture of a few muscle segments may develop, causing interference with finger function only. (2) If the condition develops further the forearm becomes very tense and swollen ; finger



FIG. 631. An ossifying hematoma in the brachialis. The outline of the fragment is definite, and active new bone formation has therefore ceased. (From "A Complete Outline of Fractures," by J. G. Bonnin, by courtesy of William Heinemann Ltd.)

movements become impossible, and the hand is held flexed. Attempts to straighten the fingers are excessively painful. Signs of interference with nerve function may be more marked. The muscles are tense, swollen and blue, due to congestion and multiple capillary hæmorrhages. (3) In the third stage, the muscle fibres lose their characteristics, and are invaded by lymphocytes and phagocytes. Slowly they are replaced by fibrous tissue and the muscle belly contracts. When the flexors of the forearm are affected, the fingers curl up in flexion, followed, if contracture continues, by flexion of the wrist. Characteristically, the patient can extend the fingers with the wrist flexed, or extend the wrist with the fingers flexed (Fig. 632), but cannot extend the wrist and fingers together. Theories. If the blood supply to a muscle is cut off by ligation of its

artery or its artery and vein, fibrosis without contracture occurs. Therefore there must be a special mechanism which produces the marked contractile qualities of the fibrous tissue in Volkmann's contracture. Arterial injury has been found in association with myositis fibrosa, and has led to localised arterial spasm (possibly associated with distal spasm chiefly involving the flexor muscles) being put forward as the cause of the condition. This would also explain the occasionally absent radial pulse.

*Treatment.* Two known predisposing factors must always be avoided, namely, tight splinting of any kind, particularly encircling plaster, and the maintenance of the acutely flexed position of the elbow when swelling is likely to develop. If flexion is necessary for the maintenance of the position of the fracture, it should be instituted gradually or in three or four days after injury when swelling has subsided.

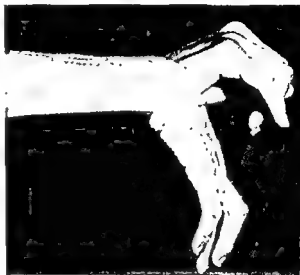


FIG. 632. Volkmann's ischaemic contracture. Note that in the flexed position of the wrist, the fingers can be extended, but as the wrist is dorsiflexed, the fingers are forced to flex.

The appearance of any signs of Volkmann's contracture is an indication for reduction, if not already carried out; it should be done at once with the minimum of manipulation, and the elbow should be loosely maintained at right angles. Reduction may release any pressure on the brachial artery or the veins and should free the collateral circulation. The arm should then be elevated to reduce further swelling. If the condition continues to develop in spite of good reduction, relaxation of splints and elevation of the arm, open exploration must be carried out, and indeed may be indicated earlier when there is severe hæmorrhage or a large hæmatoma suggesting arterial damage, and/or an absent radial pulse. Attention at operation is directed to two things, the state of the artery and its distal branches and the tension in the forearm fascia which may need a long incision to decompress the muscles. Nipping, kinking or spasm of the brachial artery has been found at operation.

In the advanced stage, attention can only be directed to prevention of contracture by use of light spring-loaded extension on the fingers. In severe cases, the ultimate outlook is poor, but many milder cases occur in which prompt treatment avoids severe contracture or limits it to one or two muscle segments.

**Traumatic Atrophy of Bone.** In all injuries in which a limb is immobi-

recognised : (1) The patient complains of severe pain in the limb within eight to forty-eight hours of injury, or after manipulation of an injury of the forearm; the hand is swollen and engorged; and there is a limitation of finger movement, which is painful. The radial pulse may be absent or diminished. Sensory disturbance of the hand may be found and may sometimes mask the pain. Recovery from this stage may be complete, or localised contracture of a few muscle segments may develop, causing interference with finger function only. (2) If the condition develops further the forearm becomes very tense and swollen; finger



FIG. 631. An ossifying hæmatoma in the brachialis. The outline of the fragment is definite, and active new bone formation has therefore ceased. (From "A Complete Outline of Fractures," by J. G. Bonnin, by courtesy of William Heinemann Ltd.)

movements become impossible, and the hand is held flexed. Attempts to straighten the fingers are excessively painful. Signs of interference with nerve function may be more marked. The muscles are tense, swollen and blue, due to congestion and multiple capillary hæmorrhages. (3) In the third stage, the muscle fibres lose their characteristics, and are invaded by lymphocytes and phagocytes. Slowly they are replaced by fibrous tissue and the muscle belly contracts. When the flexors of the forearm are affected, the fingers curl up in flexion, followed, if contracture continues, by flexion of the wrist. Characteristically, the patient can extend the fingers with the wrist flexed, or extend the wrist with the fingers flexed (Fig. 632), but cannot extend the wrist and fingers together. Theories. If the blood supply to a muscle is cut off by ligation of its

friction. The lesion is commonly seen in the ulnar nerve, due to distortion of the elbow joint and an increased carrying angle, but may occur following simple displacement of the epicondyle (see under *The Peripheral Nerves*, Chap. 61).

**Joint Stiffness.** *A. Solely from Effects of Injury.*—(1) Injury to the joint. Fractures may involve and thus cause irregularity of articular surfaces ; damage may be limited to the cartilaginous surface and so be radiologically invisible. Loose fragments or an epiphysis may be detached and lie in the joint. (2) Injury to the capsule. Extensive ligamentous injury, even if carefully repaired, may cause stiffness. (3) Injury to muscles and tendons. If these structures are bound down by fibrosis, movements of the adjacent joint are thereby restricted. The nearer the fibrosis to the joint, the greater is the limitation of movement. Adhesion of the quadriceps to the femur, following a fracture, is a good example. (4) The result of ossifying hæmatoma or myositis ossificans.

*B. From Exacerbation of Pre-existing Arthropathy.* Injury to and immobilisation of arthritic joints causes increased stiffness and pain. Either osteo-arthritis or rheumatoid arthritis may be made worse by injury of an affected joint.

*C. From Effects of Faulty Treatment.* Joint stiffness may result from : (1) Prolonged immobilisation. (2) Prolonged or too powerful traction involving a joint. (3) Extensive operative exposures through muscles (thus the quadriceps should be avoided by use of a posterior approach between the hamstrings). (4) Failure adequately to treat any infection of, or near a joint.

**Traumatic Arthritis.** The late degenerative changes in joints due to any of the causes outlined above are similar to those of osteo-arthritis and call for the same treatment. They may arise from : (a) Direct damage to the joint ; (b) abnormal stresses on the joint produced by malunion of a bone (usually one of the lower limbs because of the effect of weight-bearing) ; or (c) excessive stresses on a joint of one leg as the result of injury of the other.

**Oedema and Vascular Disturbances.** After immobilisation, the circulatory tone of a limb is temporarily impaired, and thus oedema occurs when the limb is dependent. This is aggravated if there has been any soft tissue injury which has interfered with venous or lymphatic return. The time taken for oedema to subside is roughly equal to the period of immobilisation of the part. Patients should be warned of its likelihood, and should be told to rest with the leg elevated ; elastic bandages are worn till tone returns to the vascular system.

## DELAYED UNION AND NON-UNION

**Delayed Union.** The causes of delayed union are substantially the same as those of non-union. Attention has already been drawn to the delay

lised as part of treatment, post-traumatic atrophy occurs, indicated by muscular wasting and slight often spotty decalcification of the bones. Occasionally this decalcification becomes abnormal in degree, is concentrated around nearby joints, and is accompanied by circulatory

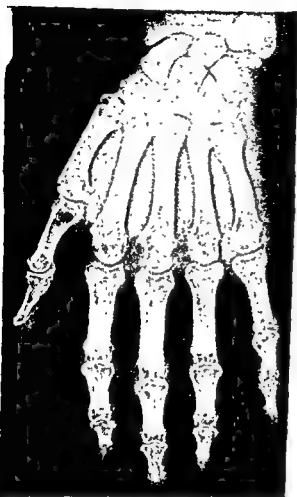


FIG. 633. Sudeck's atrophy (traumatic atrophy of bone) in the wrist and fingers. Note the mottled decalcification around the joints.

disturbances, namely, vasodilatation and oedema (Fig. 633). As the swelling subsides periarticular thickening, limited articular movement, thinning and atrophy of the skin and hyperhidrosis are observed. Pain may be severe in the early stages. The severity of the condition bears no relationship to the severity of the original injury. Young people are less often affected than older people. The known predisposing factors are tight plasters and multiple reductions. Sympathectomy has been tried without real success. Physiotherapy is given to try to overcome the inevitable stiffness of the affected joints. Months of treatment may be necessary before the condition subsides.

**Avascular Necrosis.** This has already been discussed together with the factors influencing union (Chap. 92). Because of the time necessary for the typical radiological changes to develop, the condition is not

diagnosed until between the fourth and the tenth week after injury. It is an important cause of non-union, or the later development of osteoarthritis of an affected joint, e.g. the hip in fracture of the femoral neck.

**Involvement of a Nerve in Callus.** This very rare condition may be met with in fractures of the humerus. Radial nerve paralysis appears in about the fourth week. Spontaneous recovery occurs, but is slow, i.e. up to a year. Some surgeons prefer to explore and free the nerve.

#### LATE COMPLICATIONS

**Late "Neuritis."** This may follow injuries which elongate or roughen the path of a nerve, so that it is continually subjected to abnormal

of the lesion. Scurvy, tabes, and rapidly growing tumours are almost the only conditions causing absolute non-union. Deep X-ray therapy given for malignant tumours of bone commonly causes non-union of associated fractures.

**Established Non-union.** It takes a long time for the characteristics of this condition to be fully established clinically. Therefore, as early diagnosis is the first step towards rectifying the condition, it is based on the radiological appearances. The tell-tale features of Nature's acceptance

of the ununited state are the tidying up and organisation of the bone ends, which assume sharply defined outlines and are covered with a thin layer of organised bone (Fig. 634).

Healthy active callus margins are radiologically soft, but are continuous across the fracture line. In non-union the organisation of the fracture ends varies. Three main clinical types are recognised: (1)

*Absolute Non-union.*—This may be due to interposition of soft tissue between the bone ends; the fragments are therefore characteristically loose and lacking in any rigidity. Atrophic non-union may produce this picture. (2)

*Fibrous Non-union.*—The fracture surfaces are united by firm fibrous tissue which imparts some rigidity to the junction, but the bone ends show such organisation that it is unlikely that new bone formation will bridge

the gap. Such a condition may follow hypertrophic callus forma-

tion (*vide infra*). (3) *False Joint Formation.*—This represents a further stage of organisation in which fibrous tissue forms the capsule of the false joint; one of the bone ends tends to become concave and the other convex, and both ends become covered with cartilage. Thus a true joint is closely imitated. The formation of a false joint is often associated with lack of fixation of the part, but other factors may play a role. These stages of non-union are readily distinguished radiologically, and, as indicated above, show some relationship to the amount of callus formation.

From the point of view of treatment, the amount of callus formed is important. Thus two kinds of non-union are recognised: (1) *Atrophic non-union*, in which the bone ends are scarcely expanded by the callus (Fig. 634). (2) *Hypertrophic non-union*, in which the bone ends are



FIG. 634. Atrophic non-union of a fracture of the tibia and fibula. Note the small quantity of new bone, and the organised closure of the medullary cavities. Established non-union.



that occurs in diagnosing union if radiological union is the criterion rather than clinical union. The rate of union varies with the bone, the age of the patient and the type of fracture. It is thus impossible to fix any specific period beyond which union may be said to be delayed, except on the basis of long experience.

**Non-union.** The criteria for non-union are not related to time, but to radiological appearances. Thus it may be possible to establish the diagnosis of non-union (i.e. permanent cessation of repair) before the period during which the diagnosis of delayed union would be tenable has elapsed. The causes of difficulty in obtaining union may be general or local. General causes are few; thus active syphilis is almost alone in preventing union. The local causes are many (among them must be rated highly the effects of injudicious treatment): (1) *Infection*.—This is a serious cause of non-union, for it adds chronic osteomyelitis to any other difficulties. (2) *Interposition of Soft Parts*.—When living tissue such as muscle is interposed, its rapid adhesion to the bone ends may reduce the amount of callus formed and thus delay union, or produce a line of organised fibrous tissue which prevents union altogether. A similar effect is produced when a fragment of compact bone is jammed across the medullary canal of one of the fragments. Removal of the block is necessary. (3) *Wide Separation of the Bone Ends*.—This may be brought about by loss of bone in compound fractures, by excessive distraction with skeletal traction, or by fixation of the bone ends apart by internal fixation badly applied. (4) *Constant Movement*.—This is a debatable point. Ribs though constantly moving unite. But it is idle to pretend that the best fitting plaster does more than prevent excessive movement. Some movement, e.g. as during weight-bearing in a walking plaster, seems to encourage union. Excessive movement, especially in the presence of another deleterious factor, may produce non-union or a false joint. (5) *Interference with the Blood Supply*.—This is an important cause of non-union, particularly in young people, in whom the force necessary to produce a fracture often causes widespread soft tissue damage. It may take three forms: (a) *Avascular necrosis*. One fragment may be cut off entirely from its blood supply, e.g. some fractures of the femoral neck. Union cannot possibly occur. (b) *Double fractures*. The shaft of a long bone, commonly the tibia, may be broken in two parts, leaving a central segment of bone isolated, and dependent for its blood supply on surface adhesions. In the tibia, the upper fracture usually unites, but non-union of the lower fracture is common; when union does occur it is always delayed. (c) *Stripping of the bone ends*. If the soft tissues are widely torn off the bone ends when they are displaced at the time of fracture, necrosis of the surface cells may occur with resultant failure of formation of effective callus, and consequent atrophic non-union. (6) *Local Disease*.—Cysts and tumours—a cause of pathological fracture—may delay union, but few cause non-union unless there is very active growth

the medullary cavity. (3) The fixation of the part by internal means to restrict movement, e.g. by a metal plate or intramedullary nail. (4) The addition of fresh bony material to form a matrix on which new bone may form. This may be done by the use of cancellous bone chips packed around the bone ends, which are internally splinted, or by use of a compact bone graft, thus combining the addition of new bone with internal fixation. Any one of these methods may be used alone, but generally a combination of them is used according to circumstances. Thus in a fracture of the humerus, in which a little length may be lost without loss of function, a step-cut operation, in which the fragments are fixed with two cross screws, may be sound and technically the easiest procedure. In the leg, when bone has been lost, a gap may be filled with cancellous bone chips packed around a Küntscher nail, or by a long compact bone graft screwed into place.

A bone graft may be obtained from the same limb, the ilium, or from a bone-bank. Fresh bone is more satisfactory than stored bone, and cancellous bone more satisfactory than compact bone. From the technical point of view, the following types of graft may be distinguished: (1) *Cancellous Chip Grafts*.—Broken fragments of cancellous bone are packed around the freshened bone ends, which may be retained in position by internal fixation. (2) *Onlay Grafts*.—After the bone ends have been denuded, a graft of compact bone is laid across the fracture and screwed in position. The disadvantages are that the width of the bone is increased and union is encouraged only along one side of the bone. (3) *Inlay Grafts*.—A gutter is cut in continuity along the length of both fragments of the fractured bone. Two-thirds of the length of the gutter are cut on one side of the fracture and one-third is cut on the other. The two pieces of the graft thus formed are reversed in the gutter so that the longer piece bridges the fracture (Fig. 636). The shorter piece is placed in the remaining part of the gutter and both pieces are then screwed in place. This method provides firm internal fixation and a combination of compact and cancellous bone grafting. (4) *Massive Sliding Grafts*.—In this method the full width of the bone is employed for the graft, and the bone is split in half so that two-thirds of the length of the graft are cut from one fragment and one-third is cut from the other. As in inlay grafting, the pieces are then reversed in position so that the longer piece bridges the fracture. The graft is fixed with screws. The advantages gained are the greater strength of the graft, and the ease of fitting flat surfaces together.

**Malunion.** This may take the form of any of the previously mentioned displacements to which a fracture is liable, singly or in combination, and is due to a failure of treatment. In addition, there may be excessive formation of callus which may obstruct movement of an adjacent joint,

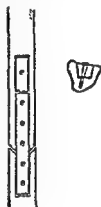


FIG. 636. The sliding inlay bone graft.

surrounded by a mass of callus, which, however, is divided by a layer of fibrous tissue with well-defined limits (Fig. 635).

Atrophic non-union may follow fractures in which there is widespread stripping of the bone ends of their soft tissue cover; the fractures are not necessarily compound. Hence probably arises the bad reputation of fractures at the junction of the lower and middle thirds of the tibia and the junction of the upper and middle thirds of the humerus in young men; both of these fractures require great force and therefore there is likely to be much stripping of soft tissue. In atrophic non-union, open procedures are indicated, as there is practically no tendency to new bone formation. In hypertrophic non-union, the cause is inexplicable. The gap to be bridged, compared with the gap already bridged, is often very small. Some last-minute failure of fibroblasts to become osteoblasts must have occurred. Hope of union by simple treatment still exists. Attempts to stimulate union may be made by the following methods: (1) In the leg, simple fixation in a walking plaster and encouragement of use of the limb;

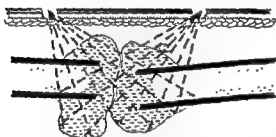


FIG. 635. Hypertrophic non-union. There is considerable new bone formation around the bone ends, but an irregular line of separation runs between them. Consolidation eventually occurs in many cases, and is assisted by weight bearing. In established non-union, multiple drilling of the callus from two skin incisions (Beck's bone drilling) often causes union to proceed.

the compression effect of weight-bearing is particularly stimulating. In the arm, use of the limb may cause indolent callus in a fracture of the humerus to unite. (2) Hammering the fracture. With the object of producing a fresh hæmatoma around the fracture, it is forcibly stretched, and after being covered with a few layers of Esmarch's rubber bandage, is given a good local hammering. It is then enclosed in plaster, allowing for the likely swelling, and progress watched. (3) Beck's bone drilling (Fig. 635). This is an open and more certain method of producing a fresh hæmatoma at the fracture site. The fracture is exposed through one or two small incisions on each side, and the hypertrophic callus is pierced in many directions by a small-bore drill. The medullary cavities are thus opened up; bone drill-dust is scattered throughout the plane of non-union, and fresh hæmorrhage occurs around the ends of the fragments. With further immobilisation there is a very good chance of successful union being achieved.

If there is a false joint or atrophic non-union, or the methods indicated above have failed in hypertrophic non-union, open operation must be undertaken. The aims of internal fixation are: (1) Close approximation of the bone ends by excision of all interposed tissue. (2) Freshening of the bone ends by adequate clearing of callus and fibrous tissue, and reopening

the medullary cavity. (3) The fixation of the part by internal means to restrict movement, e.g. by a metal plate or intramedullary nail. (4) The addition of fresh bony material to form a matrix on which new bone may form. This may be done by the use of cancellous bone chips packed around the bone ends, which are internally splinted, or by use of a compact bone graft, thus combining the addition of new bone with internal fixation. Any one of these methods may be used alone, but generally a combination of them is used according to circumstances. Thus in a fracture of the humerus, in which a little length may be lost without loss of function, a step-cut operation, in which the fragments are fixed with two cross screws, may be sound and technically the easiest procedure. In the leg, when bone has been lost, a gap may be filled with cancellous bone chips packed around a Küntzner nail, or by a long compact bone graft screwed into place.

A bone graft may be obtained from the same limb, the ilium, or from a bone-bank. Fresh bone is more satisfactory than stored bone, and cancellous bone more satisfactory than compact bone. From the technical point of view, the following types of graft may be distinguished: (1) *Cancellous Chip Grafts*.—Broken fragments of cancellous bone are packed around the freshened bone ends, which may be retained in position by internal fixation. (2) *Onlay Grafts*.—After the bone ends have been denuded, a graft of compact bone is laid across the fracture and screwed in position. The disadvantages are that the width of the bone is increased and union is encouraged only along one side of the bone. (3) *Inlay Grafts*.—A gutter is cut in continuity along the length of both fragments of the fractured bone. Two-thirds of the length of the gutter are cut on one side of the fracture and one-third is cut on the other. The two pieces of the graft thus formed are reversed in the gutter so that the longer piece bridges the fracture (Fig. 636). The shorter piece is placed in the remaining part of the gutter and both pieces are then screwed in place. This method provides firm internal fixation and a combination of compact and cancellous bone grafting. (4) *Massive Sliding Grafts*.—In this method the full width of the bone is employed for the graft, and the bone is split in half so that two-thirds of the length of the graft are cut from one fragment and one-third is cut from the other. As in inlay grafting, the pieces are then reversed in position so that the longer piece bridges the fracture. The graft is fixed with screws. The advantages gained are the greater strength of the graft, and the ease of fitting flat surfaces together.

**Malunion.** This may take the form of any of the previously mentioned displacements to which a fracture is liable, singly or in combination, and is due to a failure of treatment. In addition, there may be excessive formation of callus which may obstruct movement of an adjacent joint,

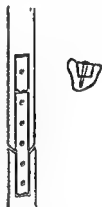


FIG. 636. The sliding inlay bone graft.

or constitute an unsightly deformity on an exposed bone such as the clavicle. When callus is *fully* organised, any excess may be safely removed.

When union has occurred with deformity, a decision has to be taken as to whether operative correction is worth while. In the arm, some deformity can be tolerated as it interferes little with function. In the forearm, pronation and supination are interfered with, and this may be serious in a manual worker. In the lower limb, the line of transmission of the body weight is important, and deviations of any magnitude from normal, by altering the sites of pressure in the joints, eventually lead to osteo-arthritis. Whereas young people correct moderate deformities as they grow, in older people deformities progressively deteriorate and correction is therefore worth while, though in the very old the advantages gained may not be worth the extra period of post-operative immobilisation and consequent undesirable effects.

## CHAPTER 96

### COLLES' FRACTURE AND FRACTURES OF THE WRIST, HAND AND FOREARM

THE commonest fracture encountered is the fracture of the lower end of the radius, which bears the name of Colles, who in 1814 first elucidated this injury. Hitherto it had been regarded as some form of dislocation, for it is an impacted fracture, and crepitus is characteristically absent. Colles pointed out that if the deformity is reduced by traction, crepitus can then always be elicited. It is produced by falls on the outstretched hand and is most commonly encountered in obese middle-aged women



FIG. 637. The clinical appearance of a Colles' fracture. The "dinner fork" deformity.

with mild osteoporosis. The clinical appearance of the fracture is adequately described by the term dinner fork deformity (Fig. 637), but less marked displacements occur, and the diagnosis may then depend on radiography.

The fracture runs across the lower end of the radius about 1 inch or less from the articular surface, and is often accompanied by avulsion of the tip of the styloid process of the ulna. Normally the distal end of the radius, in contact with the proximal row of carpal bones, shows two planes of obliquity. The articular surface slopes so that the radial styloid is  $\frac{1}{2}$  inch distal to the ulnar styloid, and it also slopes so as to look partly volarwards. This double obliquity determines the displacement of the

or constitute an unsightly deformity on an exposed bone such as the clavicle. When callus is *fully* organised, any excess may be safely removed.

When union has occurred with deformity, a decision has to be taken as to whether operative correction is worth while. In the arm, some deformity can be tolerated as it interferes little with function. In the forearm, pronation and supination are interfered with, and this may be serious in a manual worker. In the lower limb, the line of transmission of the body weight is important, and deviations of any magnitude from normal, by altering the sites of pressure in the joints, eventually lead to osteo-arthritis. Whereas young people correct moderate deformities as they grow, in older people deformities progressively deteriorate and correction is therefore worth while, though in the very old the advantages gained may not be worth the extra period of post-operative immobilisation and consequent undesirable effects.

of the fingers. The plaster must be well moulded around the base of the thumb, and extend to the metacarpal heads.

The first plaster applied after a fresh injury must always leave room for the swelling around the fracture. This may be done by : (a) Padding the plaster with an even layer of cotton wool around the fracture ; (b) splitting the plaster along the volar surface, so that it can expand (the plaster must then not be excessively thick) ; or (c) applying a thick plaster slab which encircles the wrist for three-quarters of its circumference, leaving a gap an inch or so wide on the volar aspect of the wrist ; this slab must be held in place by bandages while it is setting, but as bandages are also constrictive they must be cut and the plaster lightly rebandaged over a thin layer of cotton wool ; such a plaster may be reinforced by a circular plaster bandage after forty-eight hours when the swelling has subsided a



FIG. 639. A plaster applied for a Colles' fracture. Note the good functional position of the hand ; full flexion of the fingers should be possible.

little. The retentive powers of a forearm plaster are small, and, as swelling subsides rapidly, the plaster soon becomes ineffective. The plaster must therefore be renewed at the end of the first seven to ten days. This can be done under gentle traction without an anæsthetic if the position is still clinically and radiologically good. When reduction is felt to be stable, traction can be dispensed with and the wrist put up in the functional position.

All Colles' fractures do not require the same time in plaster, as they vary in severity, and the rate of union is affected by the age of the patient. The usual times are as follows :

	<i>Time in Plaster</i>	
	Under 40	Over 40
<i>Uncomminuted Fractures</i>		
(1) Undisplaced . . . . .	2-3 weeks	3-4 weeks
(2) Displaced . . . . .	3-4 weeks	4-5 weeks
<i>Comminuted Fractures</i>		
(1) With intact joint surface . . . . .	4-5 weeks	5-6 weeks
(2) With fissuring of the joint surfaces . . . . .	5-6 weeks	6-7 weeks



lower end of the radius in Colles' fracture in which the articular surface of the radius looks dorsally (Fig. 638), and at the same time the styloid process of the radius is displaced proximally and may reach the level of the ulnar styloid process. The fracture is divided clinically into two types, those with little displacement, in which reduction may not be needed, and those in which there is deformity, and perhaps associated injuries, such as fracture of the navicular or dislocation of the inferior radio-ulnar joint; often there is comminution of the lower end of the radius.

When there is marked deformity, the fracture must be reduced. In a recent case this can be carried out under local anaesthesia (injection of

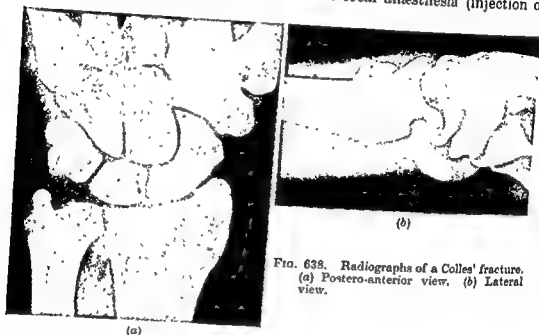


FIG. 638. Radiographs of a Colles' fracture. (a) Postero-anterior view, (b) Lateral view.

2 per cent. procaine into the fracture hæmatoma), although it is most often done under general anaesthesia. The fracture is first disimpacted by firm palmar flexion and traction on the wrist. Reduction is facilitated if the limb is fixed by a webbing band just above the elbow to a firm support such as a hook in the wall, and the patient's hand is grasped by the thumb with one hand and the radial three fingers with the other hand (leaving the little finger free avoids cramping the metacarpals). Reduction is maintained by traction, while a plaster cast is being applied. The plaster of Paris extends from the elbow to the metacarpal heads (Fig. 639). It should be sloped across the elbow to permit full flexion of the joint, and the band across the palm should permit full flexion of the fingers. When a plaster is being applied without an anaesthetic, full flexion of the fingers can be achieved by getting the patient to grasp the plaster firmly as it is setting. Opportunity should also be taken under these conditions to put the wrist in *slight* dorsiflexion, a position which encourages the better use

displacement a forearm plaster is applied in the functional position for three to four weeks. If there is subluxation, the wrist must be slightly flexed to the side of the lesion (as flexion to the opposite side displaces the fracture further), and the position carefully checked radiologically. *Vertical fissure fractures* of an incomplete type are sometimes encountered at the lower end of the radius.

### FRACTURES AND DISLOCATIONS OF THE CARPUS

**Fractures of the Navicular.** The bone most commonly fractured in the wrist is the navicular. It may be fractured by compression between the distal row of carpal bones and the radial styloid process, or by forced dorsiflexion of the wrist. As the navicular acts as a tie between the proximal and midcarpal regions, it is liable to be displaced by the latter mechanism. Two types of navicular injuries may therefore be distinguished: (1) *Fractures without Displacement.*—This is a fine fracture of trabecular bone (Fig. 640); the cartilaginous envelope of the bone often remains intact. In consequence, the fracture may not easily be seen at the first radiological examination, although an oblique view of the wrist may reveal it. When the diagnosis is in doubt the wrist is immobilised in plaster of Paris for a fortnight and then re-X-rayed; local bone absorption around a fracture will then make it visible. (2) *Fractures with Displacement* (Fig. 641).—These are notoriously difficult to treat and may be followed by non-union. The



FIG. 640. An undisplaced fracture through the waist of the carpal navicular.



FIG. 641. A fracture of the carpal navicular with displacement.

clinical signs in both types are pain, bruising and deep tenderness in the anatomical snuff box. The patient is unable to press firmly on the palm of the hand with the wrist dorsiflexed without producing pain. The diagnosis can only be made with certainty by radiological examination.

**Treatment.** In undisplaced fractures, the bone fragments are held in position by ligamentous and cartilaginous attachments, and therefore to obtain union it is only

Throughout treatment, particularly in the elderly, attention must be directed to seeing that the fingers are exercised fully, and that the shoulder, which is often bruised in the fall, is not allowed to become stiff while the forearm is carried in a sling. The sling should therefore be dispensed with when the swelling has subsided, and active use of the hand should be encouraged. When full activity entails getting the hand wet, a plastic splint may replace the plaster.

A well-reduced fracture whose position has been retained by a change of plaster and checked by radiography about the seventh to tenth day, should show no clinical deformity and radiologically there should be restoration of the length of the radius and of the volar tilt of the articular surface, and the dorsal surface of the bone should be smooth. In elderly people, this ideal may not be achieved, but as function is fairly good despite deformity, re-reduction need not be insisted on except in cases of severe malposition. Frequent reductions cause pain and stiffness and may lead to Sudeck's atrophy. Possible late complications are: (1) Pain over an ununited ulnar styloid process; this may be relieved by infiltration with hydrocortisone or it may subside with time. (2) Loss of pronation and supination; this occurs in severe fractures in which the lower radio-ulnar joint is involved in the comminution. (3) Traumatic arthritis of the wrist caused by damage to the joint surfaces; it may be bad enough to require a moulded leather splint or arthrodesis. (4) Rupture of the extensor pollicis longus tendon may eventually occur by attrition.

**Smith's Fracture (Syn. Reversed Colles' Fracture).** This fracture is produced by falls with the wrist flexed, and the displacement of the lower fragment is volarwards. The treatment is similar to that of a Colles' fracture.

**Other Fractures of the Lower End of the Radius. Epiphyseal Separation.**—This can occur up to the sixteenth year, and may be accompanied by separation of the lower ulnar epiphysis. The latter complication does not alter the treatment. The radial epiphysis may be displaced in any direction, and as it moves takes with it a wedge of diaphysis on the side to which it is displaced. This wedge is useful, for it prevents over-correction when the epiphysis is reduced by pressure in the opposite direction. In epiphyseal separation any crepitus obtained is soft. Separation always occurs on the diaphyseal side of the epiphysis. Repair is rapid, and is firm in three weeks. After reduction, a forearm plaster, as used for Colles' fracture, is applied. **Infraction Fractures.**—As a variant of the greenstick fracture in the young, there may be a transverse plane of impaction across the radius and an expanded ridge of cortical bone around it, resembling the ridge on a bamboo (hence the term bamboo fracture). No reduction is necessary, but a supporting plaster is applied for a fortnight. **Marginal Fractures.**—Fissure fractures, sometimes accompanied by subluxation of the wrist, may occur. The dorsal or volar margin of the radius or the radial styloid is separated. In the absence of

displacement a forearm plaster is applied in the functional position for three to four weeks. If there is subluxation, the wrist must be slightly flexed to the side of the lesion (as flexion to the opposite side displaces the fracture further), and the position carefully checked radiologically. *Vertical fissure fractures* of an incomplete type are sometimes encountered at the lower end of the radius.

## TRACTIONS AND DISLOCATIONS OF THE CARPUS

**Fractures of the Navicular.** The bone most commonly fractured in the wrist is the navicular. It may be fractured by compression between the distal row of carpal bones and the radial styloid process, or by forced dorsiflexion of the wrist. As the navicular acts as a tie between the proximal and midcarpal regions, it is liable to be displaced by the latter mechanism. Two types of



FIG. 640. An undisplaced fracture through the waist of the carpal navicular.

navicular injuries may therefore be distinguished: (1) *Fractures without Displacement*.—This is a fine fracture of trabecular bone (Fig. 640); the cartilaginous envelope of the bone often remains intact. In consequence, the fracture may not easily be seen at the first radiological examination, although an oblique view of the wrist may reveal it. When the diagnosis is in doubt the wrist is immobilised in plaster of Paris for a fortnight and then re-X-rayed; local bone absorption around a fracture will then make it visible. (2) *Fractures with Displacement* (Fig. 641).—These are notoriously difficult to treat and may be followed by non-union. The



FIG. 641. A fracture of the carpal navicular with displacement.

clinical signs in both types are pain, bruising and deep tenderness in the anatomical snuff box. The patient is unable to press firmly on the palm of the hand with the wrist dorsiflexed without producing pain. The diagnosis can only be made with certainty by radiological examination.

**Treatment.** In undisplaced fractures, the bone fragments are held in position by ligamentous and cartilaginous attachments, and therefore to obtain union it is only

Throughout treatment, particularly in the elderly, attention must be directed to seeing that the fingers are exercised fully, and that the shoulder, which is often bruised in the fall, is not allowed to become stiff while the forearm is carried in a sling. The sling should therefore be dispensed with when the swelling has subsided, and active use of the hand should be encouraged. When full activity entails getting the hand wet, a plastic splint may replace the plaster.

A well-reduced fracture whose position has been retained by a change of plaster and checked by radiography about the seventh to tenth day, should show no clinical deformity and radiologically there should be restoration of the length of the radius and of the volar tilt of the articular surface, and the dorsal surface of the bone should be smooth. In elderly people, this ideal may not be achieved, but as function is fairly good despite deformity, re-reduction need not be insisted on except in cases of severe malposition. Frequent reductions cause pain and stiffness and may lead to Sudeck's atrophy. Possible late complications are: (1) Pain over an ununited ulnar styloid process; this may be relieved by infiltration with hydrocortisone or it may subside with time. (2) Loss of pronation and supination; this occurs in severe fractures in which the lower radio-ulnar joint is involved in the comminution. (3) Traumatic arthritis of the wrist caused by damage to the joint surfaces; it may be bad enough to require a moulded leather splint or arthrodesis. (4) Rupture of the extensor pollicis longus tendon may eventually occur by attrition.

**Smith's Fracture (Syn. Reversed Colles' Fracture).** This fracture is produced by falls with the wrist flexed, and the displacement of the lower fragment is volarwards. The treatment is similar to that of a Colles' fracture.

**Other Fractures of the Lower End of the Radius. Epiphyseal Separation.**—This can occur up to the sixteenth year, and may be accompanied by separation of the lower ulnar epiphysis. The latter complication does not alter the treatment. The radial epiphysis may be displaced in any direction, and as it moves takes with it a wedge of diaphysis on the side to which it is displaced. This wedge is useful, for it prevents over-correction when the epiphysis is reduced by pressure in the opposite direction. In epiphyseal separation any crepitus obtained is soft. Separation always occurs on the diaphyseal side of the epiphysis. Repair is rapid, and is firm in three weeks. After reduction, a forearm plaster, as used for Colles' fracture, is applied. **Infraction Fractures.**—As a variant of the greenstick fracture in the young, there may be a transverse plane of impaction across the radius and an expanded ridge of cortical bone around it, resembling the ridge on a bamboo (hence the term bamboo fracture). No reduction is necessary, but a supporting plaster is applied for a fortnight. **Marginal Fractures.**—Fissure fractures, sometimes accompanied by subluxation of the wrist, may occur. The dorsal or volar margin of the radius or the radial styloid is separated. In the absence of

similar strain fracture of the posterior tubercle of the triquetrum may also be encountered; union of the tubercle does not always occur.

**The Lunate.** This bone is occasionally fractured in compression injuries, and may undergo avascular necrosis, producing patchy sclerosis and carpal arthritis known as Kienbock's disease. The treatment is excision of the bone. More commonly the bone is dislocated and comes to lie among the flexor tendons, with resultant interference with their function and sometimes pressure on the median nerve (Fig. 613). At first, the bone can usually be reduced if the wrist joint is opened up by skeletal traction. One wire is passed through the olecranon and the other through the metacarpal heads. If manipulative reduction fails, excision is recommended, for open reduction is apt to be followed by necrosis.

**Dislocation of the Wrist.** This may be volar or dorsal, but is more commonly the latter. It may be confused with a severe Colles' fracture. It is easily reduced and retained. More complicated paths of separation may be followed, leaving the lunate attached to the radius and passing through the waist of the navicular. Reduction in these cases is usually simple, but there is a risk of non-union of the fracture or avascular necrosis of the navicular and/or lunate.

**Fractures of the Metacarpals.** Fracture of the first metacarpal is important, for as the bone is unsupported by the other metacarpals, the fragments are liable to displacement, and consequent malunion seriously affects the function of the hand. There are fractures of the shaft, which may be spiral, oblique or comminuted, and fractures of the base, which are often impacted and are then readily treated by immobilisation on a finger wire; they often require little reduction as they are little displaced. There is also a marginal fracture of the saddle-shaped base of the metacarpal (Fig. 643); by reducing the grip of the bone on the trapezium (os multangulum majus) it often causes a subluxation of the carpo-metacarpal joint (*Bennett's stove fracture*). This is a fracture dislocation and has earned the reputation of being difficult to retain. Two forces are needed to produce stable reduction, namely, traction on the thumb, and lateral pressure against the base of the metacarpal. This is easily achieved by applying a finger wire along the dorsum of the thumb, and incorporating it in a forearm plaster as a foundation (Fig. 644). If the wire is covered with strapping so that the thumb



FIG. 643. Bennett's stove fracture of the thumb. Note the marked dislocation of the first metacarpal.

necessary to fix the wrist and the base of the thumb in a Colles' type of plaster. Firm union usually occurs in seven weeks, but must be checked radiologically. In displaced fractures, much firmer immobilisation of the wrist is necessary, and the plaster should therefore enclose the thumb as far as the interphalangeal joint. At least ten weeks are required for union, which must be checked radiologically at the end of this period.

Union is judged to have occurred by absence of the signs of non-union (*vide infra*), and the presence of bony trabeculation across the fracture site. When union has occurred, the plaster may be discarded. If union is firm, light use of the wrist is followed by steady improvement in function. Deterioration in function or persistence of pain call for careful reassessment, as they may be due to non-union.

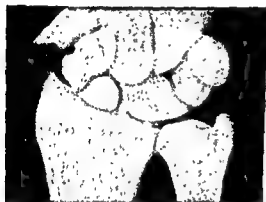


Fig. 642. An ununited fracture of the carpal navicular. Note the broad fracture line, with organised sclerotic surfaces, and the increased density affecting the proximal fragment (avascular necrosis).

*Non-union.* The signs of non-union consist of a gap at the fracture site (Fig. 642). If the adjacent surfaces of the fragments are still active, they are radiologically soft and ill-defined. In these circumstances, the wrist should be immobilised again for a further month, after which the plaster is removed and X-ray examination repeated.

This treatment should be continued until union occurs or until there is established non-union. If there is established non-union, the surfaces are radiologically sharply defined and possibly covered with a thin layer of bone; in these circumstances, it is hopeless to expect bony union to occur. Fibrous union is, however, probably present, and therefore a trial of wrist function should be given, for often, if work is light, the wrist will prove sufficiently strong. If the wrist is weak or painful, it is unlikely that conservative treatment will improve it. Arthrodesis of the wrist, with its disadvantages, remains the only choice.

*Avascular Necrosis.* Because of the oblique entry of vessels into the bone and the fact that the proximal pole is largely articular, fractures of the navicular may result in avascular necrosis of the proximal pole. This is a common cause of non-union. The changes producing the relative increase in density of the proximal pole (Fig. 642) have been outlined in Chap. 92. Degeneration of the avascular bone eventually results in carpal arthritis. Removal of the proximal pole before this has occurred is indicated, though the wrist will be left permanently weakened.

*Fracture of the Navicular Tuberosity.* A strain fracture of the tuberosity of the navicular may occur due to pull on the radial collateral ligament. Immobilisation in plaster for three weeks will relieve the pain. A

pressure over the head of the proximal phalanx (Fig. 645). This can be arranged by plaster of Paris or by a finger wire over the dorsum of the finger.

**Fractures of the Phalanges.** These are an integral part of hand injuries, and should be considered with them, though a few specific lesions are described here. Marginal fractures of the articular ends of the bones occur, due either to avulsion by force applied through the collateral ligaments, or to direct violence applied to the shoulders of the distal ends of the phalanges. Satisfactory union occurs if finger alignment is maintained in the flexed position on a finger wire, for a fortnight. Of special importance are dorsal and volar marginal fractures of the base of the terminal phalanx following blows on the tip of the finger, and sometimes accompanied by subluxation. The dorsal marginal fracture is one of the causes of the deformity known as *mallet finger*; the other is rupture of the extensor tendon. The two injuries can only be distinguished by radiography. Fixation of the finger with the terminal interphalangeal joint in hyperextension, and the proximal interphalangeal joint flexed sometimes reduces the fracture adequately, but often fails. Then either removal of the fragment and reattachment of the extensor tendon or suture of the fragment into place with a fine steel wire is necessary.

Splinting of the hand in the functional position is a fundamental principal of hand surgery if disabling contractures are to be avoided. The hand should be *slightly* cocked up, the fingers flexed midway, and separated, and the thumb flexed and lying a little in front of the fingers. This position can be maintained by a moulded metal splint or a plaster. The fingers should be held in position by packing fluffed-out gauze around and between them. The whole is made firm by bandaging with crepe bandages. This method of dressing is as important for soft tissue injuries as for fractures. The hand is elevated for a day or two afterwards on pillows, and is not inspected before the end of the first five days unless a complication such as infection appears, or if further surgery is anticipated.

**Injuries and Infections of the Hand** (see Chap. 106).

### FRACTURES OF THE FOREARM

If pronation and supination are to be fully preserved, reduction of fractures of the forearm must be perfect. Slight deformity may be tolerated in children in whom growth will usually correct the deformity. Satisfactory retention usually demands immobilisation of both the elbow and the wrist. As a plaster often has to be put on with the forearm under traction, with countertraction through a band passed around the arm, accurate fitting of the plaster may be difficult to obtain; therefore it may be easier to release the traction and include the arm in the plaster after the forearm plaster has set.

*Fractures of the forearm are grouped as follows: (1) Fractures of Single Bones.*—They are often due to direct violence. The splinting action of the



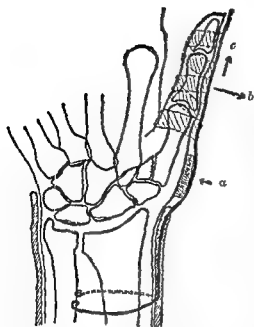


FIG. 644. The method of fixation of a Bennett's stave fracture, combining some traction with lateral pressure. (a) Sponge rubber pad between the wire and the base of the thumb. (b) Thumb and wire are bent out in this direction to obtain traction in the direction (c). (From "A Complete Outline of Fractures," by J. G. Bonnin, by courtesy of William Heinemann Ltd.)

produce shortening. Slight shortening, however, does not interfere with the function of the hand, and is better than restoration of the full length of the bone, at the price of a stiff finger. Occasionally fixation of a finger in flexion with a finger wire to restore full length may be advisable. A finger wire is incorporated in a Colles' type of plaster so that it lies under the affected finger. Strapping slings are passed across the wire, and the finger laid on them. Finger and wire are then overstrapped with small lengths of strapping. By bending finger and wire together considerable traction on the metacarpal can be produced. Traction slackens with time; therefore the finger is again bent to maintain it. Fracture of the neck of a metacarpal may be troublesome, as the head tends to bow into the palm, and then receives excessive pressure when gripping. The neck of the fifth metacarpal is most commonly deformed in this way. The fracture is reduced, and retention is obtained by full flexion of the finger and maintenance of

can be laid on it, and a small pad of sponge rubber or felt is then pushed between the wire and the base of the metacarpal lateral pressure is brought to bear on the metacarpal. If the thumb is now attached by strapping to the finger wire, and the thumb and wire are firmly abducted together, the thumb will be pulled on and also further pressure brought to bear on the metacarpal base. The fracture is thus reduced and retained. Check radiography is carried out to confirm reduction. Three weeks' immobilisation is necessary.

**Fractures of the Metacarpals of the Palm.** Fractures of the bases occur in crushing injuries and are usually well immobilised by contact with the neighbouring bones. Fractures of the shaft of the metacarpals, if transverse, can usually be manipulated into position, and will then remain in position if splinted in the functional position. Spiral fractures tend to

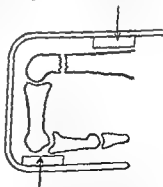


FIG. 645. The method of fixation of a flexion fracture of the metacarpal neck. Flexion of the proximal phalanx is used to hold the head of the metacarpal in place. Note the felt at the pressure points.

pressure over the head of the proximal phalanx (Fig. 645). This can be arranged by plaster of Paris or by a finger wire over the dorsum of the finger.

**Fractures of the Phalanges.** These are an integral part of hand injuries, and should be considered with them, though a few specific lesions are described here. Marginal fractures of the articular ends of the bones occur, due either to avulsion by force applied through the collateral ligaments, or to direct violence applied to the shoulders of the distal ends of the phalanges. Satisfactory union occurs if finger alignment is maintained in the flexed position on a finger wire, for a fortnight. Of special importance are dorsal and volar marginal fractures of the base of the terminal phalanx following blows on the tip of the finger, and sometimes accompanied by subluxation. The dorsal marginal fracture is one of the causes of the deformity known as *mallet finger*; the other is rupture of the extensor tendon. The two injuries can only be distinguished by radiography. Fixation of the finger with the terminal interphalangeal joint in hyperextension, and the proximal interphalangeal joint flexed sometimes reduces the fracture adequately, but often fails. Then either removal of the fragment and reattachment of the extensor tendon or suture of the fragment into place with a fine steel wire is necessary.

Splinting of the hand in the functional position is a fundamental principal of hand surgery if disabling contractures are to be avoided. The hand should be *slightly* cocked up, the fingers flexed midway, and separated, and the thumb flexed and lying a little in front of the fingers. This position can be maintained by a moulded metal splint or a plaster. The fingers should be held in position by packing fluffed-out gauze around and between them. The whole is made firm by bandaging with crepe bandages. This method of dressing is as important for soft tissue injuries as for fractures. The hand is elevated for a day or two afterwards on pillows, and is not inspected before the end of the first five days unless a complication such as infection appears, or if further surgery is anticipated.

**Injuries and Infections of the Hand** (see Chap. 106).

### FRACTURES OF THE FOREARM

If pronation and supination are to be fully preserved, reduction of fractures of the forearm must be perfect. Slight deformity may be tolerated in children in whom growth will usually correct the deformity. Satisfactory retention usually demands immobilisation of both the elbow and the wrist. As a plaster often has to be put on with the forearm under traction, with countertraction through a band passed around the arm, accurate fitting of the plaster may be difficult to obtain; therefore it may be easier to release the traction and include the arm in the plaster after the forearm plaster has set.

*Fractures of the forearm* are grouped as follows: (1) *Fractures of Single Bones*.—They are often due to direct violence. The splinting action of the

second bone usually ensures that displacement is small, and can be corrected by moulding the plaster. (2) *Fractures of Both Bones of the Forearm*: (a) Due to direct violence. The bones are broken at the same level. In severe injuries, a confluent hæmatoma may produce cross union, whereas in milder injuries a bridge of soft tissue usually separates the two sets of bone ends and a satisfactory position can be achieved by traction. Avoid tight plastering which may bow the bones together. Splint the bones in the mid-prone position. (b) Due to indirect violence. The bones are broken at different levels, and the two fractures may differ in shape. Displacement may be marked and manipulative reduction is often imperfect; open reduction is necessary if there is any remaining deformity,



FIG. 646. A Monteggia fracture-dislocation of the forearm. Note the anterior dislocation of the radial head.

and the procedure adopted must be varied to suit the particular fractures. Thus, an intramedullary nail may be passed down both bones (in the ulna from the olecranon, and in the radius from the styloid process); one bone only may be plated if the other fracture is stable. The second fracture may also be fixed with a plate or a single screw; for a screw to be safe, the fracture must be very oblique. Greenstick fractures of the forearm are common, but are easily straightened and fixed in plaster, for soft tissue support prevents displacement. (3) *Fractures of the Radius or Ulna Associated with Dislocation of the Upper or Lower Radio-ulnar Joint*.—This combination is important, as the dislocated joint may be overlooked clinically and also radiologically; and the joint lesion adds marked instability to the fracture, which must often be fixed by open operation. (a) Fracture of the shaft of the ulna associated with dislocation of the radio-humeral and upper radio-ulnar joint (*Monteggia fracture*—Fig. 646). The head of the radius may be dislocated, forwards, backwards or laterally. Reduction of forward dislocations is more stable in flexion, backward dislocations in slight extension. The fragments of the ulna are difficult to retain in position after manipulation and if imperfectly reduced

or if displacement occurs a few days after reduction they are best internally fixed. Fractures of the head of the radius may be associated with this injury, but never remove the head of the radius till the ulna is stable, for elbow stability is thereby lost. (b) Fracture of the lower end of the radius with dislocation of the lower radio-ulnar joint (Fig. 647). This injury is also difficult to stabilise and may require operative fixation. Alternatively a Kirschner wire may be passed either anteroposteriorly or



FIG. 647. Fracture of the lower third of the radius accompanied by anterior dislocation of the lower radio-ulnar joint.

laterally through the lower end of the radius and used to manipulate it ; the wire is then incorporated in the plaster. Attempting to stabilise the lower end of the radius by traction on the thumb is unsatisfactory and leads to stiffness of the thumb.

Union of the ulna is inclined to be slow ; in adults, a minimum of six weeks' fixation is required, but the time may have to be prolonged if the radiological appearances are unsatisfactory. However, it may then be sufficient to fix the forearm, and leave the elbow free for exercises. In children, although the fracture may feel firm, too early removal of the plaster may be followed by bowing, and as children do not suffer from longer fixation, the plaster should be left in place for a minimum of a month.

## CHAPTER 97

### FRACTURES AROUND THE ELBOW JOINT, FRACTURES OF THE HEAD AND SHAFT OF THE HUMERUS, THE CLAVICLE, SCAPULA, RIBS AND STERNUM

#### FRACTURES AROUND THE ELBOW JOINT

THESE are relatively common in children, and a sharp distinction must be drawn between the treatment in adults and in children. In children, epiphyseal separations as well as fractures may occur, and although open operation may be needed, excision of bone is totally contraindicated, for it interferes with growth. In children also, early reduction is necessary, as callus forms in the first week or ten days; therefore manipulation after this time is liable to failure. Swelling may, however, obscure the bony points, and render retention difficult. Fractures around the elbow may be complicated by dislocation of the elbow, which may have been spontaneously reduced, and therefore overlooked, or rupture of a collateral ligament (e.g. the medial collateral ligament in association with a fracture of the head of the radius). A hæmarthrosis accompanies most injuries of the elbow and may need aspiration to relieve pain and tension. Fractures of all three bones of the elbow joint may occur together, but are rare. Fractures of one or two bones are common. The radiograph may not show all the damage done, for fractures of the cartilaginous surface may not be visible (e.g. injury of cartilage of the capitellum in association with fracture of the head of the radius). Small displacements of an epiphysis may be difficult to determine, and the use of a comparative X-ray of the opposite elbow in the same degree of flexion as the injured one is valuable. Accurate comparison may be facilitated by drawing lines on the film.

**Fractures of the Head of the Radius.** These are due to direct compression or to forced abduction of the extended elbow. In abduction injuries, the medial collateral ligament may be torn. Possible associated injuries are: (a) Dislocation of the elbow; (b) dislocation of the head of the radius alone; (c) fracture of the ulna with dislocation of the head of the radius (Monteggia fracture); (d) fracture of the lateral condyle of the humerus; or (e) flake fractures of the capitellum.

The following are the various types of fracture of the head of the radius: (1) *Fissure Fracture*.—If undisplaced treat simply with a sling and early exercises. If the fragment is displaced, excise the head of the radius. (2) *Chip Fractures*.—If the chip is large, the head of the radius is excised, as the resultant callus prevents pronation and supination. If the chip is small it may be removed and the remainder of the head of the

radius left in place. (3) *Epiphyseal Fracture Displacements*.—If the angulation of the head is small, it can be corrected by rotation of the head of the radius under firm pressure from a padded board pressed across the neck of the bone. If the displacement is great, open operative reduction is needed, but the head of the radius must never be excised, though a few undergo avascular necrosis. (4) *Compression Fractures*.—If impaction is slight, and the head is intact and almost undisplaced, leave the head undisturbed. If there is comminution or displacement, the head of the radius should be excised.

If the head of the radius is to be excised, an early decision is necessary, as the results of late excision are much less satisfactory. In all cases of correctly treated fracture of the head of the radius, pronation and supination return completely before full extension of the elbow.

**Fractures of the Upper End of the Ulna.** These fractures may be produced by direct violence or by excessive pull of the triceps, but mixed varieties occur. Treatment is largely determined by the position and shape of the fracture, and is directed to restoration of the continuity of the triceps and its expansions, and avoidance of bony irregularity in the semilunar notch. The types of injury are : (1) Avulsion of the tricipital insertion and tip of olecranon. Excise the small fragment of bone and re-attach the triceps to the ulna, with stainless steel wire sutures. (2) Undisplaced fissure fractures of the notch. Treat simply in a sling for a week. (3) Oblique fractures. Fix in position, after removal of any loose fragments, with a screw passed towards the coronoid process. (4) Fractures in the anterior part of the notch, such as accompany forward dislocation of the radius and ulna, and render the elbow unstable. A long screw or intramedullary pin is used to fix the fragments. (5) Fractures of the coronoid process occur with posterior dislocation of the elbow. No special treatment is required, though an ossifying hæmatoma may occur in this area, and may require subsequent removal.

**Fractures of the Lower End of the Humerus.** These are divided into the complete and incomplete fractures. In the incomplete fractures, some part of the articular surface of the humerus remains fixed to the shaft. The incomplete fractures are : (1) *Fractures of the Epicondyles*.—These are avulsion fractures and the medial epicondyle is the more commonly injured ; rarely direct violence may be responsible. Coincident injury to the ulnar nerve may occur. Rest in a sling is all that is required. Union with slight displacement is consistent with sound function. (2) *Fractures of the Condyles*.—These may carry an epiphysis with them, and they sometimes rotate so that the fracture surface looks away from the humerus. If the fragment is undisplaced, fixation of the elbow in plaster at a right angle for a month is required. If there is displacement and manipulation is unsuccessful, open fixation may be needed. A single screw may be used in adults, but sutures are often better in children. (3) *Fractures of the Capitellum*.—Half the capitellum may be separated as a loose body.

## CHAPTER 97

### FRACTURES AROUND THE ELBOW JOINT, FRACTURES OF THE HEAD AND SHAFT OF THE HUMERUS, THE CLAVICLE, SCAPULA, RIBS AND STERNUM

#### FRACTURES AROUND THE ELBOW JOINT

THESE are relatively common in children, and a sharp distinction must be drawn between the treatment in adults and in children. In children, epiphyseal separations as well as fractures may occur, and although open operation may be needed, excision of bone is totally contraindicated, for it interferes with growth. In children also, early reduction is necessary, as callus forms in the first week or ten days; therefore manipulation after this time is liable to failure. Swelling may, however, obscure the bony points, and render retention difficult. Fractures around the elbow may be complicated by dislocation of the elbow, which may have been spontaneously reduced, and therefore overlooked, or rupture of a collateral ligament (e.g. the medial collateral ligament in association with a fracture of the head of the radius). A hæmarthrosis accompanies most injuries of the elbow and may need aspiration to relieve pain and tension. Fractures of all three bones of the elbow joint may occur together, but are rare. Fractures of one or two bones are common. The radiograph may not show all the damage done, for fractures of the cartilaginous surface may not be visible (e.g. injury of cartilage of the capitellum in association with fracture of the head of the radius). Small displacements of an epiphysis may be difficult to determine, and the use of a comparative X-ray of the opposite elbow in the same degree of flexion as the injured one is valuable. Accurate comparison may be facilitated by drawing lines on the film.

**Fractures of the Head of the Radius.** These are due to direct compression or to forced abduction of the extended elbow. In abduction injuries, the medial collateral ligament may be torn. Possible associated injuries are: (a) Dislocation of the elbow; (b) dislocation of the head of the radius alone; (c) fracture of the ulna with dislocation of the head of the radius (Monteggia fracture); (d) fracture of the lateral condyle of the humerus; or (e) flake fractures of the capitellum.

The following are the various types of fracture of the head of the radius: (1) *Fissure Fracture*.—If undisplaced treat simply with a sling and early exercises. If the fragment is displaced, excise the head of the radius. (2) *Chip Fractures*.—If the chip is large, the head of the radius is excised, as the resultant callus prevents pronation and supination. If the chip is small it may be removed and the remainder of the head of the

Open operation is technically difficult and is often followed by worse results than closed methods if injudiciously carried out.

### FRACTURES OF THE HEAD AND SHAFT OF THE HUMERUS

**Fractures of the Upper End of the Humerus.** The disability likely to follow these injuries is stiffness of the shoulder. Early exercises, which, in their simplest form, consist of pendulum swinging of the dependent arm at the shoulder should be instituted as soon as possible. For this reason, it is unwise to disimpact these fractures in the aged. They should be left undisturbed and physiotherapy begun at once. *The Tuberosities.*—Fractures may occur alone due to avulsion, or be associated with fracture-dislocations. If the dislocation is reduced, the detached tuberosity usually falls back into place. If the greater tuberosity is displaced, its position is usually improved by abduction. This position is maintained for three weeks, and then the arm is gradually brought to the side. If this fails to reduce the displacement, the best procedure in young patients may be screwing the fragment back. This method restores the continuity of the torn rotator cuff, and thus permits early exercises. Fractures of the lesser tuberosity are due to avulsion by the subscapularis, and so often accompany dislocation of the shoulder joint or ruptures of the short rotator cuff. When these fractures occur alone, a sling and early exercises are all that are required.

**Ruptures of the Short Rotator Cuff.** The cuff is composed of the conjoint tendons around the head of the humerus. It may be ruptured in part or as a whole, with or without dislocation of the shoulder (Fig. 648). Avulsion of bone from its humeral attachments may provide radiological evidence of injury of the cuff. If there has been an associated dislocation it immediately recurs if the cuff is completely ruptured, and the head of the humerus remains quite unstable. In partial ruptures (rupture of the supraspinatus), the patient is unable to initiate abduction, but full, if painful, passive abduction can be obtained, and the patient is often able to maintain abduction once it has been achieved passively. Repair of the cuff is essential in complete detachment. In incomplete rupture, repair is unwise in the aged, but is necessary in some



FIG. 648. Avulsion injury to the insertions of the short rotator cuff, accompanying dislocation of the shoulder.



There is no alternative but to remove this or smaller fragments. Damage to the cartilaginous surface may lead later to osteochondritis dissecans. (4) *Epiphyseal Injuries*.—The epiphysis for the medial epicondyle may be avulsed, and in association with a dislocation of the elbow, may be caught between the humerus and the semilunar notch. Unless its absence from its normal situation is noted, the displacement may be overlooked, with disastrous effects on the joint (Fig. 612). It should be removed from the joint by open operation. The ulnar nerve is almost inevitably injured in these cases. More difficult to determine are the lesions of the capitellar epiphysis, which may be pushed as a whole laterally, and displaced backwards or forwards. It can be manipulated into position by direct pressure with a padded board.

The complete fractures of the lower end of the humerus are divided into flexion and extension fractures according to whether the fracture occurs with the elbow flexed or extended. The most common injury is the supracondylar extension fracture of childhood. In this and other extension fractures, the line of fracture runs from behind downwards and forwards, and a sharp spicule of bone may project anteriorly. This can damage the vessels in the antecubital fossa, and thus be one of the factors causing Volkmann's contracture. The natural position for splinting is in flexion, but full flexion is dangerous, because of possible vascular obstruction. Reduction is carried out by drawing the forearm forward in relation to the lower end of the humerus; this corrects the posterior angulation of the fragment, but rotation and side-to-side angulation are not so easy to correct. Pronation and supination of the forearm may be used to correct medial and lateral angulation. Check radiography is essential, and a second reduction, when swelling has subsided, is often necessary. In children, considerable deformity will be ironed out by growth. In adults, retention for six weeks is often necessary; in children the fracture is firm in a month.

Flexion fractures are uncommon. In them the line of fracture slopes from in front downwards and backwards. The spicule of bone can only damage the triceps. The natural position to reduce this fracture is extension, but it must not be forced; the arm is best put up with 135 degrees of extension. The straighter the arm the easier it is to control lateral angulation of the fragment. There should be no prejudice against the extended position of the elbow; in epiphyseal lesions, by giving more accurate radiological control, it may be particularly useful. Flexion force may be responsible for T-shaped fractures into the joint. These are notoriously difficult to reduce, as tension in the collateral ligaments pull the fragments apart. The most successful closed reduction is provided by the use of skeletal traction through the olecranon, with the elbow held at right angles and the arm abducted. Some manipulation may also be required although continuous traction slowly reduces the displacement, and at the same time retains the fragments in position.

growth. Women dislike any deformity, but operative treatment leaves a scar which may draw more attention to the injury than a lump does. Children may suffer a greenstick fracture, indicated by an unwillingness to use the arm, and a tender point on the bone ; occasionally the appearance of a small mass of callus is the first indication of the injury, function soon returns, pain is slight, and retention (in any case being inadequate) is often unnecessary.

Complete fractures of the bone are oblique, transverse or comminuted, and whereas the centre of the shaft is the common site, fractures may occur at the junction of compact and cancellous bone at either end. Associated injury to underlying vessels and nerves is uncommon. Associated fractures of the ribs sometimes occur. The shoulder tends to drop leaving the inner end of the clavicle cocked up by the sternomastoid.

All methods of reduction and retention are directed to lifting and drawing the shoulder backwards, but must be adapted to the age and the needs of the patient and the site of the fracture. The simplest is the figure-of-eight sling around each shoulder, tightened across the back. The arm is supported in a sling. Almost daily adjustment is necessary if the effectiveness of the method is to be maintained. It is most suitable for children. In adults, firmer methods of fixation have the disadvantage of restricting shoulder movement, and thus often leading to stiffness in the elderly. The older method (Sayre's), strapping the arm to the side and pulling the elbow backwards, also provokes stiffness of the shoulder. Rest in bed, with a pillow between the shoulders so that the affected shoulder may drop back, does not always produce perfect reduction.

**Fractures of the Scapula.** These are classified into fractures of the body of the bone and those of the processes. Fractures of the body may be comminuted and accompanied by fractures of the ribs. The fragments are held in good position by the surrounding muscles. Therefore a sling is all that is required. The processes which may be fractured are the root of the spine of the scapula, the acromion, the coracoid process, and the neck of the glenoid, and the inferior angle of the glenoid may be avulsed by the triceps. Displacement is usually small. Therefore little is to be gained by firm external fixation or open reduction. Treatment is simply directed to relief of pain by rest in a sling, and early use of the shoulder when pain has diminished.

**Fractures of the Ribs.** The ribs may be broken by direct or indirect violence. In indirect violence, the compressing force may break the neck or the angle of the rib. A force producing a direct fracture may bend the ribs sufficiently to produce also an indirect fracture posteriorly. An osteoporotic rib may be fractured by the violence of coughing. Thus in chronic inflammatory diseases of the lung both old and new fractures of the ribs may be seen in X-rays. Blows insufficient to produce a fracture sometimes cause local pain due to detachment of intercostal muscles,

younger patients. Recovery of reasonable function without operation is common ; therefore two weeks' observation may be advisable before operation is considered but, after this time, repair becomes difficult due to contracture, and should not be further delayed. An approach is made through a sabre-shaped incision across the acromion, and the deltoid is detached. The rotator cuff is then sewn by wire sutures to a groove made in the anatomical neck of the humerus.

**Fractures of the Surgical Neck of the Humerus.** These fractures may be impacted, often with reasonable preservation of function, or unimpacted. Unless the deformity is marked, it is unwise to disimpact, especially in the aged. The arm is treated in a sling. In unimpacted fractures, the arm is placed in a sling ; padding is applied to the chest wall and a swathe of bandage wrapped over sling and arm. After a fortnight, a sling alone is substituted and gravity exercises are then begun. Very rarely, the displacement due to rotation of the head cannot be reduced, and internal fixation is required. The alternative, traction with the arm abducted, is sometimes effective in retaining the fragments. The fracture may be accompanied by a dislocation of the shoulder. Reduction of the dislocation is then difficult although it may sometimes be accomplished by manipulation. A Steinmann's pin inserted into the head of the humerus to control it is often successful in aiding reduction. Otherwise, open reduction is required, and may be combined with internal fixation of the fracture.

**Fractures of the Shaft of the Humerus.** Fractures at the junction of the middle and upper thirds of the shaft of the humerus may be complicated by radial nerve injury ; signs of nerve injury should be sought prior to manipulation, for otherwise the surgeon may be erroneously blamed for the lesion. Transverse fractures in particular are unstable, and firm external fixation is difficult. The lower the fracture, the easier it is to control. Immobilisation is best obtained by a plaster cast. The elbow is fixed at a little more than a right angle and the wrist is supported by a collar and clove hitch. The weight of the plaster produces a little traction (hanging plaster cast). When this method is unsuccessful, traction is applied through a Kirschner wire in the olecranon. Traction is maintained on the abducted arm for two or three weeks till the fracture is "sticky." A third method is to apply a light plaster support to the arm and bandage it against the side, with the forearm in a sling. Non-union of fractures in the upper third of the humerus is not uncommon in young men due to interposition of soft tissues, or stripping of the bone ends at the time of injury.

**Fractures of the Clavicle.** The clavicle owes its susceptibility to injury to being the only bony bridge between the arm and the trunk ; and for this reason also its fixation after injury is difficult. Fortunately, however, union with deformity is consistent with good function, and, furthermore, in children, in whom the injury is frequent, deformity is corrected by

growth. Women dislike any deformity, but operative treatment leaves a scar which may draw more attention to the injury than a lump does. Children may suffer a greenstick fracture, indicated by an unwillingness to use the arm, and a tender point on the bone; occasionally the appearance of a small mass of callus is the first indication of the injury, function soon returns, pain is slight, and retention (in any case being inadequate) is often unnecessary.

Complete fractures of the bone are oblique, transverse or comminuted, and whereas the centre of the shaft is the common site, fractures may occur at the junction of compact and cancellous bone at either end. Associated injury to underlying vessels and nerves is uncommon. Associated fractures of the ribs sometimes occur. The shoulder tends to drop leaving the inner end of the clavicle cocked up by the sternomastoid.

All methods of reduction and retention are directed to lifting and drawing the shoulder backwards, but must be adapted to the age and the needs of the patient and the site of the fracture. The simplest is the figure-of-eight sling around each shoulder, tightened across the back. The arm is supported in a sling. Almost daily adjustment is necessary if the effectiveness of the method is to be maintained. It is most suitable for children. In adults, firmer methods of fixation have the disadvantage of restricting shoulder movement, and thus often leading to stiffness in the elderly. The older method (Sayre's), strapping the arm to the side and pulling the elbow backwards, also provokes stiffness of the shoulder. Rest in bed, with a pillow between the shoulders so that the affected shoulder may drop back, does not always produce perfect reduction.

**Fractures of the Scapula.** These are classified into fractures of the body of the bone and those of the processes. Fractures of the body may be comminuted and accompanied by fractures of the ribs. The fragments are held in good position by the surrounding muscles. Therefore a sling is all that is required. The processes which may be fractured are the root of the spine of the scapula, the acromion, the coracoid process, and the neck of the glenoid, and the inferior angle of the glenoid may be avulsed by the triceps. Displacement is usually small. Therefore little is to be gained by firm external fixation or open reduction. Treatment is simply directed to relief of pain by rest in a sling, and early use of the shoulder when pain has diminished.

**Fractures of the Ribs.** The ribs may be broken by direct or indirect violence. In indirect violence, the compressing force may break the neck or the angle of the rib. A force producing a direct fracture may bend the ribs sufficiently to produce also an indirect fracture posteriorly. An osteoporotic rib may be fractured by the violence of coughing. Thus in chronic inflammatory diseases of the lung both old and new fractures of the ribs may be seen in X-rays. Blows insufficient to produce a fracture sometimes cause local pain due to detachment of intercostal muscles,

and thus lead to suspicion of a fracture. In direct violence, the ribs are pushed in and tend to lacerate the lung. Separation of the junction of the rib from its costal cartilage may occur.

*Treatment* is chiefly concerned with the relief of pain and the prevention or management of complications. Pain is most quickly and effectively relieved by infiltration of the fracture with local anæsthetic. A long-acting local anæsthetic may be used. This allows the patient to move and cough, and thus tends to prevent complications due to pulmonary stasis. An attempt is often made to rest the ribs with strapping. This method must, however, be used judiciously if at all; rib movements must not be restricted in the elderly. It is almost impossible to fix the upper five ribs but the lower ribs can be effectively supported; this is best done with broad elastic adhesive strapping passed completely around the chest at the level of the fractured ribs, and made firm but not too tight. Strapping half the circumference of chest only fixes the skin.

*Complications.* (1) *Hæmoptysis*, due to laceration of the lung, is often small and of no importance. Absence of hæmoptysis does not rule out a serious lung injury. (2) *Surgical Emphysema*.—This is usually not serious in itself even if extensive. (3) *Hæmothorax*.—Small amounts are common and can be neglected. Larger amounts need aspiration under radiological control. Rarely, continued hæmorrhage requires surgical arrest. (4) *Pneumothorax*.—This may be open through the chest wall or closed. In the open variety the lung always collapses. The wound must be immediately sealed with a wet dressing pending suture of the wound, in order to prevent mediastinal movement. Closed pneumothorax is not important unless it is of the "valvular" type which allows a continuous build up of pressure in the pleural cavity and ultimately causes displacement of the mediastinum to the opposite side. The thorax should be aspirated and the needle attached to a water-sealed tube. (5) *Traumatic Cyanosis*.—Following crush injuries of the chest, there may be subconjunctival hæmorrhages, and petechial hæmorrhages over the face and upper part of the thorax, due to capillary rupture. There is no specific treatment for this condition which may not, however, be as serious as it appears. Treatment is directed to any associated complications. (6) *Collapse of the lung*, with laceration of the liver or spleen, may be associated with lower rib injuries.

*Fractures of the Sternum.* These are classified into those due to direct violence, and those due to hyperflexion of the thoracic spine. In direct injuries, sternal alignment is restored by the ribs; local bruising and tenderness, and pain on thoracic compression are found. In indirect injuries the sternal deformity persists due to the spinal angulation. Therefore the discovery of such a deformity should at once direct attention to the spine. Often no treatment is possible, though manipulation of the spine may be attempted bearing in mind the difficulties encountered in this procedure in the thoracic region.

## CHAPTER 98

### FRACTURES OF THE SPINE

FRACTURES of the spine are usually due to a combination of compression with forward flexion of the spine. Only rarely, and then only in an osteo-arthritic cervical spine, is there likely to be injury from hyperextension. Inclination of the body to one or other side may produce a fracture by lateral flexion; in the cervical spine this may be accompanied by dislocation. Injuries throughout the spine follow a uniform basic pattern, but differ according to the level affected and the ease of dislocation of the posterior diarthrodial joints. Thus in the thoracic spine the support of the ribs prevents dislocation, and also makes reduction of a compression fracture of a vertebral body difficult. In the cervical spine, the shallow oblique facets of the diarthrodial joints make dislocation with or without fracture more likely, whereas in the lumbar spine the depth of these articular facets makes dislocation impossible without severe accompanying injuries, e.g. fracture of the articular facets and rupture of the posterior interspinous ligaments. The complication to be feared most is injury to the spinal cord. The displacement necessary to produce such injury is due to rupture of the posterior supporting tissues. Spinal injuries may be divided into three groups: A. *Those with the Posterior Tissues Intact.*—(1) Compression fractures of the vertebral bodies. (2) Acute posterior rupture of an intervertebral disc—surprisingly rare in severe spinal injuries. B. *Those with Rupture of the Interspinous Ligament and other Tissues.*—(1) Severe and multiple compression fractures of the bodies. (2) Fracture dislocations. (3) Dislocations. C. *Fractures of the Vertebral Processes not Affecting the Continuity of the Spine.*—(1) Fractures of laminae and pedicles. (2) Fractures of the transverse processes. (3) Fractures of the spinous processes.

**First Aid and Transport.** Patients with a severe spinal injury are usually rendered helpless, and the temptation to lift them or to encourage them to stand is understandable. They should, however, be kept lying where they are until experienced assistance is available. A careful examination should be made, particularly to see that the airway is clear. Three or four assistants are necessary to move the patient; the most experienced one guards the head and maintains gentle traction. *All flexion or extension must be avoided.* Therefore the patient is lifted *en masse* or rolled on to a firm stretcher. The supine position should be maintained in cervical injuries, and a support should be placed on either side of the face. Injuries to the thoracic or lumbar spine may be transported prone or supine, but normal lordosis of the lumbar spine should be maintained with a folded blanket.

and thus lead to suspicion of a fracture. In direct violence, the ribs are pushed in and tend to lacerate the lung. Separation of the junction of the rib from its costal cartilage may occur.

*Treatment* is chiefly concerned with the relief of pain and the prevention or management of complications. Pain is most quickly and effectively relieved by infiltration of the fracture with local anæsthetic. A long-acting local anæsthetic may be used. This allows the patient to move and cough, and thus tends to prevent complications due to pulmonary stasis. An attempt is often made to rest the ribs with strapping. This method must, however, be used judiciously if at all; rib movements must not be restricted in the elderly. It is almost impossible to fix the upper five ribs but the lower ribs can be effectively supported; this is best done with broad elastic adhesive strapping passed completely around the chest at the level of the fractured ribs, and made firm but not too tight. Strapping half the circumference of chest only fixes the skin.

*Complications.* (1) *Hæmoptysis*, due to laceration of the lung, is often small and of no importance. Absence of hæmoptysis does not rule out a serious lung injury. (2) *Surgical Emphysema*.—This is usually not serious in itself even if extensive. (3) *Hæmothorax*.—Small amounts are common and can be neglected. Larger amounts need aspiration under radiological control. Rarely, continued hæmorrhage requires surgical arrest. (4) *Pneumothorax*.—This may be open through the chest wall or closed. In the open variety the lung always collapses. The wound must be immediately sealed with a wet dressing pending suture of the wound, in order to prevent mediastinal movement. Closed pneumothorax is not important unless it is of the "valvular" type which allows a continuous build up of pressure in the pleural cavity and ultimately causes displacement of the mediastinum to the opposite side. The thorax should be aspirated and the needle attached to a water-sealed tube. (5) *Traumatic Cyanosis*.—Following crush injuries of the chest, there may be subconjunctival hæmorrhages, and petechial hæmorrhages over the face and upper part of the thorax, due to capillary rupture. There is no specific treatment for this condition which may not, however, be as serious as it appears. Treatment is directed to any associated complications. (6) *Collapse of the lung*, with laceration of the *liver* or *spleen*, may be associated with lower rib injuries.

*Fractures of the Sternum.* These are classified into those due to direct violence, and those due to hyperflexion of the thoracic spine. In direct injuries, sternal alignment is restored by the ribs; local bruising and tenderness, and pain on thoracic compression are found. In indirect injuries the sternal deformity persists due to the spinal angulation. Therefore the discovery of such a deformity should at once direct attention to the spine. Often no treatment is possible, though manipulation of the spine may be attempted bearing in mind the difficulties encountered in this procedure in the thoracic region.

## CHAPTER 98

### FRACTURES OF THE SPINE

FRACTURES of the spine are usually due to a combination of compression with forward flexion of the spine. Only rarely, and then only in an osteo-arthritic cervical spine, is there likely to be injury from hyper-extension. Inclination of the body to one or other side may produce a fracture by lateral flexion; in the cervical spine this may be accompanied by dislocation. Injuries throughout the spine follow a uniform basic pattern, but differ according to the level affected and the ease of dislocation of the posterior diarthrodial joints. Thus in the thoracic spine the support of the ribs prevents dislocation, and also makes reduction of a compression fracture of a vertebral body difficult. In the cervical spine, the shallow oblique facets of the diarthrodial joints make dislocation with or without fracture more likely, whereas in the lumbar spine the depth of these articular facets makes dislocation impossible without severe accompanying injuries, e.g. fracture of the articular facets and rupture of the posterior interspinous ligaments. The complication to be feared most is injury to the spinal cord. The displacement necessary to produce such injury is due to rupture of the posterior supporting tissues. Spinal injuries may be divided into three groups: A. *Those with the Posterior Tissues Intact.*—(1) Compression fractures of the vertebral bodies. (2) Acute posterior rupture of an intervertebral disc—surprisingly rare in severe spinal injuries. B. *Those with Rupture of the Interspinous Ligament and other Tissues.*—(1) Severe and multiple compression fractures of the bodies. (2) Fracture dislocations. (3) Dislocations. C. *Fractures of the Vertebral Processes not Affecting the Continuity of the Spine.*—(1) Fractures of laminae and pedicles. (2) Fractures of the transverse processes. (3) Fractures of the spinous processes.

**First Aid and Transport.** Patients with a severe spinal injury are usually rendered helpless, and the temptation to lift them or to encourage them to stand is understandable. They should, however, be kept lying where they are until experienced assistance is available. A careful examination should be made, particularly to see that the airway is clear. Three or four assistants are necessary to move the patient; the most experienced one guards the head and maintains gentle traction. *All flexion or extension must be avoided.* Therefore the patient is lifted *en masse* or rolled on to a firm stretcher. The supine position should be maintained in cervical injuries, and a support should be placed on either side of the face. Injuries to the thoracic or lumbar spine may be transported prone or supine, but normal lordosis of the lumbar spine should be maintained with a folded blanket.



**Fractures of the Cervical Spine.** The shape and position of the first two cervical vertebræ render their fractures unique and important. The attachment of the posterior arches is weak in both vertebræ, and there is the special liability of the dens (odontoid process) to be fractured, or the transverse ligament to be ruptured.

Clinically, injuries of the cervical spine may be divided into serious and simple. In a simple injury, possibly accompanied by an undisplaced fracture, the patient complains of a stiff neck and restriction of movement, but the head is well supported by the cervical muscles; some voluntary movement of the neck is possible and muscle spasm can be relaxed by supporting the head. In a serious injury, the patient usually supports the head with the hands; the deformity is fixed by intense muscle spasm, and there may be deviation of the head to the side; the patient is unable to relax, and is terrified of any withdrawal of support. To these features may be added evidence of nerve root or spinal cord compression (see under *The Spinal Cord and its Nerve Roots*, Chap. 59). Accurate diagnosis is dependent on good radiography, and this means moving the patient. This should not be done till the patient has recovered from shock, and a good airway has been assured.

**Treatment.** Simple injuries (minor vertebral compressions, minor injuries to osteo-arthritic spines, fractures of bodies or their appendages without displacement) only require rest and support followed by gradually increasing freedom of movement. Relief may be given merely by rest in bed, or with the addition of sandbags on both sides of the head. The simple Schanz collar (a triangle of gamgee tissue, 1 inch wide at the tip, 11 inches wide at the base, and 2 yards long, wound gently around the neck, starting with the tip of the bandage, and held in place by a calico bandage) may be very effective. The inflatable rubber collar may be used instead. A plaster collar may be made, though it is difficult to fit comfortably and efficiently.

The more serious injuries, such as those accompanied by dislocations, require traction to steady them, and, if there is a dislocation, very

carefully controlled reduction is necessary. Traction may be applied by means of a simple appliance gripping forehead and occiput, e.g. the Crile head tractor, but in all serious injuries skeletal traction from the outer tables of the skull is best. Controlled traction usually enables sufficient separation of injured vertebræ to permit reduction of a dislocation. Less traction may

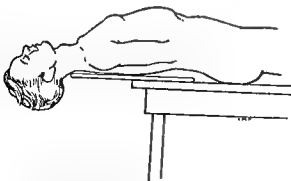


FIG. 649. The use of a narrow board fixed to a table to support the cervical spine in applying a cervical plaster.

then be used for retention, or the neck may be supported by a plaster cast. In applying plaster of Paris to the cervical region great help is given by the use of a supporting rod projecting over the end of the table, along which the upper part of the thorax and the neck can be laid (Fig. 649). The head is steadied in the hands of a seated assistant.

**Fractures of the Thoracic Spine.** Apart from injuries by missiles, fractures of this region tend to be of a minor nature because of the supporting action of the ribs. The common injury is a slight wedging of one or two vertebrae. The deformity is slight, and it is often overlooked; it may be discovered after a few days when persistent pain compels further investigation. Reduction of such wedging is difficult, and retention almost impossible. Interference with subsequent function is usually undetectable, so that anatomical restitution is unjustified. Rest, followed by progressive activity graded in accordance with the discomfort produced, is the correct treatment. Fractures of the last three thoracic vertebrae are discussed with fractures of the lumbar spine which they resemble more closely.

**Fractures of the Lumbar Spine.** The lumbar vertebrae are unsupported by ribs, and so are virtually the last two thoracic vertebrae. Fractures are most common in the region of the thoracolumbar junction, T10-L1 (70 per cent.). The most common injury is a compression fracture of the vertebral body with slight to moderate wedging. Greater displacements are easily observed on clinical examination; a slight kyphosis is noted on running the fingers over the line of the spinous processes. The history of a flexion injury, and the persistent pain with local tenderness, are sufficient to determine the need for radiography, on which diagnosis depends. Treatment, which formerly consisted in an effort to reduce and retain the fracture, has had to be modified, firstly, because experience has shown that considerable deformity is compatible with hard manual labour, and, secondly, because the plaster jacket used for retention produced excessive stiffness, in spite of heroic exercises carried out during the period of spinal fixation. Treatment of a young girl may differ from that of a working man. In the latter, the goal is return of function, which is best achieved by progressive spinal exercises, at first in bed and then when up. An angulation of up to 25 degrees may be treated in this manner. When, as in a young girl, postural perfection is desired, or when there is considerable deformity or accompanying instability of the spine due to rupture of the interspinous ligaments or fracture of articular processes, reduction followed by immobilisation in extension in a plaster jacket is necessary.

**Reduction.** This may be accomplished gradually using the patient's weight to extend the spine as on a hinged Bradford frame. This is a useful preliminary method of treatment when observation of associated injuries would be obstructed by a plaster jacket. However, the reduction is often incomplete and needs to be completed later by hyperextension. This is

painful and needs a general anaesthetic. Hyperextension may be carried out in various ways. Thus the patient with pelvis and legs supported is arched back over a flexible operating table, or may be laid prone in a long towelling sling like a hammock, and the legs and head gradually raised. After this manipulation, a further period on a frame or hinged bed is advisable, as it is difficult to apply a plaster jacket to an anaesthetised patient, but this difficulty can be best overcome by using the towelling



FIG. 650. The supine sling method. (From "A Complete Outline of Fractures," by J. G. Bonnin, by courtesy of William Heinemann Ltd.)

sling, which is incorporated in the plaster. There is always a danger of producing too much extension, and consequent discomfort, in patients relaxed by general anaesthesia (*vide infra*). After a few days' rest the patient will be found to maintain the reduced posture himself more easily, and can then be easily placed in the correct position in a plaster jacket without anaesthesia. Three methods of applying the plaster are described: (1) *The Hammock Method*.—The patient lying face down in a towelling hammock, has the plaster applied over the towelling, which is finally cut away in trimming the plaster. (2) *The Two-table Method*.—The patient lies sagging between two tables, one supporting the legs as far as the pubes, and the other the forearms and chin. This method requires considerable effort on the part of the patient, and is only suitable

for light and active people. No plaster jacket is suitable for the excessively obese. (3) *The Supine Sling*.—A broad webbing band is passed behind the lumbar spine and held tense by a suitable large stirrup (Fig. 650). The sling is raised by means of a block and tackle, and the patient is allowed to sag across it till the desirable degree of extension is reached. The head and legs are then supported on pillows, and the plaster jacket is applied on the suspended body. Subsequently the webbing sling is pulled out or incorporated in the plaster.

Whatever method is adopted, care must be taken of three pressure areas, the spinous processes of the lumbar spine, the pubes and the manubrium sterni. These areas must be padded with orthopædic felt or other suitable material. Often a narrow window over the padded spinous processes is also necessary to relieve local pressure. The plaster jacket must be judiciously constructed of slabs and circular bandages, both to obtain strength, and yet avoid excessive weight. It must also be applied with some regard for time, so that it sets *en masse* and not in layers; this calls for team work. The plaster must extend from the manubrium sterni to the pubes, and it must be trimmed so that the patient can flex the hips to a right angle, and so that it does not ride over the sternum and compress the neck. Adequate space must be left for the play of the pectoral muscles. After-treatment consists of graduated exercise, at first with the patient recumbent and then standing. The plaster must be retained for at least three months, otherwise slow recurrence of part of the original deformity will occur.

**Complications of Fracture of the Lumbar Spine.** (1) *Interlocking of Facets*.—In dislocations or fracture-dislocations, the lumbar facets may interlock and prevent reduction. Open operation is needed for reduction, which is often best accomplished by nibbling away one of the involved facets to avoid further manipulation. Often there is a spinal cord lesion, and it is therefore usually advisable to fix the spinous processes by metal plates bolted on each side of them, so that patients can be nursed without reproducing the displacement and thereby perhaps adding to any neurological damage. (2) *Retropulsion of a Disc*.—This may be central with spinal cord, or cauda equina damage, or lateral with predominantly nerve root damage. Acute disc prolapse is best treated by immediate surgery (Chap. 59). (3) *Paralytic Ileus*.—This may be produced by spinal hyperextension, which is therefore to be avoided in the early stages, but it may also occur before treatment and is thought to be attributable to a retroperitoneal hæmatoma. Its chief features are abdominal distension and vomiting, and its importance is due to its possible confusion with abdominal injury with which indeed it is commonly associated. It is treated in the same way as when due to other causes, but if a plaster jacket has been applied it may have to be removed. (4) *Injury to the Spinal Cord* (see under *The Spinal Cord and its Nerve Roots*, Chap. 59).

## CHAPTER 99

### FRACTURES OF THE PELVIS AND SACRUM

THE pelvis is best regarded as a ring of uneven strength in which there are three weak zones: (a) *Anterior*.—The pubic symphysis and the ischiopubic rami. (b) *Lateral*.—The floor of the acetabulum. (c) *Posterior*.—The sacro-iliac joint. Arising from the irregular-shaped ring, there are a number of processes jutting out for the attachment of muscles and ligaments; these may be broken by direct violence or avulsion. There are two groups of fractures, those of the pelvic processes and those of the pelvic ring. Fractures of both groups may, however, be found in combination.

**Fractures of the Pelvic Processes.** (1) *The Anterior Inferior Iliac Spine*.—This may be avulsed by the pull of the rectus femoris. The injury is most commonly met with in adolescents, often after kicking a football. Bruising appears from beneath the attachment of the sartorius and tensor fasciæ latæ, often outlining these muscles. The pain is aggravated by contraction of the quadriceps, e.g. by straight leg lifting. The displaced fragment is visible on radiography, and although separated for  $\frac{1}{2}$  inch or more, fibrous union follows with little interference with function. (2) *The anterior superior iliac spine* is fractured by direct violence, but is usually maintained in good position by the surrounding muscles. (3) *The ischial tuberosity*, or more commonly its secondary centre of ossification, may be avulsed by the pull of the hamstrings in young sprinters or hurdlers. There is local bruising, pain and inability to raise the leg when lying prone. Fibrous union occurs, but has little effect on function. (4) *The Ala of the Ilium*.—This may be fractured by direct violence, and displacement is prevented by the attached muscles. An extensive hæmatoma may form, and may be worth evacuation by incision. (5) *The Posterior Lip of the Acetabulum*.—This may be fractured in association with posterior dislocation of the hip. The treatment of all these fractures consists of initial bed rest and a graduated return to activity as pain subsides. Although there may still be some discomfort, functional recovery is usually complete by the end of the third week.

**Fractures of the Pelvic Ring.** These may be due to violent blows on the iliac crest, to crushing and run-over injuries, and may be associated with direct damage to the soft parts. More serious are the injuries in which the hemipelvis is twisted off the sacrum by forced hyperextension or abduction of the leg. There is liable to be considerable displacement. Falls on the trochanter may drive the femoral head through the acetabular floor (Fig. 651), and may at the same time fracture the centre of the hemi

pelvis and drive the two fragments inwards. Usually the pelvis is fractured by distortion (cf. the skull). It is impossible to have distortion in one part of a ring without distortion in another. In many cases of so-called single fracture of the pelvis, the secondary distortion has occurred at the pubic symphysis or the sacro-iliac joint which has subsequently sprung back into position. These joints must therefore be carefully examined in all such cases. The only true single fracture of the pelvis is the undisplaced fissure fracture through the brittle ala of the ilium due to a direct blow.

In the posterior part of the pelvic ring a fracture may run through the sacrum on one side of the sacro-iliac joint, or through the ilium on the other side, or it may take an irregular path through part of the joint and part of either bone. A similar range of possibilities exists with the pubic symphysis. Fractures of the pelvic ring may be double, triple or quadruple; therefore a large variety of injuries may be encountered. A few representative examples are outlined below. *Double Fractures.*—(1) The ischiopubic rami on either side of the symphysis.



FIG. 631. A fracture of the acetabular floor. The acetabular roof is not broken and the result was therefore moderately good.

(2) Anterior fracture and sacro-iliac dislocation or fracture-dislocation. (3) Fracture through the acetabular floor with displacement of a quarter of the pelvic ring. *Triple Fractures.*—(1) Double fracture anteriorly with a single sacro-iliac injury posteriorly. (2) Fracture of the acetabular floor with displacement of the corresponding two quarters of the pelvis inwards. (3) Diastasis of the symphysis pubis, and double sacro-iliac dislocation. *Quadruple Fractures.*—A fracture through the ischiopubic rami anteriorly and a double sacro-iliac injury posteriorly.

*Treatment of Fractures without Displacement.* The treatment is solely directed to the nursing comfort of the patient. Many patients merely require rest in bed with a gradually increasing range of exercise for the legs and spine as pain diminishes. The length of time spent in bed is determined by the pain and the recovery of muscular control. In the young with a minor fracture it may be merely a week, whereas in the older patient with a more severe injury, sitting out of bed may begin in

the third week and walking in the fourth. Regular turning of the patient for care of the skin is necessary. A divided mattress makes attention to the bowels easier. Some patients get comfort from a firm pelvic binder. Very heavy patients may be helped by a counterbalanced pelvic sling which facilitates raising the buttocks and turning.

*Treatment of Fractures with Displacement.* The same three stages of treatment apply as for fractures of long bones, namely, reduction, retention and rehabilitation. The method of reduction and retention employed depends on the type of deformity. The understanding of the displacements is facilitated if the sacro-iliac joint is regarded as a hinge.



FIG. 652. Diastasis of the symphysis pubis, with sacroiliac displacement on the right (reader's left). This is easily closed by the use of a pelvic sling.

The hinge may permit excessive opening or closing ; it may also permit rotary displacement of the hemipelvis, or it may be torn out as though by the screws and permit upward displacement of the hemipelvis which is maintained in its abnormal position by the pull of the pelvivertebral muscles. (1) *Outward Hinging.*—This is classically shown in diastasis of the pubis symphysis (Fig. 652). The ligaments of the sacro-iliac joint remaining partly intact still function as a hinge, and the symphysis may be closed by a sling passed around the pelvis and attached to crossed suspension cords. (2) *Inward Hinging.*—For this to occur there must be overlap of the two pubic bones. The use of a sling would increase the deformity, and reduction when there is displacement of the quarter pelvis must be accomplished by traction on the legs in abduction. When there is an alteration in level of the two sides of the symphysis, this must be adjusted by altering the level of the legs. Inward hinging by reducing the diameter of the true pelvis may be important in women, as it may later interfere

with labour. (3) *Rotational Displacement*.—This may be combined with inward hinging, and is corrected by adjustment of the relative leg levels (i.e. flexion of one leg and extension of the other). (4) *Combined displacements* with elevation of the hemipelvis. In these cases, the posterior hinge is destroyed and is unstable. In a small number of cases, the interlocking of the posterior injury after reduction is sufficient for retention to be maintained by a pelvic sling and crossed bands, but it may be supplemented by skin traction on the appropriate leg.

In most cases the course of treatment runs as follows (complications are discussed later): After recovery from shock, a Steinmann's pin or Kirschner's wire is passed through the lower end of the femur. This site is used in preference to the tibial tubercle, for the force employed might otherwise damage the knee. The leg is then slung with the knee flexed on a Thomas's or Braun's splint, and 20 to 40 lb. traction applied. It may be necessary to splint the opposite leg in a similar way, though on this side the tibial tuberosity may be used for the traction required to balance the pelvis. The foot of the bed is raised for countertraction. If gradual reduction of the deformity occurs, the traction may be reduced, and may be dispensed with between the third and fourth weeks. If no reduction occurs, the pelvis is manipulated during skeletal traction on a Hawley table. Check radiographs are taken on the table. A firm sling is applied around the pelvis, and the traction reduced or omitted. If a further check X-ray shows that reduction has been maintained, a double hip spica of plaster of Paris may be applied. This is, however, unsuitable for fat patients, and often in slim ones permits redisplacement of the fracture or rotation of the pelvis in the plaster as swelling subsides. In such cases, therefore, return to bed and the use of continuous traction is safer. It is usually impossible to affect the position of the pelvis after the third week. All pelvic fractures are firm in six weeks, and therefore after this period patients should be left free in the bed and made to carry out exercises. In most cases the retention may be reduced to a minimum at the end of the third week. Active exercises should be carried out from the beginning within the limits imposed by the injury.

**Complications.** (a) *Hæmatoma. Superficial*.—May require incision and expression of the contents followed by a drain for forty-eight hours. *Subperitoneal*.—This may be associated with extraperitoneal rupture of the bladder. Alone, it may be responsible for the development of paralytic ileus. (b) *Rupture of the Urethra*.—This is shown by the triad of perineal bruising, escape of blood from the urethra, and retention of urine. (c) *Rupture of the Bladder*.—This may be intraperitoneal or extraperitoneal. The distinction may require careful examination in the theatre. (d) *Injury to the Rectum*.—An iliac colostomy is indicated. (e) *Injury to the Bowel*.—Laparotomy and closure of small bowel wounds; a colostomy is made of an opening in the large bowel. (f) *Injury to the Sacral Plexus*.—See under fractures of the sacrum. Although lower



the third week and walking in the fourth. Regular turning of the patient for care of the skin is necessary. A divided mattress makes attention to the bowels easier. Some patients get comfort from a firm pelvic binder. Very heavy patients may be helped by a counterbalanced pelvic sling which facilitates raising the buttocks and turning.

*Treatment of Fractures with Displacement.* The same three stages of treatment apply as for fractures of long bones, namely, reduction, retention and rehabilitation. The method of reduction and retention employed depends on the type of deformity. The understanding of the displacements is facilitated if the sacro-iliac joint is regarded as a hinge.



FIG. 652. Diastasis of the symphysis pubis, with sacroiliac displacement on the right (reader's left). This is easily closed by the use of a pelvic sling.

The hinge may permit excessive opening or closing ; it may also permit rotary displacement of the hemipelvis, or it may be torn out as though by the screws and permit upward displacement of the hemipelvis which is maintained in its abnormal position by the pull of the pelvivertebral muscles. (1) *Outward Hinging*.—This is classically shown in diastasis of the pubis symphysis (Fig. 652). The ligaments of the sacro-iliac joint remaining partly intact still function as a hinge, and the symphysis may be closed by a sling passed around the pelvis and attached to crossed suspension cords. (2) *Inward Hinging*.—For this to occur there must be overlap of the two pubic bones. The use of a sling would increase the deformity, and reduction when there is displacement of the quarter pelvis must be accomplished by traction on the legs in abduction. When there is an alteration in level of the two sides of the symphysis, this must be adjusted by altering the level of the legs. Inward hinging by reducing the diameter of the true pelvis may be important in women, as it may later interfere

## CHAPTER 100

### FRACTURES OF THE UPPER END OF THE FEMUR

FRACTURES of the upper end of the femur are the bane of the elderly in whom senile osteoporosis is the predisposing factor. They result from tripping, or falls on the great trochanter. The difficulties they present in treatment are due to the complications likely to occur during bed rest in the elderly. Although anatomical reposition is aimed at, the prime purpose of treatment is to permit early mobilisation of the patient. For this reason, open fixation is the best treatment; and if the patient's general condition is too bad for operation, hope of survival is slight.

The risks of bronchopneumonia have been greatly reduced by the use of antibiotics, which are administered routinely to such patients both before and after operation. Deep venous thrombosis and embolism are somewhat rare, especially if the patients are treated actively. More frequent and important is general deterioration indicated by mental changes, noisiness and disorientation at nights, apathy, and incontinence of bowel and bladder. This leads to the development of bed sores, especially over the sacrum, and they may become extensive and cause toxæmia. In spite of efforts to encourage activity, appetite and interest in the patient, a slow and unhalting decline may set in.

Such considerations do not influence the treatment of younger patients. In the young, however, it takes far greater force to fracture the femur, and there is consequently a greater risk of damage to soft tissues, and so to the blood supply to the head of the femur, with a consequent greater incidence of avascular necrosis. The child and adolescent tolerate fixation in plaster well; therefore for them immobilisation in a plaster spica is often the best treatment. In the young adult, operation is recommended, for it diminishes the risk of non-union in fractures of the femoral neck; produces a better anatomical reposition; and permits early exercise and weight-bearing.

**Classification.** (1) *Fracture of the Femoral Neck.*—The fracture may be close to the femoral head (subcapital) or nearer the intertrochanteric line (Fig. 653). The nearer the fracture to the head of the femur the greater is the risk of interference with the blood supply of the head. The fracture is either an adduction fracture which is

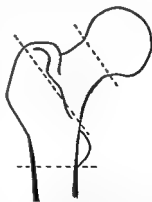


FIG. 653. The levels of fractures of the upper end of the femur, subcapital, peritrochanteric and subtrochanteric.

abdominal rigidity may be present in fractures of the pelvis without visceral injury, all patients with a fractured pelvis should be carefully examined for internal complications.

**Fractures of the Sacrum.** The margins of the sacro-iliac joint are commonly involved in pelvic injuries, but serious injury is only found in association with pelvic displacement. It is diagnosed by carefully comparing the sacral pattern on the two sides; good definition in the radiographs is essential. Sacral injury may involve the S1 and S2 nerve roots and produce a neurological syndrome resembling that of disc protrusion. Treatment of sacral fractures is similar to that of pelvic fractures with which they are so often combined.

**Coccygeal Injuries.** Fractures of the coccyx may occur in falls on the buttocks. More commonly the sacrococcygeal articulation is strained. Fractures seldom give trouble, but the vaguer injuries are apt to be followed by persistent pain, *coccydynia*. The patient complains of persistent pain on sitting, and often prefers to sit with only one buttock on the chair. On rectal examination, a tender coccygeal area is found. The pain is reproduced by manipulation of the coccyx. Treatment is difficult and often unsatisfactory. Local infiltration of the tissues around the coccyx with hydrocortisone or a long-acting local anæsthetic may be effective. Removal of the coccyx tends to perpetuate the pain in obsessional patients, but its removal may be necessary for cure.

## CHAPTER 100

### FRACTURES OF THE UPPER END OF THE FEMUR

FRACTURES of the upper end of the femur are the bane of the elderly in whom senile osteoporosis is the predisposing factor. They result from tripping, or falls on the great trochanter. The difficulties they present in treatment are due to the complications likely to occur during bed rest in the elderly. Although anatomical reposition is aimed at, the prime purpose of treatment is to permit early mobilisation of the patient. For this reason, open fixation is the best treatment; and if the patient's general condition is too bad for operation, hope of survival is slight.

The risks of bronchopneumonia have been greatly reduced by the use of antibiotics, which are administered routinely to such patients both before and after operation. Deep venous thrombosis and embolism are somewhat rare, especially if the patients are treated actively. More frequent and important is general deterioration indicated by mental changes, noisiness and disorientation at nights, apathy, and incontinence of bowel and bladder. This leads to the development of bed sores, especially over the sacrum, and they may become extensive and cause toxæmia. In spite of efforts to encourage activity, appetite and interest in the patient, a slow and unhalting decline may set in.

Such considerations do not influence the treatment of younger patients. In the young, however, it takes far greater force to fracture the femur, and there is consequently a greater risk of damage to soft tissues, and so to the blood supply to the head of the femur, with a consequent greater incidence of avascular necrosis. The child and adolescent tolerate fixation in plaster well; therefore for them immobilisation in a plaster spica is often the best treatment. In the young adult, operation is recommended, for it diminishes the risk of non-union in fractures of the femoral neck; produces a better anatomical reposition; and permits early exercise and weight-bearing.

**Classification.** (1) *Fracture of the Femoral Neck.*—The fracture may be close to the femoral head (subcapital) or nearer the intertrochanteric line (Fig. 653). The nearer the fracture to the head of the femur the greater is the risk of interference with the blood supply of the head. The fracture is either an adduction fracture which is

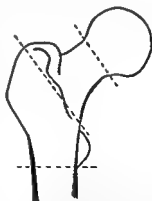


FIG. 653. The levels of fractures of the upper end of the femur, subcapital, peritrochanteric and subtrochanteric.

unimpacted and in which the neck of the femur is pulled up above the level of the fractured head, or an abduction fracture, in which the femoral head is turned posterolaterally over the femoral neck and impacted there with a variable degree of security. Should the fracture become disimpacted, it then assumes the characteristics of the adduction variety; for this reason and because an unfixed fracture cannot be safely exercised, impacted fractures are best treated as unimpacted fractures. (2) *Fracture of the Trochanteric Region* (intertrochanteric, transtrochanteric, or pertrochanteric, whichever is preferred). This fracture may be comminuted and there may be an associated fracture of the lesser trochanter. The average age of patients is seventy-five years (ten years older than those with subcapital fractures). Intertrochanteric fractures, like subcapital ones, may be impacted or unimpacted. They unite readily without risk of avascular necrosis. However, early operation is carried out to obtain good reduction and fixation so that rapid mobilisation of the patient is possible. (3) *The Subtrochanteric Fracture*.—This is really the highest fracture of the femoral shaft. The iliopsoas and the glutei flex and abduct the upper fragment. Although flexion and abduction of the femoral shaft brings it into line with the displaced upper fragment, the fracture is most readily fixed by insertion of a pin and plate as for an intertrochanteric fracture.

**Signs of Fractures of the Upper End of the Femur.** There is usually the history of a fall from which the patient cannot rise without assistance. Some function may remain when a fracture is impacted and the patient may take a few steps on the leg. The presence of such a fracture cannot be excluded clinically. Radiography of all patients with a suspicious history is essential. In the unimpacted fracture the leg lies *completely* externally rotated and powerless. There is measurable shortening, except in abduction fractures of the femoral neck, and in these there is also *slight* external rotation to distinguish them. Greater shortening may be found with pertrochanteric fractures than with fractures of the femoral neck, and there may be more palpable swelling around the trochanteric region. The differential diagnosis of the various fractures of the upper end of the femur may, however, be difficult before radiography.

**Immediate Treatment.** Although immediate operation may be carried out in younger patients, and has been also recommended for older patients, a preliminary period of skeletal traction has much to recommend it in the elderly. These patients are often worn out by pain and transportation; a good night's sleep before operation is therefore valuable. Time is also given to assess and, if necessary, improve the patient's condition, though, for the reasons already given, if operation cannot be carried out in the elderly, the chances of survival are poor. Preliminary continuous traction reduces the fracture and, in the case of the fractured femoral neck, releases any pressure on the soft tissues around the neck, and so gives the remaining blood supply of the femoral head the best chance of survival; delay in treatment is an important contributory

factor in avascular necrosis and non-union. If the fracture remains untreated for a week the prognosis is bad.

A Kirschner's wire or Steinmann's pin is inserted through the tibial tuberosity under a local anæsthetic, and the leg is elevated with the hip and knee flexed on a Braun's or bent Thomas's splint (Fig. 654). Some skin traction may be applied to the leg to steady the foot. This gives the patient considerable relief from pain, and slowly reduces the fracture; no manipulation is then necessary as a preliminary to open fixation. All violent manipulation is to be avoided, as it may damage the blood supply

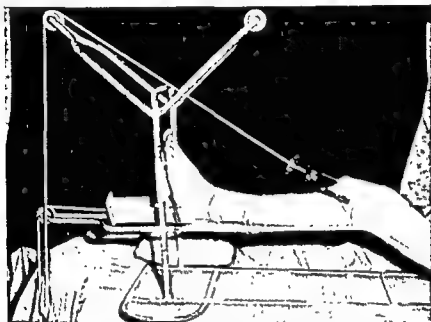


FIG. 654. The emergency treatment of fractures of the upper end of the femur. This consists of skin traction to the leg, and skeletal traction through the tibial tuberosity. The leg is supported on a bent Thomas's splint or, as in this case, on a Böhler-Braun splint.

of the femoral head. A weight of 8 to 10 lb. (4 to 5 kg.) on the tibia and 1 to 2 lb. ( $\frac{1}{2}$  to 1 kg.) on the skin traction on the leg is sufficient. No preliminary treatment is necessary for impacted abduction fractures, though skin traction on the leg may be used to steady the limb, and the weight of the bed clothes must be removed from the foot by a cradle.

**Definitive Treatment.** In young children a double hip spica or Bryant's method may be used for simple support to the fracture. In the older child or adolescent, continuous traction, having produced radiological reduction, may be maintained with reduced weight for retention until union has occurred. This should be sound in ten to twelve weeks. For the reasons given, such simple measures as a hip spica or continuous traction are unsuited in the aged. There may be one exception to this rule, namely, the firmly impacted abduction fracture; this may be

unimpacted and in which the neck of the femur is pulled up above the level of the fractured head, or an abduction fracture, in which the femoral head is turned posterolaterally over the femoral neck and impacted there with a variable degree of security. Should the fracture become disimpacted, it then assumes the characteristics of the adduction variety; for this reason and because an unfixed fracture cannot be safely exercised, impacted fractures are best treated as unimpacted fractures. (2) *Fracture of the Trochanteric Region* (intertrochanteric, transtrochanteric, or pertrochanteric, whichever is preferred). This fracture may be comminuted and there may be an associated fracture of the lesser trochanter. The average age of patients is seventy-five years (ten years older than those with subcapital fractures). Intertrochanteric fractures, like subcapital ones, may be impacted or unimpacted. They unite readily without risk of avascular necrosis. However, early operation is carried out to obtain good reduction and fixation so that rapid mobilisation of the patient is possible. (3) *The Subtrochanteric Fracture*.—This is really the highest fracture of the femoral shaft. The iliopsoas and the glutei flex and abduct the upper fragment. Although flexion and abduction of the femoral shaft brings it into line with the displaced upper fragment, the fracture is most readily fixed by insertion of a pin and plate as for an intertrochanteric fracture.

**Signs of Fractures of the Upper End of the Femur.** There is usually the history of a fall from which the patient cannot rise without assistance. Some function may remain when a fracture is impacted and the patient may take a few steps on the leg. The presence of such a fracture cannot be excluded clinically. Radiography of all patients with a suspicious history is essential. In the unimpacted fracture the leg lies *completely* externally rotated and powerless. There is measurable shortening, except in abduction fractures of the femoral neck, and in these there is also *slight* external rotation to distinguish them. Greater shortening may be found with pertrochanteric fractures than with fractures of the femoral neck, and there may be more palpable swelling around the trochanteric region. The differential diagnosis of the various fractures of the upper end of the femur may, however, be difficult before radiography.

**Immediate Treatment.** Although immediate operation may be carried out in younger patients, and has been also recommended for older patients, a preliminary period of skeletal traction has much to recommend it in the elderly. These patients are often worn out by pain and transportation; a good night's sleep before operation is therefore valuable. Time is also given to assess and, if necessary, improve the patient's condition, though, for the reasons already given, if operation cannot be carried out in the elderly, the chances of survival are poor. Preliminary continuous traction reduces the fracture and, in the case of the fractured femoral neck, releases any pressure on the soft tissues around the neck, and so gives the remaining blood supply of the femoral head the best chance of survival; delay in treatment is an important contributory

neck (Fig. 656*a*). In the anteroposterior view, the level of the head and its relation to the neck are observed (Fig. 656*b*).

A longitudinal incision is made over the greater trochanter, and deepened to the bone through the lower fibres of tensor fasciæ latæ and the upper fibres of vastus lateralis. Two inches below the line of attach-

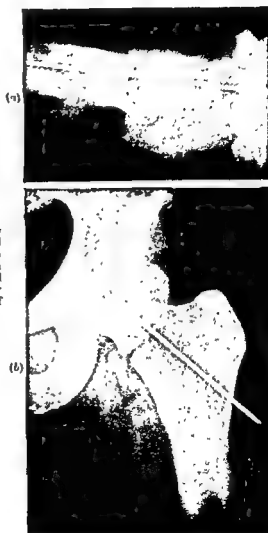


FIG. 656. Insertion of the guide wire. The wire is in excellent position in the lateral view (*a*), but in the anteroposterior film (*b*) the wire is a little high. This position had to be accepted as the patient had Paget's disease, and the sclerosis below the wire prevented a lower point of entry.

ment of the latter muscle to the greater trochanter, and in the midline of the bone a small hole  $\frac{1}{4}$  inch in diameter is gouged out. A guide wire is inserted through it parallel to the table top, and in the direction of the opposite anterior superior iliac spine (or the centre of the femoral head as judged by palpation of the femoral artery crossing it). The wire is inserted until it is felt to encounter the strong resistance of the articular surface of the femoral head. The operation is always more difficult in young patients, for the greater density of the bone makes introduction of the



treated in a short hip spica, but owing to the risk of disimpaction such a case must be carefully checked from time to time radiologically.

### OPERATIVE TREATMENT

**Fractures of the Femoral Neck.** A Smith-Petersen type of pin is used. Its advantages are : (a) Accurate apposition of the fragments is obtained ; (b) the fragments can be impacted, while being held rigidly in position, without risk of deviation of the femoral head ; (c) the early reduction and retention diminish the risk of non-union and avascular necrosis ; (d) early exercises and weight-bearing are permitted—of such vital importance in the elderly. The pin is three-flanged with a central canal which enables the pin to be passed along a guide wire to facilitate its insertion (Fig. 655). The flanges of the pin get a grip on the head of the femur and



FIG. 655. The Smith-Petersen pin.

prevent rotation. To permit early and safe weight-bearing, the pin must be inserted low in the femoral neck so that it lies tangential to the calcar femorale, and is supported by it. This means that a long pin is used, and that it lies at 45 degrees to the central axis of the femur. It is easier to insert a pin higher in the trochanter and nearer to a right angle with the femoral shaft, but on weight-bearing, the head becomes displaced downwards, carrying the pin lower in the femoral neck. With a pin so placed, therefore, weight-bearing has to be delayed till some union has occurred.

**Operative Technique.** In order to insert the pin in the desired position, two angles in the femoral neck have to be allowed for, namely, the angle of the neck to the shaft in the anteroposterior view (135 degrees) and the angle of anteversion of the femoral neck in the lateral view (20 degrees). Various appliances have been devised to facilitate calculation of these angles, but each appliance introduces its own special difficulties, and in practice the simple method to be described is effective.

The patient is placed on a Hawley table with the feet attached to the foot pieces, and the legs are abducted to between 45 and 60 degrees so that a lateral view of the femoral neck is possible. One X-ray machine lies on the inner side of the normal leg pointing to the affected hip, and another lies directly above the affected hip joint. The first angle to be considered is the angle of anteversion of the femoral neck, and the calculation of the line of insertion is facilitated by internally rotating the foot about 30 degrees, so that the neck of the femur lies parallel to the top of the operating table (it is easy to see that two lines—in this case the guide wire and the table top—are parallel, whereas it is difficult for the eye to allow for a few degrees of angulation). Anteroposterior and lateral radiographs are then taken. In the lateral view the neck of the femur should be in line with the greater trochanter and parallel to the top of the table, and the reduced femoral head should sit squarely on the femoral

head, but at the same time to allow for impaction. If too long a pin is chosen it may be driven into the joint. When the fracture is well reduced, and the guide wire point reaches the under surface of the articular cartilage, a nail  $\frac{1}{2}$  to  $\frac{3}{4}$  inch shorter than the length of the wire within the bone is chosen.

The nail is placed with two fins lying below, a nail starter is used to penetrate the cortex. The nail is then threaded over the guide and driven home. The guide is withdrawn. The traction on the leg is released, and with an impactor, which permits blows to be struck on the outer side of the great trochanter around the head of the nail, the fracture is impacted. This causes protrusion of the head of the nail which must then again be hammered flush with the femoral cortex. Check X-rays are taken, and if satisfactory the wound is closed (Fig. 657).

**Complications of Fracture of the Femoral Neck.** (1) *Avascular Necrosis.*—The blood supply of the femoral head arises from three sources. (a) The ligamentum teres—of little importance in the old. (b) The nutrient artery of the femur and its intramedullary branches. These are torn in fracture of the femoral neck. (c) Three arteries lying in the reflected ligaments along the femoral neck, and lying on its surface. These are the important vessels. They may be ruptured, kinked or thrombosed. Early reduction of the fracture is thus important to give them their best chance to continue to function. Injury to the blood vessels may cause: (a) Avascular necrosis, when the three vessels are torn; (b) non-union, an inevitable accompaniment of avascular necrosis, but also found in cases in which one or two vessels survive; (c) osteo-arthritis of the hip, which may be segmental, when one vessel is damaged producing avascular necrosis of a third of the head.

Avascular necrosis is indicated by the radiological changes in the density of the head previously outlined (Fig. 620). Non-union always occurs. The pin may be obtruded through the head, due to absorption of bone around the fracture site ("overdrive"). The condition becomes obvious about the sixth to eighth week. Hip function steadily deteriorates. The pin and the avascular head may be removed, together with a portion of the overhanging acetabulum, and a false joint allowed to develop, which is often very efficient and free from pain, or the femoral head may be replaced by a prosthesis of the Judet type, made of metal.

(2) *Non-union.* This occurs in association with complete or partial avascular necrosis of the femoral head. It is uncommon in the well-nailed and well-impacted fracture. Its incidence is a guide to the efficiency or otherwise with which the operation is carried out. In X-rays, non-union is indicated by failure of formation of new bone at the fracture site, and by the rounding off and increasing density of the surfaces of the fracture. Changes in the position of the pin may also be found. If the head is viable and the patient is young, an attempt may be made to obtain

wire and pin more difficult, and alters the surgeon's appreciation of position of the wire in the neck. The guide wire should penetrate about 4 inches, and lie at an angle of 45 degrees to the lateral femoral cortex. Check anteroposterior and lateral radiographs are then taken.

If the radiographs show the wire to lie in an unsatisfactory position a



(a)

FIG. 657. A Smith-Petersen pin inserted in the ideal position for early weight bearing. (a) Anteroposterior view. (b) Lateral view.



(b)

fresh guide wire is inserted at the appropriate angle to the old wire, which is then withdrawn. A new radiograph is taken in both planes. It is often better to insert the second wire through a fresh opening in the cortex, as the presence of the first wire may make it difficult to start the second wire in the correct direction. When a wire is inserted correctly, its length in the neck is calculated by measuring the length protruding and subtracting this from the known length of the wire. A pin of adequate length is then chosen. It must be long enough to get a good grip on the

have serious disadvantages in the elderly. In the child, however, a plaster may be most satisfactory.

**Operative Treatment.** As the femur cannot alone give the necessary support to a pin to permit early weight-bearing, a plate is attached to the pin (Fig. 658). Two types of fixation are available. The pin and plate may be at a fixed angle; they are then inserted with a template to make certain that the angle of insertion of the pin will enable the plate to lie on the surface of the femur. Alternatively, the angle of the pin and plate may be adjustable. If the adjustment is carried out by merely bending the plate, exercise later on may also bend it; therefore such appliances are not rigid enough. In the McLaughlin nail-plate, the plate and nail are bolted strongly together at any chosen angle (Fig. 658). The technique of inserting the nail is the same as that of the Smith-Petersen pin. It does not, however, have to be so accurately inserted, for it has more bone to grip on; this speeds up the operation. After its insertion, the plate is screwed to the femoral shaft and the junction of the nail and plate is secured. This needs a little longer incision than for insertion of the Smith-Petersen nail. In *subtrochanteric fractures* the treatment is the same.

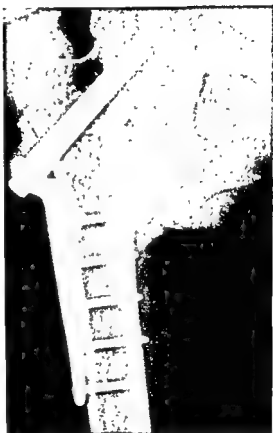


FIG. 658. A pertrochanteric fracture fixed with MacLaughlin nail-plate.

#### After-treatment of Elderly

##### Patients.

Routine antibiotics are given, not because of possible post-operative infection of the wound, but because of the risk of pulmonary infection. The patient is encouraged to sit up, and to sit out of bed if possible on the day following operation. Breathing exercises and leg exercises are begun as soon as the patient can co-operate. If the nail is in good position, weight-bearing is permitted as soon as the patient has good muscular control of the leg, usually at the end of the first week. If the position of the pin is not ideal, weight-bearing is postponed till the third or fourth week. The average stay in hospital of these patients treated by operation, including delays due to social difficulties in disposal, should not exceed seven weeks.

union by renailing and inserting a bone graft at the same time. This is often successful. In the elderly, early mobility is the aim. Therefore excision of the femoral head, or the insertion of a metal Judet prosthesis is preferred.

(3) *Osteo-arthritis*. The hips may already be the seat of osteo-arthritic changes. If these are severe, immediate arthroplasty may be preferable to nailing the neck. Osteo-arthritis is a frequent sequel to otherwise successful nailing. It is due to partial avascular necrosis, with death of a segment of the femoral head, or damage to the joint surfaces at the time of injury and the effects of long immobilisation. Moderate arthritic changes, if painless, may be tolerated. Pain may be partially relieved by physiotherapy or by administration of Butazolidin. If it becomes severe, a weight-relieving caliper may be tried, but old people do not tolerate the weight of the instrument well. Alternatively excision of the femoral head or displacement osteotomy of the McMurray type may be carried out.

Avascular necrosis occurs in 15 per cent. of cases, and osteo-arthritis develops in 15 per cent. This includes most of the cases of non-union, but non-union occurs in a further 5 per cent. At the end of two years, 35 per cent. of fractures of the femoral neck are more or less unsatisfactory. This fact had encouraged the performance of arthroplasty as an emergency procedure, but results are not thereby improved. A good nailing operation produces better results than any other treatment; the bad results of nailing are largely due to bad surgical technique. Results better than those indicated by the above figures are obtained by surgeons with long experience.

**Pertrochanteric Fractures.** These may be impacted or unimpacted. In the unimpacted fractures there is often comminution of the surrounding bone, e.g. separation of the lesser trochanter. Whether the fracture is impacted or not, the patient is unlikely to have walked on the leg. Shortening is often greater than that in subcapital fractures, but external rotation may not be so complete.

All pertrochanteric fractures unite, so that operation cannot be justified by risk of non-union. It is, however, justified by the risk to life of immobilisation in bed of an elderly patient for the time necessary for union to occur, and also by the difficulty in maintaining effective reduction of the fracture, so that a coxa vara does not develop. In this fracture the head and neck of the femur lie at a reduced angle to the femoral shaft—often at a right angle or less. This prevents proper function of the hip. A positive Trendelenberg sign appears, the patient being unable to balance on the injured hip. Training the patient to walk afterwards is therefore difficult and there is often a severe limp. The average length of stay in hospital for a patient with a correctly fixed fracture should be about six weeks. The conservative methods of treatment are continuous traction, a plaster spica, or a special well-leg traction splint. All these

Continuous traction is a valuable *emergency* measure. A Kirschner wire is inserted through the tibial tuberosity under local anæsthesia ; some skin traction is applied to the leg below the knee and the whole limb is then supported on a Braun's frame. A Thomas's splint may be used with a bend at the knee (Fig. 659) or a hinged attachment for the leg below the knee (Fig. 660). More manipulation is required to use this splint ; it is therefore reserved for patients who are fit enough for general anæsthesia. Continuous traction alone is not a good method of stabilising the femur, for angulation can occur at the fracture site; the added support of a Thomas's splint is needed, as the ring is stabilised by pressure against the pelvis. Temporary lateral splints of wood or padded Cramér wire may be strapped on to the splint to assist stabilisation while continuous traction is being applied. For a compound fracture, continuous traction alone is quite insufficient, for support and stabilisation of the soft tissues is as essential as it is for the bone. The combination of the Thomas's splint and plaster of Paris (the "Tobruk" splint) is particularly valuable for compound fractures of the femur (Fig. 659).

Manipulative reduction is needed in many cases. In comminuted, oblique and spiral fractures the thigh muscles tend to produce shortening, and the distension of the fascial envelope aids and abets it. Although continuous traction in these circumstances usually produces full restoration of length, if it can be obtained under general anæsthesia, there is a greater certainty of it being maintained afterwards. For these fractures continuous traction is needed to begin with, but it may be changed to fixed traction as callus forms in the third week. If the fracture is transverse, unless the ends can be interlocked, there will be shortening and usually angulation. If the ends can be interlocked, the fracture becomes more stable ; the amount of traction can be reduced, and angulation is more easily dealt with. The ends cannot, however, always be easily interlocked. This is due to distension of the fascial envelope, interposition of soft tissues or a fragment of bone. A second attempt should be made to interlock the fragments when swelling has subsided at the end of the first week. In the meantime, continuous traction should be used.

If the fracture can be reduced and held in a stable position by the attachments of a Hawley table, it is possible to apply a long hip spica for retention. This is a clumsy method in adults ; it is impossible in a heavy adult, but may occasionally be useful in the older child or the young adolescent.

The Thomas's splint provides the most satisfactory basis for fixation (Figs. 659 and 660). It is very adaptable, for it can be used with the knee straight or flexed ; it can be altered for early knee flexion exercises, and can be used for fixed or continuous traction, or combined with plaster of Paris. It is particularly valuable when a fracture of the tibia has occurred on the same side as a fracture of the femur. The tibia can be fixed in plaster and then the femur fixed with skeletal traction to the Thomas's splint.

## CHAPTER 101

### FRACTURES OF THE SHAFT OF THE FEMUR

THE shaft of the femur is the strongest long bone in the body and the force required to break it therefore is very great. It is consequently not surprising that fractures of the shaft of the femur are often comminuted, widely displaced and often indirectly compound. They present problems in treatment which are not easily overcome, and demand unremitting care if successful results are to be obtained. The difficulties encountered in treatment are determined by the following facts : (1) In fractures of the upper third of the shaft, the iliopsoas tends to flex the upper fragment. The thigh must therefore be flexed to a similar degree to obtain alignment, or open operation undertaken (see also subtrochanteric fractures). (2) Severe swelling of the thigh distends the fascial envelopes of the quadriceps and other muscles, and makes it impossible to obtain full distraction till it subsides. Continuous traction is necessary to obtain gradual reduction while the swelling is subsiding. (3) The bulky muscles around the thigh prevent direct pressure on bone, and tend to nullify attempts to control angulation by local pressure. (4) Because of the presence of the mass of the hamstrings, gravity tends to produce sagging of the badly supported fragments, and because the femur is normally bowed forward, a pad must always be placed behind the fractured bone. (5) In fracture of the shaft of the femur the quadriceps is always damaged, and becomes adherent to the bone. The nearer the fracture is to the knee the more likely is the gliding mechanism of the quadriceps to be affected and the knee to become stiff. Although early quadriceps exercises are important, to minimise adhesions, limitation of movement of the knee is usually due to damage to the quadriceps at the time of the accident, provided treatment has been correctly carried out. (6) Surgical approaches through the quadriceps also provoke muscular adhesions to the bone, and are therefore to be avoided. The high lateral approach for fractures of the upper third of the femur is less damaging. For fractures in the lower two-thirds, a posterolateral approach is advised. It passes behind the vastus lateralis and thus does not produce adhesions.

**Methods of Treatment.** The following methods are available ; sometimes they may be combined, or one may be replaced by another as the progress of the patient dictates : (1) Continuous skeletal traction on a Thomas's or Braun's splint. (2) Manipulative reduction and fixed traction on a Thomas's splint. (3) Manipulative reduction and plaster of Paris, or the combination of a Thomas's splint and plaster. (4) Open operation and fixation. (5) Continuous skin traction for children under four (Bryant's method).

There are many ways in which traction and fixation of the femur can be combined on a Thomas's splint. It is first of all important to see that the ring fits well, and does not slide off the ischial tuberosity. If a perfect fit cannot be obtained, it can be much improved by wrapping a pad of cotton wool in plaster of Paris and inserting it wet between the ring and the great trochanter. If fixed distraction is to be used, the traction cord of the skin or skeletal traction apparatus is tied to the end of the splint, and thus the ischial tuberosity acts as a counter-pressure point. This may be painful, but the pressure can be reduced by traction applied to the lower end of the splint—a combination of fixed and continuous traction. If the splint is merely to be used as a cradle for the leg, it is suspended from a Balkan beam. Traction in the line of the femur is usually steadied by skin traction of a few pounds in the line of the leg (Fig. 660). If it is felt that the ring of the Thomas's splint must be kept firmly against the ischium, the below-knee traction may be passed over a pulley attached to the end of the bent Thomas's splint, while the traction is continued in the line of the femur. This is often a useful combination.

The limb is supported on the Thomas's splint by a series of slings of some firm material. It is necessary, however well these are applied, to place a large pad behind the lower fragment to maintain the forward bowing of the shaft of the femur. The limb may then be further steadied by bandages passed over the splint and slings. Much firmer support is obtained by the use of plaster bandages. If skeletal traction is employed, the wire or pin is incorporated in the plaster, and the pull is then applied to both splint and wire. It must be increased a little to overcome the friction of the splint on the bed clothes. This method is thus another combination of fixed and continuous traction. The leg should be padded with cotton wool before the plaster is applied, and the plaster should be moulded around the iron bars with the fingers. If inspection of the limb becomes necessary, the plaster may be split down both sides and the top lifted off.

Traction is usually applied through the tibial tuberosity, but may be applied through the lower end of the femur, although care must be taken to see that the pin or wire does not transfix the hæmatoma, as there would be a risk of infection. If the tibial tuberosity cannot be used because of an associated fracture of the tibia, the calcaneus may be used instead. In general, traction is bad for intervening joints and must therefore be reduced to the minimal amount necessary, and applied for the minimal time; 10 to 15 lb. (5 to 8 kg.) is usually all that is needed and this may be reduced to 5 lb. as soon as the length of the leg is restored. This is especially so if the pin or wire is incorporated in the plaster to maintain fixed distraction. The tendency of the femur to shorten disappears about the fourth week with the formation of callus. If the fracture is comminuted or badly displaced, traction is necessary for a longer time.





FIG. 659. The Tobruk splint (plaster of Paris bandages are applied around the limb and the Thomas's splint).

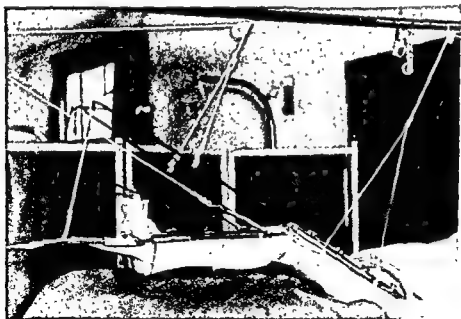


FIG. 660. The slung Thomas's splint and Pearson knee flexion attachment, in use with traction through the tibial tuberosity. This arrangement, when properly balanced, permits knee flexion exercises in the appliance.

There are many ways in which traction and fixation of the femur can be combined on a Thomas's splint. It is first of all important to see that the ring fits well, and does not slide off the ischial tuberosity. If a perfect fit cannot be obtained, it can be much improved by wrapping a pad of cotton wool in plaster of Paris and inserting it wet between the ring and the great trochanter. If fixed distraction is to be used, the traction cord of the skin or skeletal traction apparatus is tied to the end of the splint, and thus the ischial tuberosity acts as a counter-pressure point. This may be painful, but the pressure can be reduced by traction applied to the lower end of the splint—a combination of fixed and continuous traction. If the splint is merely to be used as a cradle for the leg, it is suspended from a Balkan beam. Traction in the line of the femur is usually steadied by skin traction of a few pounds in the line of the leg (Fig. 660). If it is felt that the ring of the Thomas's splint must be kept firmly against the ischium, the below-knee traction may be passed over a pulley attached to the end of the bent Thomas's splint, while the traction is continued in the line of the femur. This is often a useful combination.

The limb is supported on the Thomas's splint by a series of slings of some firm material. It is necessary, however well these are applied, to place a large pad behind the lower fragment to maintain the forward bowing of the shaft of the femur. The limb may then be further steadied by bandages passed over the splint and slings. Much firmer support is obtained by the use of plaster bandages. If skeletal traction is employed, the wire or pin is incorporated in the plaster, and the pull is then applied to both splint and wire. It must be increased a little to overcome the friction of the splint on the bed clothes. This method is thus another combination of fixed and continuous traction. The leg should be padded with cotton wool before the plaster is applied, and the plaster should be moulded around the iron bars with the fingers. If inspection of the limb becomes necessary, the plaster may be split down both sides and the top lifted off.

Traction is usually applied through the tibial tuberosity, but may be applied through the lower end of the femur, although care must be taken to see that the pin or wire does not transfix the hæmatoma, as there would be a risk of infection. If the tibial tuberosity cannot be used because of an associated fracture of the tibia, the calcaneus may be used instead. In general, traction is bad for intervening joints and must therefore be reduced to the minimal amount necessary, and applied for the minimal time; 10 to 15 lb. (5 to 8 kg.) is usually all that is needed and this may be reduced to 5 lb. as soon as the length of the leg is restored. This is especially so if the pin or wire is incorporated in the plaster to maintain fixed distraction. The tendency of the femur to shorten disappears about the fourth week with the formation of callus. If the fracture is comminuted or badly displaced, traction is necessary for a longer time.

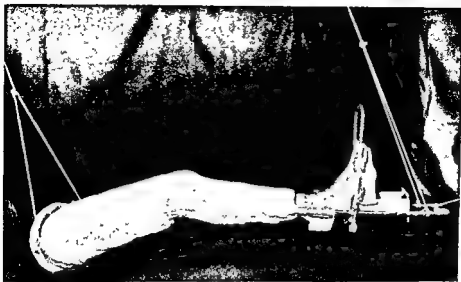


FIG. 659. The Tobruk splint (plaster of Paris bandages are applied around the limb and the Thomas's splint).

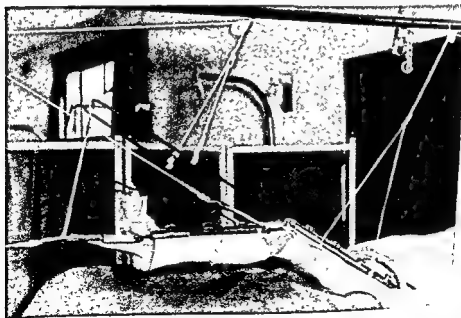


FIG. 660. The slung Thomas's splint and Pearson knee flexion attachment, in use with traction through the tibial tuberosity. This arrangement, when properly balanced, permits knee flexion exercises in the appliance.

There are many ways in which traction and fixation of the femur can be combined on a Thomas's splint. It is first of all important to see that the ring fits well, and does not slide off the ischial tuberosity. If a perfect fit cannot be obtained, it can be much improved by wrapping a pad of cotton wool in plaster of Paris and inserting it wet between the ring and the great trochanter. If fixed distraction is to be used, the traction cord of the skin or skeletal traction apparatus is tied to the end of the splint, and thus the ischial tuberosity acts as a counter-pressure point. This may be painful, but the pressure can be reduced by traction applied to the lower end of the splint—a combination of fixed and continuous traction. If the splint is merely to be used as a cradle for the leg, it is suspended from a Balkan beam. Traction in the line of the femur is usually steadied by skin traction of a few pounds in the line of the leg (Fig. 660). If it is felt that the ring of the Thomas's splint must be kept firmly against the ischium, the below-knee traction may be passed over a pulley attached to the end of the bent Thomas's splint, while the traction is continued in the line of the femur. This is often a useful combination.

The limb is supported on the Thomas's splint by a series of slings of some firm material. It is necessary, however well these are applied, to place a large pad behind the lower fragment to maintain the forward bowing of the shaft of the femur. The limb may then be further steadied by bandages passed over the splint and slings. Much firmer support is obtained by the use of plaster bandages. If skeletal traction is employed, the wire or pin is incorporated in the plaster, and the pull is then applied to both splint and wire. It must be increased a little to overcome the friction of the splint on the bed clothes. This method is thus another combination of fixed and continuous traction. The leg should be padded with cotton wool before the plaster is applied, and the plaster should be moulded around the iron bars with the fingers. If inspection of the limb becomes necessary, the plaster may be split down both sides and the top lifted off.

Traction is usually applied through the tibial tuberosity, but may be applied through the lower end of the femur, although care must be taken to see that the pin or wire does not transfix the hæmatoma, as there would be a risk of infection. If the tibial tuberosity cannot be used because of an associated fracture of the tibia, the calcaneus may be used instead. In general, traction is bad for intervening joints and must therefore be reduced to the minimal amount necessary, and applied for the minimal time; 10 to 15 lb. (5 to 8 kg.) is usually all that is needed and this may be reduced to 5 lb. as soon as the length of the leg is restored. This is especially so if the pin or wire is incorporated in the plaster to maintain fixed distraction. The tendency of the femur to shorten disappears about the fourth week with the formation of callus. If the fracture is comminuted or badly displaced, traction is necessary for a longer time.

*Bryant's Method.* In children, the skin area is greater in proportion to the body-weight and it is therefore possible to maintain adequate extension with skin traction. The child's body-weight forms the best counter-traction, and in Bryant's method both legs are slung vertically so that the buttocks are just lifted off the bed. This is comfortable for children and facilitates nursing. A preliminary manipulation and traction to restore full length may be necessary. Callus forms rapidly in children and satisfactory reduction should be achieved by the end of the first ten days. Alternatively, a small Thomas's splint or a plaster hip spica may be used in children.

**Special Problems.** Double fractures of the femoral shaft present the same difficulties as double fractures elsewhere. The two fractures are more difficult to control, and the central fragment, being partially or completely deprived of its blood supply, delays or prevents union. Sometimes it is possible to fix one fracture by open operation, e.g. a sub-trochanteric fracture, and then treat the other by one of the routine methods. Often the risk of further disturbing the blood supply is too great, and the fracture must be left to look after itself until it is clear that interference is needed to obtain union. Some improvement in blood supply will have occurred by then.

Comminution may be considerable and yet produce little effect on union, though it increases the adhesions to the quadriceps. Sometimes a single "butterfly" fragment may become avascular and delay or prevent union. If the fracture is compound, there is a temptation to remove loose fragments, but if they are large, their removal carries a definite risk of non-union; therefore the fragments should be left to act as bone grafts.

Compound fractures are treated on the same general principles as compound fractures elsewhere. Shock is great in fractures of the femur. Loss of blood into the thigh may be considerable, without much external escape of blood. Transfusion is often required. Soft tissue immobilisation, as well as fixation of the bones, is important, and for this purpose the combination of a Thomas's splint, with skeletal traction through the tibial tuberosity and plaster of Paris over the limb, including the traction wire, is invaluable.

Fractures of the femur are firm about the *fourth* month, though active non-weight-bearing knee exercises may be begun well before this. Delayed union is common in complicated fractures, and once smooth sclerotic bone ends are seen radiologically, there is little hope of union. Open operation must then be undertaken. There is seldom any need to use a graft to obtain union, unless there has been much bone loss. The bone ends are freshened and fixed with a plate and screws, or by an intramedullary nail (*vide infra*). If grafts are to be used, the best method is to pack cancellous chips around the bone ends and the plate.

**Open Fixation.** In a clean compound fracture, advantage may be

taken of the open wound to fix the fracture. The incision is then planned to include the wound and to add minimal damage to the quadriceps. Fractures of the upper third are plated through a lateral approach. Fractures of the lower two-thirds of the shaft are approached laterally or from behind. The only difficulty encountered is bleeding from the perforating vessels, but these have often been thrombosed as a result of the fracture. Fixation of the fracture with a long Eggers' plate and screws is satisfactory (Fig. 628), but requires a longer period of external support than fixation with a Küntscher nail (Fig. 629). The latter requires an approach to the trochanter for insertion, and to be efficient must be inserted with care and precision. For the inexperienced surgeon the plate and screws are safer.

**Graduated Return of Function.** In the treatment of a fractured femur graduated increase in function is the aim. Early static quadriceps exercises are instituted. Too early abandonment of splinting results in bowing of the femur by the strongest muscle groups, the adductors and the hamstrings. A scheme of management for a compound fracture of the femoral shaft is as follows: (1) Skeletal traction in a Thomas's splint and plaster of Paris (Tobruk plaster)—two weeks. (2) Removal of plaster followed by replaster to correct any deformity, and to take up slack due to subsidence of swelling and disuse atrophy. (3) Removal of traction wire in fourth week. (4) Removal of plaster in sixth week, for better access to quadriceps for exercises. (5) Addition of a knee-flexion splint between the eighth and tenth week and increased exercises including knee bending. (6) Abandonment of splint and skin traction about the twelfth week, but patient remains in bed. (7) Patient gets up on crutches and gradual weight-bearing begins from the fourteenth week. (8) Full weight-bearing and active exercises from the sixteenth week.

If there is delay in union, but it is desirable to get the patient up, a plaster pylon may be made taking weight through the ischial tuberosity. This has the disadvantage of fixing the knee, but if the union is uncertain it may be necessary. Alternatively, a knee flexion device may be incorporated in the splint, or a knee flexion caliper used.

In children the rate of union is much more rapid; thus the femur is firm in six to eight weeks. No fear of joint stiffness affects treatment; therefore the knee may be kept immobile throughout treatment.

*Bryant's Method.* In children, the skin area is greater in proportion to the body-weight and it is therefore possible to maintain adequate extension with skin traction. The child's body-weight forms the best counter-traction, and in Bryant's method both legs are slung vertically so that the buttocks are just lifted off the bed. This is comfortable for children and facilitates nursing. A preliminary manipulation and traction to restore full length may be necessary. Callus forms rapidly in children and satisfactory reduction should be achieved by the end of the first ten days. Alternatively, a small Thomas's splint or a plaster hip spica may be used in children.

*Special Problems.* Double fractures of the femoral shaft present the same difficulties as double fractures elsewhere. The two fractures are more difficult to control, and the central fragment, being partially or completely deprived of its blood supply, delays or prevents union. Sometimes it is possible to fix one fracture by open operation, e.g. a sub-trochanteric fracture, and then treat the other by one of the routine methods. Often the risk of further disturbing the blood supply is too great, and the fracture must be left to look after itself until it is clear that interference is needed to obtain union. Some improvement in blood supply will have occurred by then.

Comminution may be considerable and yet produce little effect on union, though it increases the adhesions to the quadriceps. Sometimes a single "butterfly" fragment may become avascular and delay or prevent union. If the fracture is compound, there is a temptation to remove loose fragments, but if they are large, their removal carries a definite risk of non-union; therefore the fragments should be left to act as bone grafts.

Compound fractures are treated on the same general principles as compound fractures elsewhere. Shock is great in fractures of the femur. Loss of blood into the thigh may be considerable, without much external escape of blood. Transfusion is often required. Soft tissue immobilisation, as well as fixation of the bones, is important, and for this purpose the combination of a Thomas's splint, with skeletal traction through the tibial tuberosity and plaster of Paris over the limb, including the traction wire, is invaluable.

Fractures of the femur are firm about the *fourth* month, though active non-weight-bearing knee exercises may be begun well before this. Delayed union is common in complicated fractures, and once smooth sclerotic bone ends are seen radiologically, there is little hope of union. Open operation must then be undertaken. There is seldom any need to use a graft to obtain union, unless there has been much bone loss. The bone ends are freshened and fixed with a plate and screws, or by an intramedullary nail (*vide infra*). If grafts are to be used, the best method is to pack cancellous chips around the bone ends and the plate.

*Open Fixation.* In a clean compound fracture, advantage may be

bone resulting from operative interference. Open operation should be limited to the rare fractures in which a single femoral condyle is displaced, and cannot be reduced by manipulation. Open reduction and fixation with a screw or screws may then be advisable.

The routine treatment in other fractures should be insertion of a Steinmann's pin in the tibia, and aspiration of the hæmarthrosis. The leg is supported on a slung Thomas's splint for a few days till the acute swelling has subsided, and then a counterbalanced knee flexion splint is applied. This may consist of a modified Thomas's splint, or slings may merely be passed under the femur and under the calf and counter-weighted. Knee flexion exercises are begun and gradually increased in range. Five to 7 lb. (2 to 3 kg.) countertraction is all that is required and this may be maintained for a month or more. In six to eight weeks patients may be allowed up on crutches, but are not permitted to bear weight till after the third month. Occasionally in a minimal injury non-weight-bearing ambulation on crutches and exercises are permissible after a few days.

**Fractures of the Tibial Condyles (*Fractures of Tibial Plateau*).** (1) Comminuted and crush fractures with severe surface damage. (2) Inverted T-fracture, often impacted and sometimes with little change in plateau levels. (3) Isolated condylar fractures, usually the lateral condyle. Although treatment can often be reduced to the same simplicity as in condylar fractures of the femur, there is a greater variety of fractures to be considered, and there is also an interrelation with ruptures of the collateral ligaments of the knee joint. Compression injury may produce the severely comminuted tibial plateau, or the impacted T-fracture into the joint. Generally speaking, however, the tibial plateau retains a roughly even level, and little can be done to improve it. The correct treatment is that outlined for the corresponding femoral fracture, but the Steinmann's pin must be placed a little lower in the tibia to avoid transfixing the hæmatoma. Early exercises are instituted and particular care is taken to obtain full extension. Sometimes the knee is so stable that all that is necessary is skin traction to the leg, maintained over a pulley at the end of the bed. As soon as the fragments are consolidated, patients are allowed non-weight-bearing activity on crutches. These injuries are most common after middle life, and many patients, being elderly, may have some trouble balancing on crutches.

In the few cases in which there is marked displacement of a part of the tibial plateau, attempts may be made to replace it. Open operation is not usually advisable, owing to the risk of avascular necrosis. The simplest method is the application of temporary very firm compression with an Esmarch bandage, or pressure may be applied by means of a Böhler compression clamp. A displaced fragment may be levered into position by transfixion with a Steinmann's pin.

The knee may also be injured by abduction or adduction forces. Adduction injury is the more common and is due to pressure on the outer



## CHAPTER 102

### FRACTURES AROUND THE KNEE JOINT

THE aim of treatment is the preservation of knee movement. Fractures seldom jeopardise the stability of the joint ; early exercises are therefore advisable. A certain uniformity of approach is thus possible to fractures above and below the joint. There are three separate but comparable groups of fractures on either side of the joint. Fractures of the patella are considered as a separate problem.

**Supracondylar Fractures of the Femur.** These fractures are uncommon. The gastrocnemius tends to rotate the short condylar fragment posteriorly while the lower end of the upper fragment penetrates the suprapatellar bursa and thus causes stiffness of the knee. There is risk of injury to the popliteal structures. Relaxation of the gastrocnemius is essential for reduction, but immobilisation in flexion, if followed by quadriceps adhesions, may make rehabilitation difficult, and it may require a plastic operation on the quadriceps to restore extension. So difficult may this fracture be to control, that the cardinal principle of not transfixing a hæmatoma must be ignored. A Kirschner wire is run through the lower fragment as near the fracture line as possible, and with the knee flexed and stabilised by light traction through the tibial tuberosity, a pull is exerted at right angles to the femoral shaft. The upper fragment may have to be steadied by downward pressure of the hand or by means of a sling. Check radiography is necessary during reduction. When this is achieved, both wires are incorporated in a plaster cast from groin to ankle, and the whole supported on a Thomas's splint with a bend at the knee. Only light traction is maintained through the tibial tuberosity. At the end of a fortnight, a circular cut is made in the plaster around the knee, which is gradually extended, by straightening the Thomas's splint. Full extension of the knee should be obtained by the fourth week, and the wire may be removed at the end of the sixth week (earlier in children). Exercises are not advisable until after the pin has been removed for fear of infecting the wire track. The range of flexion achieved may be disappointing, but if full or almost full extension is possible, the patient will be able to walk reasonably well.

**Fractures of the Femoral Condyles.** (1) Comminuted and crush fractures with severe surface damage. (2) T- or Y-shaped fractures separating both condyles. (3) Isolated condylar fractures. There is a temptation to try to restore the joint surfaces to normal by open operation, and to fix the fragments with plates, screws or bolts, or to pack spaces with bone chips. These procedures are not only technically difficult, but often unsatisfactory, and there is a risk of avascular necrosis of fragments of

(2) A Robert Jones' compression bandage and skin traction below the knee. (3) Skeletal traction through the tibia, and slings under the thigh and calf, with early exercises. A knee flexion device may be substituted for the slings. (4) A knee fixation plaster. This may be used to permit early walking in minimal injuries in young people when preliminary exercises have shown little likelihood of stiffness.

It is often remarkable how much function returns to the knee despite severe damage. In younger patients, traumatic arthritis may be an inevitable late sequel, but the knee usually functions well for many years before this develops. The duration of fixation and avoidance of weight-bearing depends on the severity of the injury and varies from four weeks to three months.

**Fractures of the Patella.** There are two guiding principles in the treatment of fractures of the patella. The first is that if the lateral insertions of the quadriceps (the patellar retinaculæ) are torn, their repair is essential regardless of the state of the bone. The second is that the articular surface of the patella must be restored to its normal contour, if subsequent patello-femoral arthritis is to be avoided. In severe damage to the patella when restoration of the smooth surface is impossible, it is best to excise the bone, but compromises are possible as with other fractures, and indeed are desirable, as the patella is not to be lightly discarded.

Fractures of the patella may occur from direct or indirect violence. In direct injuries the bone is broken inside its capsule of fibrous tissue and the fragments remain interlocked. The degree of shattering is variable, as is the degree of damage to the opposed femoral articular surface. In an elderly patient with an irregular undisplaced fissure fracture, concentration on active knee exercises may be all that is required. Very occasionally in direct injuries an almost transverse fracture occurs. The slight gap and any alteration in level of the articular surfaces of the two fragments may be corrected in the younger patient with crossed screws through the fracture line. If the patella is grossly shattered, and especially if there is damage to the femur, excision is the best treatment (Fig. 661).

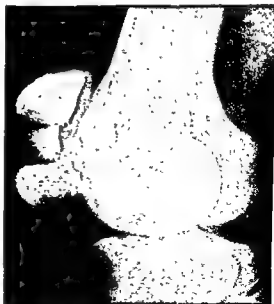


FIG. 661. A comminuted fracture of the patella, best treated by excision.

side of the knee, as from the bumper of a car. Abduction or adduction may be combined with a variable degree of compression. If, however, the force is one producing only angulation of the knee, the stresses are evenly distributed between the condyle which is compressed and the opposite ligament which is stretched. If the ligament ruptures, then the pressure on the condyle is released. If at the time this occurs, the knee is under compression, rupture of the ligament may not prevent fracture. Therefore it is possible to have a depressed condylar fracture, and a rupture of the opposite collateral ligament, though it is more usual to find the ligament intact if the condyle gives way, or condyle intact if the ligament gives way.

Fractures of the condyles are variable. They may consist of an almost vertical split; a depressed area may be surrounded by intact cortical bone, or a condyle may be moved downwards and outwards *in toto*. With fractures of the lateral condyle the neck of the fibula is frequently fractured. If the angulation is great, one of the cruciate ligaments may be partially or completely ruptured (with rupture of a collateral ligament). This adds to the instability of the knee, but, apart from making sure that the knee is fully extended, especially if there has been avulsion of the anterior cruciate ligament with its bony attachment which tends to lock the joint, there is no specific treatment required, as experience shows that repair of the cruciate ligaments is unsatisfactory and uncertain.

The picture may be further complicated by avulsion of various fragments of bone around the joint corresponding to ligamentous or fascial attachments. The fragments may arise from: (1) The tip of the fibula, the attachment of the fibular collateral ligament. (2) The upper end of the fibula, the attachment of the biceps femoris. (3) A flake from the lateral tibial condyle, the attachment of the fascia lata. (4) A flake from the medial femoral condyle, the attachment of the medial collateral ligament. (5) A flake from the surface of the tibia (rare), the lower attachment of the medial collateral ligament. (6) Marginal fragments, attachments of the coronary ligaments. (7) Fragments anterior and posterior to the tibial spine avulsed by the cruciate ligaments.

These detached fragments serve as a valuable indication of ligament injury. It is most important that complete ruptures of the collateral ligaments of the knee joint should be repaired accurately by open operation.

**Summary of Treatment.** Early exercises are important. Aspiration of the knee is often valuable to relieve pain and to avoid adhesions. If skeletal traction is to be used, a Steinmann's pin is passed through the tibia usually at a lower level than the tuberosity to avoid entering the hæmatoma. The following degrees of fixation are adapted to the degree of damage to the tibial plateau and the age of the patient. (1) A Robert Jones' compression bandage\* only and bed rest with early exercises.

\* A Robert Jones' compression bandage consists of a layer of wool and a calico bandage, covered by another layer of wool and a bandage.

(2) A Robert Jones' compression bandage and skin traction below the knee. (3) Skeletal traction through the tibia, and slings under the thigh and calf, with early exercises. A knee flexion device may be substituted for the slings. (4) A knee fixation plaster. This may be used to permit early walking in minimal injuries in young people when preliminary exercises have shown little likelihood of stiffness.

It is often remarkable how much function returns to the knee despite severe damage. In younger patients, traumatic arthritis may be an inevitable late sequel, but the knee usually functions well for many years before this develops. The duration of fixation and avoidance of weight-bearing depends on the severity of the injury and varies from four weeks to three months.

**Fractures of the Patella.** There are two guiding principles in the treatment of fractures of the patella. The first is that if the lateral insertions of the quadriceps (the patellar retinaculæ) are torn, their repair is essential regardless of the state of the bone. The second is that the articular surface of the patella must be restored to its normal contour, if subsequent patello-femoral arthritis is to be avoided. In severe damage to the patella when restoration of the smooth surface is impossible, it is best to excise the bone, but compromises are possible as with other fractures, and indeed are desirable, as the patella is not to be lightly discarded.

Fractures of the patella may occur from direct or indirect violence. In direct injuries the bone is broken inside its capsule of fibrous tissue and the fragments remain interlocked. The degree of shattering is variable, as is the degree of damage to the opposed femoral articular surface. In an elderly patient with an irregular undisplaced fissure fracture, concentration on active knee exercises may be all that is required. Very occasionally in direct injuries an almost transverse fracture occurs. The slight gap and any alteration in level of the articular surfaces of the two fragments may be corrected in the younger patient with crossed screws through the fracture line. If the patella is grossly shattered, and especially if there is damage to the femur, excision is the best treatment (Fig. 661).

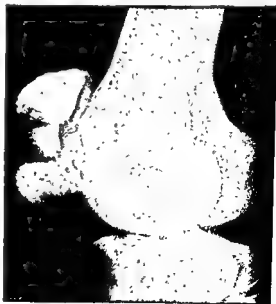


FIG. 661. A comminuted fracture of the patella, best treated by excision.

Fractures of the patella by indirect violence occur in attempts to control the bent knee by violent contraction of the quadriceps. The patella caught across the femoral condyles is torn in half and *the retinaculæ on each side are rent apart*. The patellar fragments separate; communication is established between the prepatellar bursa and the knee joint, and often both are filled with blood and synovial fluid. All power of lifting the extended knee is lost. This is in contrast to direct injuries in which the continuity of the quadriceps is not lost. The gap between the two fragments may be palpated if not obscured by hæmatoma. A hæmatoma in the prepatellar bursa may sometimes give a deceptive feeling of a gap.

Although the fracture is commonly through the centre of the patella, a fragment may be avulsed from its upper margin, or the small almost non-articular lower pole may be avulsed. In these cases, the small fragment is excised, and the continuity of the patellar ligament restored by suturing it to the bone. If the joint is already arthritic; if there has been comminution of the margins of the fracture; or there are technical difficulties in reforming a nearly perfect articular surface, the patella may be excised *and the retinaculæ repaired at the same time*. They can often be repaired in two layers, synovial and fascial.

*Excision of the Patella.* A tourniquet is first applied to the thigh. The secret of success is careful suture of the quadriceps tendon and the patellar ligament so that early quadriceps and flexion exercises can be begun without fear of opening up the suture line. This can be achieved by use of plaited stainless steel wire as suture material; it is passed as a figure-of-eight through both ligament and tendon. The hæmatoma around the fractured fragments is easily entered through a curved vertical or horizontal incision. The two fragments of bone are excised, and the quadriceps tendon is pulled down by means of two sharp hooks. The stainless steel sutures are then inserted and tied, while the two tendons are held in contact. Tissue can often be overlapped about the midline. The retinaculæ if torn are repaired. The tourniquet is released and complete hæmostasis obtained (a hæmatoma under the prepatellar skin may produce skin gangrene). A Robert Jones' compression bandage is applied. The after-treatment differs little from that after meniscectomy though progress is slower. Full strong extension of the knee is aimed at. Gentle flexion of the knee is begun early, but is slower in returning than extension.

*Restoration of the Patella.* This has been attempted with circular sutures, and with sutures partially transfixing the bone. Neither are satisfactory, as the fragments tend to tilt on each other, and to separate, and therefore there is anxiety about allowing early exercises. The only satisfactory method of fixing the two halves in perfect apposition (after inspection of the margins of the fracture to be assured of a perfect fit) is to restore perfectly the visible outer surface of the bone and to fix the two

fragments with screws crossing at different levels in the bone ; they can be conveniently inserted at the depressions on each side of the lower pole. Laterally inserted staples may also be used. Early active exercises are the essential feature of post-operative treatment.

Fractures of the patella by indirect violence occur in attempts to control the bent knee by violent contraction of the quadriceps. The patella caught across the femoral condyles is torn in half and *the retinaculæ on each side are rent apart*. The patellar fragments separate; communication is established between the prepatellar bursa and the knee joint, and often both are filled with blood and synovial fluid. All power of lifting the extended knee is lost. This is in contrast to direct injuries in which the continuity of the quadriceps is not lost. The gap between the two fragments may be palpated if not obscured by hæmatoma. A hæmatoma in the prepatellar bursa may sometimes give a deceptive feeling of a gap.

Although the fracture is commonly through the centre of the patella, a fragment may be avulsed from its upper margin, or the small almost non-articular lower pole may be avulsed. In these cases, the small fragment is excised, and the continuity of the patellar ligament restored by suturing it to the bone. If the joint is already arthritic; if there has been comminution of the margins of the fracture; or there are technical difficulties in reforming a nearly perfect articular surface, the patella may be excised *and the retinaculæ repaired at the same time*. They can often be repaired in two layers, synovial and fascial.

*Excision of the Patella.* A tourniquet is first applied to the thigh. The secret of success is careful suture of the quadriceps tendon and the patellar ligament so that early quadriceps and flexion exercises can be begun without fear of opening up the suture line. This can be achieved by use of plaited stainless steel wire as suture material; it is passed as a figure-of-eight through both ligament and tendon. The hæmatoma around the fractured fragments is easily entered through a curved vertical or horizontal incision. The two fragments of bone are excised, and the quadriceps tendon is pulled down by means of two sharp hooks. The stainless steel sutures are then inserted and tied, while the two tendons are held in contact. Tissue can often be overlapped about the midline. The retinaculæ if torn are repaired. The tourniquet is released and complete hæmostasis obtained (a hæmatoma under the prepatellar skin may produce skin gangrene). A Robert Jones' compression bandage is applied. The after-treatment differs little from that after meniscectomy though progress is slower. Full strong extension of the knee is aimed at. Gentle flexion of the knee is begun early, but is slower in returning than extension.

*Restoration of the Patella.* This has been attempted with circular sutures, and with sutures partially transfixing the bone. Neither are satisfactory, as the fragments tend to tilt on each other, and to separate, and therefore there is anxiety about allowing early exercises. The only satisfactory method of fixing the two halves in perfect apposition (after inspection of the margins of the fracture to be assured of a perfect fit) is to restore perfectly the visible outer surface of the bone and to fix the two

*Transverse Fractures.* Manipulate and obtain end-to-end apposition. An above-knee plaster is applied. Replaster when swelling has subsided (seven to fourteen days). About the eighth week a below-knee plaster is applied and knee exercises are begun. Weight-bearing is allowed about the tenth week. Remove plaster at the twelfth week, or, depending on level of fracture, begin weight-bearing in above-knee plaster about the tenth week. Remove plaster at the twelfth week. If open operation is indicated use a plate or intramedullary nail.

*Oblique, Partly Oblique, or Partly Transverse Fractures.* If stable, i.e. the



FIG. 663. The use of the Böhler traction frame in reduction of a fracture of both bones of the leg. The Hawley orthopaedic table may equally well be used; the knee is then extended and counter traction obtained by means of a perineal post.

ends are interlocked, or if the fibula is intact, treat as above. If unstable and inclined to shorten treat as below. If open operation is performed, single screws often open up unsuspected fissures and plating may then be needed.

*Spiral (Helical) Oblique and Comminuted Fractures.* Distraction is necessary to prevent shortening unless the fibula is sufficiently intact to prevent it. Continuous traction is obtained with a Kirschner wire in the calcaneus, and plaster is applied above or below the knee according to the level of the fracture (Fig. 663). Replaster about the third week, and if stable remove the wire, if unstable leave wire in place for a further seven to fourteen days. Weight-bearing is allowed in above-knee plaster about the tenth week; remove the plaster about the fourteenth week. If union is firm leave, if not firm, a below-knee plaster is applied for a further period.



## CHAPTER 103

### FRACTURES OF THE TIBIAL SHAFT AND THE FIBULA

Of the long bones of the body, the tibia is the most exposed to violence and the most frequently fractured. Although fractures of the tibia occur alone, the slender shaft of the fibula is commonly fractured as well. The effect of the fractured fibula on the stability of the limb depends on its level in relation to the tibial fracture. Fractures of the tibia are a good example of the influence of the shape of the fracture on the displacement and on the treatment to be chosen. *Direct Violence.*—The fracture occurs at the point struck, with risk of skin damage and a directly compound fracture. The fibula being broken at the same level does not help to stabilise the limb (Fig. 662). *Indirect Violence.*—

Excessive rotational strain on the tibial shaft produces a spiral fracture. If the strain is transmitted to the fibula it breaks in a similar way, but usually at the upper end. The fibular shaft then helps to stabilise the fracture, minimising shortening and giving some lateral support. If the body-weight continues to act, bending of the limb occurs at the site of fracture, and the sharp bone ends may be forced through the skin, producing an indirect compound fracture. If this occurs the fibular fracture may be of the bending type and again at another level.

Accurate reduction of a tibial fracture is important, for the joints at either end of the bone are otherwise subjected to strains which may eventually produce arthritis. Shortening must also be overcome, though up to  $\frac{1}{2}$  inch is not significant if alignment is good. In the growing child reasonable correction is all that is required, as growth compensates for minor discrepancies. In the young adult an attempt at perfect reduction should always be made, and with the methods now available this is usually possible. In the older patient the need for early exercises of the knee and ankle may encourage early operation.

The influence of the shape of the tibial fracture and the state of the fibula on treatment is as follows :



FIG. 662. Union beginning in a transverse fracture of both bones of the leg at the same level, due to direct violence.

plaster. Alternatively, an assistant applies traction to the pin. The patient is then returned to bed and the leg is supported and elevated on a Braun's frame or a Thomas's splint, and a pull of 7 to 10 lb. is applied. This may be reduced at the end of the second week to 5 lb.

Between the second and third weeks the limb is replastered, and minor corrections of angulation made. If there is sufficient union to prevent shortening, the pin may be removed after the plaster is applied. If the union is weak, the pin should be retained for a further period of traction and removed at the end of the fourth or fifth week. Angulation may be corrected by wedging. Avoid overtraction with consequent separation of the bone ends. This delays union and stretches joint ligaments.

**Fixed Distraction.** It is possible to place two transfixion pins in the leg, one in the heel and one in the tibial tuberosity, and to incorporate these in the plaster. Traction will not then be necessary, and strain is thereby taken off the knee joint. Such pins, however, tend to maintain separation of the bone ends, unless carefully watched and controlled. They do, however, provide excellent stability, and any tendency of the upper fragment to flex or extend may be controlled by two wires or pins through the bone.

**Open Operation.** In a straightforward fracture with intact skin, the risks of this operation are small, and the advantages gained, i.e. perfect reduction and early exercises of the knee and ankle, are very valuable. Advantage may be taken of the opportunity provided by a compound fracture to fix the bone ends under direct vision. Skin damage, comminution of the fracture, and threatened avascular necrosis may, however, make operation unwise. Closed methods should then be applied. The aims of open reduction are: (1) Perfect reduction; (2) firm fixation of the fracture with the minimum of metallic parts; (3) minimal disturbance of the soft tissues around the bone and of the skin. The method of fixation is adapted to the shape of the fracture. The most satisfactory fracture to fix is the helical fracture, which may be very firmly fixed by two screws, with minimal disturbance of the soft tissues. So sound is this procedure and so little are the risks involved, that in some fractures of the tibia alone, it is worth while. If the fracture is transverse or there is comminution a plate will be necessary to bridge the fracture. The plate should be applied on the lateral surface of the tibia under cover of the muscles—not on the subcutaneous surface. Four to six screws are needed. The slotted plate gives greater scope for varying the position of the screws, and enables the bone ends to be firmly approximated before the screws are tightened. The intramedullary Küntscher nail is inserted through a separate incision over the tibial tuberosity. It has the advantage of firm fixation and avoids the use of metal on the external surface of the bone. Furthermore, it may be used for multiple fractures, though for the reasons given below this may be inadvisable.

**Difficulties with Tibial Fractures.** *Delayed Union.*—This can often be

If open operation is undertaken, use two-screw fixation for helical fractures and a metal plate for comminuted fractures. Either type of fracture may be fixed with an intramedullary nail.

The above methods are primarily concerned with minimising shortening, though at open operation angulation and rotation can be corrected under direct vision. Rotation is corrected by aligning the midline of the patella and the midline of the second toe; minor variations cause little trouble. Angulation is much more important than rotation and is best controlled during treatment by wedging the plaster. This need not be done till the fracture is becoming firm, and can be left, if the position is fair, till the first replaster after swelling has subsided. An attempt may then be made to correct the deformity by manipulation, but if this is unsatisfactory, more accurate control is secured by wedging.

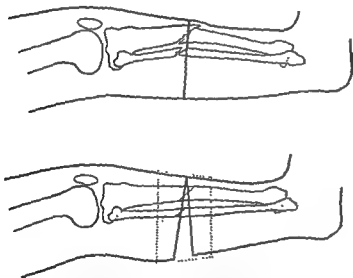


FIG. 664. The method of correcting an angular deformity of the leg bones by wedging the plaster.

**Wedging the Plaster.** An anæsthetic is usually unnecessary. The level of the fracture is marked on the plaster with the help of the radiographs. A circular cut is made through the plaster at this level with a hand saw (Fig. 664). It is quite easy to appreciate when the plaster has been cut through without the advice of the patient! The plaster is then prised open at the fracture site so that the two surfaces of the cut in the plaster subtend an angle equal to that of the deformity (Fig. 664). It is possible by prising open the plaster at the appropriate place in the circumference of the leg to correct the deformity in two directions at once. Check radiography follows. This simple method allows very accurate correction of small angles of deformity remaining after manipulative correction.

**Continuous Traction.** A Kirschner wire or Steinmann's pin is inserted through the calcaneus, at right angles to the mid-plane of the foot. The point of entry is  $\frac{1}{2}$  inch below and behind the fibular malleolus. The limb may then be steadied on a traction frame of the Böhler type (Fig. 663) or on a Hawley table, while the plaster is applied; the pin is included in the

*Popliteal Palsy.*—This may be caused by a badly designed below-knee plaster; pressure on the nerve occurs as it winds around the neck of the fibula. All plasters should be padded with felt or similar material around the fibula and the tibial tuberosity.

### FRACTURES AND DIASTASIS OF THE FIBULA

Fractures of the fibula are unimportant in themselves, as they do not affect the stability of the leg. They are important, however, as a guide to the mechanism of the injury which the leg or ankle has suffered. To have a clear picture of the ways in which the fibula may be fractured is to be more than halfway through the maze of ankle fractures.

Fractures of the fibula may occur from direct or indirect violence. Fractures by direct violence may occur at any level; they may be accompanied by open wounds and are of variable severity. Most commonly the fibula is fractured in one place, though double fractures are possible. Lying inside its musculo-fibrous envelope, the bone is seldom displaced, and therefore the treatment is that of a deep bruise, rest, cold compresses, then heat, massage and exercises to disperse the hæmatoma. Occasionally, a plaster is of value for a short time to allow full activity. Residual disability should be negligible.

Fractures produced by indirect violence are of much greater significance, as it is only possible to fracture the fibula by this means if the bone is partially or completely detached from its syndesmosis with the tibia. The type and level of the fracture is thus a guide to the ligamentous injury at the ankle, and it is as such that it must be regarded. If the lower end of the fibula is externally rotated to any degree the anterior tibiofibular ligament is torn or the lower end of the tibia is fractured. The torsion strain produces a spiral fracture of the fibula at its weakest part, the neck of the bone (*Maisonneuve's fracture*—Fig. 665). Stress may also be developed by angulation of the bone. If the tibiofibular syndesmosis is regarded as one fulcrum, and the fibular malleolus is pushed outwards, the fibular shaft is bowed towards the tibia, which then supports it and becomes another fulcrum. Then, if the force continues to act, the fibula is torn away from the tibia at the syndesmosis. Anterior and posterior tibiofibular ligaments and the interosseous ligament are torn. This condition is described as complete diastasis.



FIG. 665. Maisonneuve's fracture. A high fracture of the fibular neck, associated with rupture of the anterior tibiofibular ligament.

treated successfully with a series of weight-bearing plasters. Radiological evidence of union lags behind clinical evidence which should therefore be accepted if undue delay in walking is to be avoided. After open operation union is delayed by about two weeks, i.e. it takes fourteen weeks compared with the average of twelve weeks without operation. *Non-union.*—This may occur at the junction of the lowest and middle thirds of the bone in healthy adults. The reason is usually the wide stripping of the bone ends from their soft tissue attachments, producing a layer of avascular bone on the surface, and consequent atrophic non-union. Avascular necrosis and infection are other causes. Fix with a plate after freshening the fracture or, if there has been bone loss, use a graft. *Double Fractures.*—These result from motor-cycle accidents. The upper fracture being near the nutrient artery, and, involving more cancellous bone, usually heals well. The lower fracture, as a result of complete or partial avascular necrosis, unites very slowly or not at all. If after a reasonable time, three to four months, there is radiological evidence of absolute non-union, the fracture site should be packed with cancellous bone chips, after opening up the medullary cavity and then fixed with a plate. *Comminuted Fractures.*—There may be little delay in union with a few small loose fragments, but when fragments are large or multiple, as in a double fracture, delay in union will occur. This is due to disturbance of the blood supply, which must not be further jeopardised by unnecessary open operative procedures. Occasionally, however, operation may be necessary because of the malposition of a fragment, but it should be limited to the minimum possible disturbance of tissues. Thus in a double fracture it is usually advisable only to fix one of the fractures and to treat the other conservatively. A loose fragment may tilt so that it presses on the skin and it may ulcerate through the skin if a tight plaster is applied over it. This justifies removal of such a fragment. *Skin Loss.*—The essential principle is to get the bone enclosed in an envelope of living tissue. Part of this envelope may be constituted with viable muscle, and skin may be grafted over this surface, either immediately or within a day or two before infection can occur. To facilitate this procedure it may be advisable to fix the fracture with a plate or intramedullary nail, so that the plastic operation can be carried out on a stable base. Rotation flaps are unsatisfactory except in the upper third of the limb because of the curvature and decreasing diameter of the leg. A long tension-relieving incision may be made over the calf and a wide strip of skin slid over the tibia till it overlies the anterior muscular compartment to close a small defect. The resultant gap over the calf may be covered with Thiersch grafts. With large areas of skin loss the only safe method of obtaining skin cover is a flap of skin from the opposite leg. Even in the absence of skin loss, extensive swelling in a crush injury may make it impossible to close a wound; the same treatment is then required as for skin loss. Tension is often a greater enemy of skin survival than is infection. Haemostasis or drainage is therefore important. *External*

will break above the syndesmosis, about 1½ inches above the tip (*low Dupuytren's fracture*—Fig. 666). The fibular shaft is held in place in this injury by the intact interosseous ligament.

When the foot is examined it is found that on internal rotation the foot tends to pass into inversion, and it is this movement which, if occurring with sufficient force, produces the common accident of "going over on the ankle," i.e. a sprain or tear of the fibular collateral ligament of the ankle. If the foot is externally rotated it tends to become everted and becomes a rigid lever; it is this propensity which accounts for the fact that external rotation fractures are commonly recorded, whereas internal rotation fractures are not. Force is transmitted to the fibula by the pressure of the talus on the anterior margin of the fibular malleolus, and by the pull posteriorly of the posterior talofibular ligament. This results in a torsion strain developing in the shaft of the bone, with the results described above.

Fractures of the fibula may be summarised as follows:

**Fractures of the Upper End of the Fibula.** (a) *The Tip*.—Avulsion by the lateral collateral ligament of the knee, a sign of rupture of lateral supports of the knee. (b) *A Larger Section of the Tip*.—Avulsion by the fibular collateral ligament and the insertion of the biceps. Again a clue to damage to the knee. (c) *The Head*.—This may be crushed (direct injury) in association with depression of the tibial plateau. (d) *The Neck*.—A spiral fracture due to torsion of the shaft, and indicating a rupture of the anterior tibiofibular ligament, Maisonneuve's fracture (Fig. 666), i.e. partial diastasis.

**Fractures of the Shaft of the Fibula.** (a) High fracture, at the junction of the superior and middle thirds. This indicates complete diastasis, and is due to an abduction or external rotation force. (b) Helical fracture at the junction of the inferior and middle thirds. This is due to abduction rotation, and indicates an intact posterior tibiofibular ligament, but rupture of the anterior tibiofibular ligament and part of the interosseous ligament, i.e. partial diastasis. This is known as the high Dupuytren's fracture (Fig. 666).

**Fractures of the Lower End of the Fibula.** (a) Fracture above the syndesmosis. This is due to abduction of the malleolus and rupture of the lower parts of the anterior and posterior tibiofibular ligaments (*low Dupuytren's fracture*—Fig. 666). (b) Fracture just above the level of the tibial articular surface, produced by abduction. The ligaments are intact. (c) Fracture just below the articular surface, produced by avulsion by adduction of the talus. (d) Fracture of the tip. Avulsion by the calcaneofibular ligament, in inversion sprains. (e) Avulsion of the bony insertions of the anterior and posterior tibiofibular ligaments may replace ruptures of the ligaments in diastasis of the syndesmosis.

**Diastasis of the Fibula.** It will be seen that fractures of the fibula are closely related to separation of the tibiofibular syndesmosis (diastasis),

It is impossible to push the fibular malleolus medially and bow the fibula outwards. Excessive force of this nature inevitably results in an adduction fracture of the fibular malleolus at the level of the tibial articular surface. It is, however, possible to externally rotate the foot, so that the talus presses on the anterior aspect of the fibula, and the rotation may become more marked if the fibula is externally rotated and pushed backwards as the result of rupture of the anterior tibiofibular ligament. If this occurs, continuation of the force may rupture the

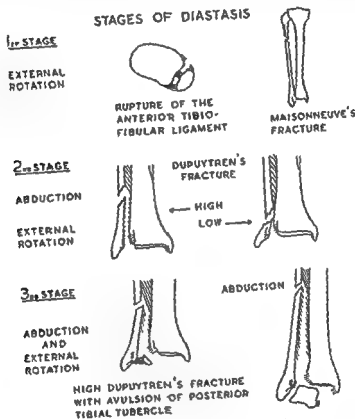


FIG. 666. The stages of diastasis.

posterior tibiofibular ligament, with complete diastasis and a fracture of the fibula high in the shaft. Alternatively, if there is more rotation than abduction in the force applied to the malleolus, the posterior tibial tubercle may become the fulcrum around which the fibula is rotated and the shaft of the fibula is then bowed forward and fractured in a spiral manner at the junction of the inferior and middle thirds of the bone (*high Dupuytren's fracture*—Fig. 666).

If the force applied to the fibular malleolus is one of pure abduction (i.e. pressure outwards on the articular surface) the resultant fracture depends on the level of tearing of the anterior and posterior tibiofibular ligaments. If the lower parts hold, the malleolus is broken off just about the level of the tibial articular surface. If the lower parts tear, the fibula

## CHAPTER 104

### FRACTURES AND FRACTURE-DISLOCATIONS OF THE ANKLE

INJURIES of the ankle present a complicated picture owing to the large number of variants which may be encountered. These variants are, however, capable of fairly simple analysis, as are the principles of treatment. The multiplicity of lesions is based on combinations of (1) Rupture of ligaments (alternatively avulsion fractures). (2) Fractures of the malleoli; the posterior tibial overhang is conveniently regarded as the "third malleolus." (3) Diastasis of the tibiofibular syndesmosis. (4) Dislocation of the talus. (5) Fractures of the articular surface of the tibia, or rarely the talus.

Radiologically these injuries may be grouped into: (a) Fractures of one malleolus, with little loss of stability; (b) fractures of both malleoli, with complete loss of lateral stability; and (c) fractures of both malleoli, and of the posterior tibial overhang ("third malleolus"), producing complete instability of the ankle. This classification, however, fails to take note of ligamentous ruptures which may replace fractures, and therefore is not a good guide to the full range of ankle injuries. However, the radiological findings should be regarded as a pattern from which ligamentous injury can be deduced, and not until ligamentous injury has been carefully considered is a full understanding of the injury possible. It is much better to approach the subject of ankle injuries by consideration of the deforming force, and to follow the sequence of injuries which may occur if the force continues to act till complete instability of the ankle is produced. The deforming forces of indirect injury may be analysed as follows: (1) External rotation; (2) abduction (fibular flexion); (3) adduction (tibial flexion); (4) compression (by body-weight or body-weight plus acceleration).

Let us examine the sequence of events in forced external rotation, the simplest and most common force acting on the ankle, and responsible for 70 per cent. of all ankle injuries. Strain falls on the fibular malleolus, chiefly from pressure on its anterior margin by the talus; thus the anterior tibiofibular ligament is stretched. If this ligament yields, the torque developed in the fibula may result in the high fracture of the fibular neck, Maisonneuve's fracture (Fig. 665). More commonly, the ligament holds, and a line of stress develops between the anterior and posterior tibiofibular ligaments, resulting in a fracture which runs from above downwards and forwards between the ligaments, and which enters the ankle joint anteriorly just below the attachment of the anterior tibiofibular ligament.



and that there are three stages of diastasis (Fig. 666). *First Stage.*—Rupture of the anterior tibiofibular ligament (partial diastasis). High fracture of the fibular neck by torsion (Maisonneuve's fracture). *Second Stage.*—Rupture of the anterior tibiofibular ligament and the interosseous ligament. Helical fracture of the fibula  $2\frac{1}{2}$  inches above the malleolus (high Dupuytren's fracture). *Third Stage.*—Rupture of all three ligaments (complete diastasis). High fracture of the fibular shaft.

The rupture of the lower fibres of the tibiofibular ligaments, associated with the low Dupuytren's fracture, can scarcely be described as a diastasis, as the shaft of the fibula still remains attached to the tibia.

Diastasis is important as it seriously affects the stability of the ankle. Failure to recognise it may result in the patient being allowed to bear weight too soon, and an abduction deformity of the ankle may develop. Although it is a complicating factor in regard to the stability of the ankle, as the articular surface of the ankle is not involved, it does not seriously affect the prognosis if the reduction is perfect and retention adequate. It may be an added reason for operative treatment in order to obtain a perfectly reduced and stable ankle.

*Recognition of Diastasis.* This may be obvious, as when the talus is displaced upwards between the tibia and fibula, or it may be extremely difficult. Normally, the shadow of the anterior tibial tubercle lies across the shadow of the fibular malleolus to a depth equal to one-third of the width of the fibula. Although this overlap is not greatly affected by rotation of the central X-ray in taking a film, it may be considerably affected by the depth of the tibial groove which varies greatly from patient to patient. If the groove is shallow, the central ray may be able to pass completely through it, and give the impression that diastasis has occurred. Diastasis may be suspected because of : (1) The fracture associated with it, e.g. Maisonneuve's fracture, the high fracture of the fibula, high and low Dupuytren's fracture. (2) Increased medial space (the gap between the medial side of the talus and the inner aspect of the medial malleolus). (3) Decreased overlap of the shadow of the anterior tibial tubercle on the fibula. (4) Avulsion fractures around the syndesmosis, indicating rupture of ligaments.

Proof is obtained by : (1) Examination of the mobility of the fibular malleolus compared with the opposite side. (2) Strain radiography of the ankle. External rotation or abduction may open up the syndesmosis; this requires general anaesthesia. (3) Comparable radiographs of both ankles in 30 degrees of internal rotation so that the shadow overlap of the tibial tubercle may be accurately measured on the two sides. It must never be forgotten that spontaneous reposition of a diastasis may mask its radiological appearance.

almost equally stressed. They may yield in their lower fibres, and angulation of the malleolus then produces a fracture of the fibula above the attachments of the ligaments, but the interosseous ligament holds the fibula shaft in good position (low Dupuytren's fracture—Fig. 666). If both ligaments and the interosseous ligament fail together, the fibula is pushed far more widely from the tibia, and the fracture of the fibula occurs high up, at the junction of the superior and middle thirds (high fibula fracture—Fig. 666). The talus is carried with the fibula and may be pushed up between it and the tibia, i.e. a complete diastasis and a third degree injury (Fig. 666). For this force to be exerted on the syndesmosis, rupture of the deltoid ligament or fracture of the medial malleolus is necessary.

The mechanism of adduction injuries is much simpler. Pressure is brought to bear on the inner aspect of the medial malleolus, and a fissure fracture occurs almost vertically; the malleolus is sometimes displaced a little upwards. Alternatively, if the component of rotation around the long axis of the foot is the dominant force, the medial malleolus may escape, and an avulsion of the fibular malleolus, *below the tibial articular surface*, occurs. A fracture of either malleolus may thus represent a first degree adduction injury.

If the adduction force continues to act after fracture of the medial malleolus, the fibular collateral ligament is stretched, and it either ruptures, or it avulses the fibular malleolus. Either is equivalent to a second degree injury. It is uncommon to get further injury to the ankle by adduction violence alone, but if combined with compression, a fracture of the posterior margin of the tibia is possible, with dislocation of the talus—a third degree lesion.

*Compression force* may act alone, producing a series of injuries to the lower end of the tibia analogous with the fractures of the lower end of the humerus. Thus there may occur an anterior marginal fracture, a posterior marginal fracture, T- or Y-shaped fractures, or severe comminution of the articular surfaces. Compression may be combined with the forces already described, and produce posterior marginal fractures with dislocation. Rotation of the cylindrical talus in the curve of the tibia tends to split the lower end of the tibia if the talus cannot escape, and this is in part responsible for the most serious posterior marginal fractures.

In the high Dupuytren fracture, the deformation ceases after fracture of the fibula and the fibula remains attached to the tibia by means of the posterior tibiofibular ligament, a fact which can easily be appreciated by manipulation of the malleolus under anæsthesia. Occasionally the posterior tibial tubercle is fractured, and rarely, if the force has continued to act, the posterior tibiofibular ligament is torn, permitting complete diastasis, and dislocation of the talus, a third degree lesion.

**Fractures of the Medial Malleolus.** The shape of the fracture is often a very good guide to the nature of the main deforming force. The perfect

This is the *first degree external rotation fracture* of the ankle which may occur with minimal displacement of the ankle (Fig. 667). If after this has occurred the force continues to act, strain is first applied to the antero-lateral part of the anterior aspect of the capsule of the joint, which may tear, and then on the anterior (talotibial) fibres of the deltoid ligament. This ligament may tear, or alternatively it may avulse the anterior aspect of the medial malleolus. Thus a bimalleolar fracture is produced, conveniently called a *second degree external rotation fracture*. The loss of



FIG. 667. A lateral view of a first degree external rotation fracture of the ankle.

stability from fracture of the medial malleolus or rupture of the anterior part of the deltoid ligament is just as great and, from the point of view of treatment, is a second degree lesion.

With the loss of the medial ligamentous support, the foot swings more widely laterally, carrying with it the fibular malleolus below the fracture. Strain is next applied to the posterior tibiofibular ligament, and its attachment to the tibia (the posterior tibial tubercle) may be avulsed, and the talus dislocated posterolaterally. This is the unstable *third degree ankle injury*. On reduction, third degree injuries may be stable or unstable (*vide infra*).

In abduction and adduction injuries there are two components in the acting force. The talus may be pushed to one or other side directly under the tibia, like a drawer, or the talus may be rotated around the anteroposterior axis of the foot, tightening one or other col-

lateral ligament, with a tendency to produce an avulsion fracture of a malleolus. The pattern of the fracture produced varies according to which component of the force is dominant. Abduction has to be considered apart from adduction, for abduction may produce diastasis, which is impossible with adduction.

In abduction, pressure is applied to the inner aspect of the fibular malleolus. If the tibiofibular ligaments hold, the malleolus breaks just above the tibial articular surface. Strain then falls on the deltoid ligament which may rupture, or the medial malleolus may be avulsed. This is a second degree lesion. If the force continues the talus may be dislocated from the articular surface of the tibia, carrying the two malleoli with it, a third degree lesion.

In abduction, both the anterior and posterior tibiofibular ligaments are

articular surface is scarcely encroached on, and the fibrous sheath around the lower end of the tibia is seldom completely torn; the fragment therefore preserves some ligamentous attachments, which allow it to be easily reduced and retained (Fig. 669).

*True Posterior Marginal Fracture.* This is due to compression, or to rotation of the talus pressed in the curve of the tibial articular surface. The fracture lies a variable distance from the posterior margin (Fig. 669). If it leaves sufficient depth of tibial articular surface, the talus will stay in position after reduction. If there is not sufficient overhang the reduction is unstable, and special care must be taken to retain the talus in position. A similar instability may be met with in anterior marginal fractures. This fracture is important because of the damage to the articular surface and the difficulties in retention. The involvement of the articular surface usually leads to traumatic arthritis of the joint.

FIG. 669. Fractures of the posterior margin of the tibia (lateral view). The small avulsion fracture of the posterior tibial tubercle does not affect the articular surface. The true posterior marginal fracture enters the articular surface and may or may not leave sufficient overhang of the tibia to make reduction stable.



Unstable lesions may be retained in position by: (1) Open operation through a posterolateral approach. There is a danger of avascular necrosis of the fragment if the joint is widely opened, and reduction of the fragment has to be determined by repositioning the visible portion of the fragment and X-ray control. It is seldom perfect. (2) Insertion of a pin through the calcaneus and incorporation of the pin in a below-knee plaster. The pin is pulled forwards, and the tibia is pulled backwards by a sling as the plaster is applied; thus the talus is pulled towards the anterior aspect of the joint. This is the safest method for inexperienced hands. The pulls are reversed for anterior marginal fractures.

*Diastasis of the Tibiofibular Syndesmosis.* The mechanism has been described under fractures of the fibula (Chap. 103). Reduction is as a rule straightforward, especially if there is a length of fibular shaft to stabilise the fracture, but, because of the soft tissue swelling and extensive soft tissue injury, it may not be perfect at the first attempt. Further reduction when the swelling has subsided may produce considerable improvement. Often such an injury is accompanied by a fracture of the medial malleolus, and if reduction is imperfect and the medial malleolus is displaced, reduction by open operation and fixation of the medial malleolus with a single screw is advisable. This has the effect of pulling the talus back into position together with the fibular collateral ligament and the fibular malleolus.

reduction and fixation of the medial malleolus is often the best method of reduction and retention of the more complicated ankle injuries. The vertical fracture caused by adduction is the most distinctive (Fig. 668C). It is little displaced as a rule; it offers a wide bony surface of contact, and it is rarely followed by any reparative difficulty. Fracture by abduction is due to avulsion and the fracture tends to be at right angles to the line of pull, i.e. across the base of the malleolus (Fig. 668A). In fracture by external rotation the pull is on the anterior fibres of the deltoid ligament, and the fracture, being at right angles to this pull, is much more angulated in the lateral view, and often has a spiral shape in the anteroposterior view (Fig. 668B). It can usually be distinguished from the abduction injury.

In abduction and adduction injuries the force may continue to act and

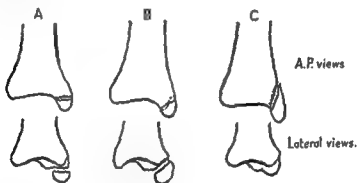


FIG. 668. Fractures of the medial malleolus due to abduction A, external rotation B, and adduction C. Note the characteristic shapes in the A.P. and lateral views.

pull the malleolus away from the tibia. On its spontaneous return, as a result of muscular action on the foot, soft tissues may be caught between the two surfaces. Alternatively, as the result of swelling, the tight fascia around the ankle may be dragged in between the two fracture surfaces. In either case, the tendency is to displace the malleolus and to cause non-union. Fibrous union of the malleolus may be satisfactory in the elderly, but it is not satisfactory in the young adult; therefore, if satisfactory reposition of the medial malleolus is not obtained, open fixation with a single screw is advisable. This procedure is often indicated for other reasons when reduction fails.

**Fractures of the "Third Malleolus."** Two types of fracture of the posterior aspect of the tibia are recognised, and the distinction is important in regard to both treatment and prognosis. The posterior aspect of the tibia carries the posterior tibial tubercle which is largely responsible for the formation of the tibial gutter in which the fibula lies.

**Fracture of the Posterior Tibial Tubercle.** This may be due to avulsion by the posterior tibiofibular ligament, or to pressure on the tubercle in rotation-abduction fractures such as the high Dupuytren's fracture. The

articular surface is scarcely encroached on, and the fibrous sheath around the lower end of the tibia is seldom completely torn; the fragment therefore preserves some ligamentous attachments, which allow it to be easily reduced and retained (Fig. 669).

*True Posterior Marginal Fracture.* This is due to compression, or to rotation of the talus pressed in the curve of the tibial articular surface. The fracture lies a variable distance from the posterior margin (Fig. 669). If it leaves sufficient depth of tibial articular surface, the talus will stay in position after reduction. If there is not sufficient overhang the reduction is unstable, and special care must be taken to retain the talus in position. A similar instability may be met with in anterior marginal fractures. This fracture is important because of the damage to the articular surface and the difficulties in retention. The involvement of the articular surface usually leads to traumatic arthritis of the joint.

FIG. 669. Fractures of the posterior margin of the tibia (lateral view). The small avulsion fracture of the posterior tibial tubercle does not affect the articular surface. The true posterior marginal fracture enters the articular surface and may or may not leave sufficient overhang of the tibia to make reduction stable.



Unstable lesions may be retained in position by : (1) Open operation through a posterolateral approach. There is a danger of avascular necrosis of the fragment if the joint is widely opened, and reduction of the fragment has to be determined by repositioning the visible portion of the fragment and X-ray control. It is seldom perfect. (2) Insertion of a pin through the calcaneus and incorporation of the pin in a below-knee plaster. The pin is pulled forwards, and the tibia is pulled backwards by a sling as the plaster is applied; thus the talus is pulled towards the anterior aspect of the joint. This is the safest method for inexperienced hands. The pulls are reversed for anterior marginal fractures.

*Diastasis of the Tibiofibular Syndesmosis.* The mechanism has been described under fractures of the fibula (Chap. 103). Reduction is as a rule straightforward, especially if there is a length of fibular shaft to stabilise the fracture, but, because of the soft tissue swelling and extensive soft tissue injury, it may not be perfect at the first attempt. Further reduction when the swelling has subsided may produce considerable improvement. Often such an injury is accompanied by a fracture of the medial malleolus, and if reduction is imperfect and the medial malleolus is displaced, reduction by open operation and fixation of the medial malleolus with a single screw is advisable. This has the effect of pulling the talus back into position together with the fibular collateral ligament and the fibular malleolus.

Imperfect reduction of diastasis may be due to interlocking of a fibular fracture, interposition of bony fragments or soft tissue in the gap, or instability of the fibular malleolus in the low Dupuytren's fracture (Fig. 666). This may demand open fixation of the fibular fracture or placing a screw across the syndesmosis to stabilise the malleolus. The screw must be placed obliquely, and must be removed before weight-bearing is begun, as some tibiofibular movement is essential to the elasticity of the joint.

**Ligamentous Injury.** This may replace avulsion fractures, and conversely the presence of a fracture may indicate ligamentous injury elsewhere. The deltoid ligament, being a thick sheet of tissue, is rarely completely torn, but if it is torn it is detached from the surface of the medial malleolus, and on reduction of the displacement it is returned to good position and requires no special treatment, other than retention of the ankle in its normal position by plaster.

The fibular collateral ligament, however, being composed of three parts, is less stable and more susceptible to injury. The common accident of "going over on the foot," the inversion sprain, often tears the anterior tibiofibular ligament, which is weak and unimportant. At the same time the tibiofibular mortice is spread by rotation of the talus, and there is a sprain of the anterior tibiofibular ligament. This is often more painful

than rupture of the ligament, and because the ligament can be pressed against bone the injury is easy to detect. A more severe inversion force may rupture both the anterior tibiofibular ligament and the calcaneofibular ligament. The latter ligament is the most important in stabilising the talus, and can be palpated as it runs from the fibular malleolus to the calcaneus. Its rupture permits rotation of the talus in the mortice up to 30 degrees (Fig. 670). This can be detected by strain radiography. This consists of giving the patient a general anaesthetic, and X-raying the ankle during forced inversion (Fig. 670). It must never be forgotten that clinical signs of fracture unaccompanied by radiological evidence of



FIG. 670. Complete rupture of the fibular collateral ligament.

fracture indicate severe soft tissue injury—probably ligamentous—and that such an injury must be sought for by further clinical or radiological examination.

Ruptures of the calcaneofibular ligament and the anterolateral capsule of the ankle permitting rotation of the talus over 15 degrees should be repaired by open operation, as the ends of the torn ligaments may be widely separated by tendons and other soft tissue. In some cases the ends are found in good position, but this cannot be determined till open operation is undertaken. Tilting of the talus over 30 degrees indicates wider tearing of the capsule of the ankle, and not rupture of the posterior talofibular ligament, which is very seldom torn. Repair of the capsule and calcaneofibular ligament is necessary if an unstable ankle is to be avoided.

**Summary of External Rotation Ankle Injuries.** *A. Fractures by External Rotation.* First degree.—Interligamentous fracture of the fibular malleolus (Fig. 667). Second degree.—Interligamentous fracture associated with: Fracture of the medial malleolus; rupture of the deltoid ligament. Third degree.—A second degree injury complicated by posterior dislocation of the talus or a posterior marginal fracture. *B. Fractures Associated with the Stages of Diastasis* (Fig. 666).—First stage.—Rupture of the anterior tibiofibular ligament, and high fracture of the fibular neck (Maisonneuve's fracture). Second stage.—Rupture of the anterior tibiofibular ligament and interosseous membrane, with a high Dupuytren's fracture (Fig. 666). Third stage.—Complete diastasis with a high Dupuytren's fracture and posterior dislocation of the talus.

**Treatment of External Rotation Injuries.** *The First Degree External Rotation Fracture.*—This may be encountered with varying degrees of soft tissue injury stopping short of injury to the deltoid ligament or the medial malleolus. The treatment should be adapted to the severity of the injury and other circumstances. Thus there may be: (a) A subperiosteal fracture with no displacement; (b) a fracture with moderate displacement; (c) a fracture with displacement and much bruising around the ankle. Treatment is chosen from: (a) Elevation, a pressure bandage, early massage and exercises and rest in bed, or ambulation on crutches. Weight-bearing may be begun at the end of the second week. The advantages of this treatment are rapid recovery in a month, but it entails bed rest for a week or more. (b) Local injection with procaine. This may permit immediate use of the ankle in an emergency. It facilitates the resorption of a hæmatoma and can be used in conjunction with (a). (c) Application of a light below-knee plaster. This has the advantage that the patient can walk on the plaster with a plaster overshoe as soon as the plaster is dry. This is almost the only example of a plaster which does not lose its effectiveness as soon as the swelling has subsided. It may be worn for two to three weeks and then removed. The foot is stiff after this and requires some physiotherapy for a further two to three



weeks before full recovery. The foot swells after removal of the plaster and a pressure bandage must be worn for a similar time afterwards. The total disability time is five to seven weeks, but patients are active in plaster after the first few days. There should be no sequelæ to this injury; complete recovery is the rule. The treatment of a Maisonneuve's fracture, i.e. first stage diastasis is similar.

*Second Degree External Rotation Injury.* The stability of the ankle is destroyed by rupture of the attachment of the deltoid ligament, or fracture of the medial malleolus; and the fibular malleolus can be displaced laterally. Usually there is sufficient ligamentous adhesion to the malleoli for manual reduction to be effective. Treatment therefore consists of manual reduction and application of plaster of Paris in the first instance. A check radiograph is then taken. For the reasons previously given, reduction of the medial malleolus may not be perfect. Open operation and screwing of the malleolus is therefore sometimes needed in young subjects for perfect reduction. The plaster is renewed when the swelling has subsided at the end of three weeks, and the opportunity taken to remove the stitches if an open reduction has been carried out. A close-fitting plaster below the knee, with padding to protect the external popliteal nerve, is then applied. Activity on crutches without weight-bearing is then permitted; and about the sixth to the eighth week weight-bearing with an overshoe is allowed. The plaster is removed about the tenth to the twelfth week, and followed by a pressure bandage to control swelling, and a period of physiotherapy to restore ankle movements. Similar treatment is required for the high Dupuytren's fracture, though weight-bearing may have to be delayed for a fortnight.

*Third Degree External Rotation Injuries.* Posterior dislocation of the talus is permitted by rupture of the posterior tibiofibular ligament which attaches the malleolus to the tibia or by a posterior marginal fracture. When the ligament is ruptured the treatment is similar to that for a second degree injury, but weight-bearing is delayed. When there is a posterior marginal fracture, the damage to the articular surface demands a long period of non-weight-bearing exercises, even if there is considerable tibial overhang. There is little chance of avoiding post-traumatic arthritis even if effective repositioning of the posterior fragment is achieved.

**Summary of Abduction Ankle Injuries (Fig. 671).** First degree.—Fracture of the medial malleolus or rupture of the deltoid ligament. Second degree.—Fracture of both malleoli. Third degree.—Fracture of both malleoli, together with posterior dislocation or posterior marginal fracture. Associated with diastasis there may be: (a) A low Dupuytren's fracture due to tearing of the lower fibres of the tibiofibular ligaments and a consequent partial diastasis (Fig. 666). (b) A high fracture of the fibular shaft with complete diastasis, and sometimes dislocation of the talus between the bones (Fig. 666).

**Treatment of Abduction Injuries.** First degree.—Manipulative reduction is often sufficient, but for the reasons already given it may be unsatisfactory, and to avoid risk of non-union a single screw may be inserted. Further treatment is similar to that of a first degree external rotation injury. Second degree.—Treatment of these injuries is comparable to that of second degree external rotation injuries, though when there is partial diastasis, as in the low Dupuytren fracture, reposition of the fibular malleolus may not be perfect even after fixation of the medial malleolus by a screw; therefore a screw in the fibular malleolus may also be needed. Third degree.—Treatment of these injuries is comparable to that of third degree external rotation fractures. It has already been mentioned that complete diastasis is no bar to good recovery, and merely requires a little longer freedom from weight-bearing after good reduction.

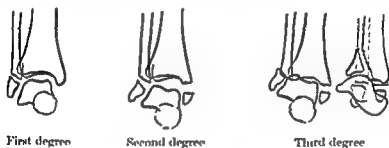


FIG. 671. Diagram of first, second and third degree abduction fractures of the ankle.

There is likelihood of post-traumatic arthritis developing after a posterior marginal fracture.

**Summary of Adduction Ankle Injuries.** First degree.—Fracture of the fibular malleolus below the tibial articular surface, or a vertical fracture of the medial malleolus (Fig. 668C). Second degree.—Both the above fractures together (bimalleolar fracture). Third degree.—A second degree injury accompanied by a posterior marginal fracture or dislocation (Fig. 669). Diastasis does not occur with adduction injuries.

**Treatment of Adduction Injuries.** Treatment is closely comparable with that of the similar stages of external rotation injuries. Non-union of the medial malleolus does not occur; therefore open operation on it is almost never required.

**Compression Fractures.** The treatment has already been partly discussed under posterior marginal fractures. Anterior marginal fractures with forward subluxation of the talus may cause similar difficulties. If there is no tibial overhang to stabilise the talus on reduction, a wire in the calcaneus may be incorporated in the plaster for retention. Retention is obtained by means of a plaster cast incorporating the calcaneal wire or pin. In grossly comminuted or T- or Y-shaped fractures, treatment by continuous traction in plaster achieves the best results. Open operation

weeks before full recovery. The foot swells after removal of the plaster and a pressure bandage must be worn for a similar time afterwards. The total disability time is five to seven weeks, but patients are active in plaster after the first few days. There should be no sequelæ to this injury; complete recovery is the rule. The treatment of a Maisonneuve's fracture, i.e. first stage diastasis is similar.

*Second Degree External Rotation Injury.* The stability of the ankle is destroyed by rupture of the attachment of the deltoid ligament, or fracture of the medial malleolus; and the fibular malleolus can be displaced laterally. Usually there is sufficient ligamentous adhesion to the malleoli for manual reduction to be effective. Treatment therefore consists of manual reduction and application of plaster of Paris in the first instance. A check radiograph is then taken. For the reasons previously given, reduction of the medial malleolus may not be perfect. Open operation and screwing of the malleolus is therefore sometimes needed in young subjects for perfect reduction. The plaster is renewed when the swelling has subsided at the end of three weeks, and the opportunity taken to remove the stitches if an open reduction has been carried out. A close-fitting plaster below the knee, with padding to protect the external popliteal nerve, is then applied. Activity on crutches without weight-bearing is then permitted; and about the sixth to the eighth week weight-bearing with an overshoe is allowed. The plaster is removed about the tenth to the twelfth week, and followed by a pressure bandage to control swelling, and a period of physiotherapy to restore ankle movements. Similar treatment is required for the high Dupuytren's fracture, though weight-bearing may have to be delayed for a fortnight.

*Third Degree External Rotation Injuries.* Posterior dislocation of the talus is permitted by rupture of the posterior tibiofibular ligament which attaches the malleolus to the tibia or by a posterior marginal fracture. When the ligament is ruptured the treatment is similar to that for a second degree injury, but weight-bearing is delayed. When there is a posterior marginal fracture, the damage to the articular surface demands a long period of non-weight-bearing exercises, even if there is considerable tibial overhang. There is little chance of avoiding post-traumatic arthritis even if effective repositioning of the posterior fragment is achieved.

**Summary of Abduction Ankle Injuries (Fig. 671).** First degree.—Fracture of the medial malleolus or rupture of the deltoid ligament. Second degree.—Fracture of both malleoli. Third degree.—Fracture of both malleoli, together with posterior dislocation or posterior marginal fracture. Associated with diastasis there may be: (a) A low Dupuytren's fracture due to tearing of the lower fibres of the tibiofibular ligaments and a consequent partial diastasis (Fig. 666). (b) A high fracture of the fibular shaft with complete diastasis, and sometimes dislocation of the talus between the bones (Fig. 666).

## CHAPTER 105

# FRACTURES OF THE TARSUS, METATARSALS AND PHALANGES

### FRACTURES OF THE TARSUS

FRACTURES of the tarsus are frequently associated with other fractures around the ankle, fractures of the metatarsals, and dislocation of the talus. The dislocations of the talus have been briefly outlined before (Chap. 104); taken in conjunction with fractures a great variety of injuries may be encountered.

**Fracture of the Neck of the Talus.** This fracture may permit the body of the talus to dislocate, leaving the talonavicular joint intact; thus a double dislocation of the ankle and subtaloid joints occurs (Fig. 672). When the talonavicular joint is also involved there is a triple dislocation. Fracture of the neck of the talus is due to excessive dorsiflexion, and may be accompanied by damage to the anterior margin of the tibia. Fissure fractures of the neck of the talus, without displacement, need immobilisation in the normal plantigrade position in plaster for eight weeks without weight-bearing. After this time, weight-bearing may be permitted and the plaster removed a fortnight later.

If the fracture is accompanied by displacement, care must be taken in reduction. There is usually subluxation of the posterior subtaloid joint, and possibly impaction of the fracture. This must be displaced by forcible plantar flexion, and the foot immobilised in plantar flexion. Dorsiflexion will almost certainly redisplace the fracture and the use of the plantar flexed position is important for success. Immobilisation in plaster is



FIG. 672. A fracture of the neck of the talus with displacement.

is technically difficult and may produce avascular necrosis. Badly damaged joints eventually come to arthrodesis, which may be performed early to save time in convalescence.

**Epiphyseal Injuries.** Up to the age of sixteen years, the tibial epiphysis may be displaced. It may be pushed off the end of the tibia in any direction, carrying with it a fragment of the tibial diaphysis on the side to which it is pushed. The fibular malleolus may or may not have an accompanying epiphyseal displacement. Reduction is easily accomplished by pressure in the opposite direction to the displacement; a padded wedge placed below the tibia is a great help in developing the necessary force. Very rarely, when the epiphyseal plate is badly pulped by a fracture crossing it, premature union of a fragment occurs and there is consequent interference with growth.

and may therefore be treated by elevation and immediate active exercises and massage. If weight-bearing is likely to produce displacement it is delayed. (1) "*Beak*" *Fractura*.—A small triangle of bone is elevated from the dorsum of the calcaneus under the tendo Achilles. The mechanism is unknown. It should be removed. (2) Fracture of the medial tuberosity. (2) Fracture of the sustentaculum tali. (4) Fissure fractures without displacement.

Fractures which damage the subtaloid joint may be divided into two main varieties: (a) Severe crush fractures, involving complete fracture of the narrowest part of the bone, and multiple fissures; this results in flattening and broadening of the bone. The deformity may be best seen in an oblique view of the calcaneus, taken with the foot in dorsiflexion and the X-ray tube at 45 degrees to the plate placed under the back of the heel. (b) Fractures of the posterolateral side of the bone involving the outer two-thirds of the posterior articular facet. This particular fracture lends itself to reduction by means of a spike placed in the posterior aspect of the bone, or by open operation; the result is often good radiologically, but poor functionally.

The function of the foot is always gravely interfered with by these fractures. Successful treatment implies mobility and freedom from pain. Mobility is best preserved by elevation, pressure bandaging, massage and early exercises. Weight-bearing is permitted when the patient can put the heel to the ground without pain. Activity on crutches can begin at an early stage. The possible results, judged on the basis of pain which is usually due to subtaloid arthritis, are: (a) Damage to the subtaloid joint permitting return of some function without pain. (b) Damage to the joint producing subtaloid arthritis. If this is painful, arthrodesis will be required (about 10 per cent. of cases). (c) Damage so severe that the subtaloid joint fuses. "Arthrodesis" has been effected spontaneously and pain is slight.

The disability period after a crushing injury to the calcaneus is long. A manual labourer may be unable to continue in his employment. The sedentary worker is able to return to duty early on crutches and continues with his job after recovery. Patients complain of pain on walking on rough ground, or on any irregular surface demanding co-ordination of pronation and supination with plantar and dorsiflexion. Wearing boots instead of shoes is always of considerable assistance.

**Fractures of other Tarsal Bones.** Crush fractures of the body of the navicular occasionally occur. They are often undisplaced, and demand merely a retentive foot plaster. Rarely, the upper fragment is extruded on the dorsum of the foot and may be difficult to retain in position. Excessive local pressure causes necrosis of the skin. Usually the combination of forced plantarflexion and light pressure is successful. The other tarsal bones are seldom injured except by direct violence such as the fall of a weight on the foot. The injuries are thus often compound. Care

maintained for eight to ten weeks. The foot may then be placed in plaster in the plantigrade position, and walking is permitted. A month later the cast may be removed.

If there is dislocation of the body of the talus it is likely to undergo avascular necrosis. There are frequently associated injuries to the malleoli and the calcaneus. The displaced body of the talus may produce pressure necrosis of the skin unless reduction is quickly carried out. The obstruction offered by the irregular processes around the bone and the displaced tendons makes closed reduction uncertain. Usually there are other indications for open reduction, which is therefore usually necessary. Control of the fragments after reduction may be difficult, and may need a single screw inserted through the head of the talus into the body. Retention in a padded plaster follows, and is renewed as swelling subsides. Weight-bearing must be considerably delayed, and radiographs repeated, without the plaster, at monthly intervals to watch for avascular necrosis. If this appears, fusion of the tibia with the head of the talus, or with the calcaneus must be carried out. It is useless to temporise.

Fractures of the posterior tubercle of the talus occur, but they are uncommon and must be distinguished from non-union of a secondary centre of ossification for the process. The margins of the line of separation of a secondary centre are smooth and organised, and there may be little evidence of soft tissue injury or hæmorrhage. A similar condition may be found in the other foot. A short period of immobilisation is all that is required for the fracture. Fissure fractures of the body of the talus, without displacement, may be seen, and occasionally there is crushing and fragmentation of the head.

**Fractures of the Calcaneus.** These follow falls from a height on to the heels, and may be accompanied by crush fractures of the vertebræ. They may be unilateral or bilateral. The two persistent complications encountered are stiffness and rigidity of the posterior part of the foot, and persistent pain. Both are due to damage to the subtaloid joint. Experience has shown that immobilisation of the foot in plaster, especially if the calcaneus is transfixed with a pin, greatly increases the stiffness of the whole foot, and so subsequently increases the strains placed on the damaged subtaloid joint. If accurate reduction is the aim, retention is necessary to maintain the position, but it is only in a few exceptional cases in which the function of the subtaloid joint is likely to be improved thereby, that these steps are justified. In inexperienced hands, and perhaps even in experienced hands, better results follow immediate massage and exercises. This disperses the hæmatoma, and preserves function in the undamaged joints of the foot; normal movement of these joints is necessary to compensate for the almost inevitable loss of subtaloid and midtarsal movement.

The following fractures do not interfere with subtaloid movements,

twenty-four to forty-eight hours. The simplest, often the most comfortable support, is provided by cutting the toe out of an old shoe.

With displaced fractures, fixation after reduction can be best achieved by means of a finger wire incorporated in a below-knee plaster ; the wire extends under the toe to which it is attached by strapping. This is also the best method for compound fractures, as the dressing can be readily removed and tightened, as swelling subsides and abrasions dry. A detached nail often slips back into an apparently normal position. As it may hide a rupture of the nail bed, or encourage infection, it is best removed when damaged. Pain due to subungual tension is often greatly relieved by removal of the nail. Complete pulping of the terminal phalanx may demand amputation, but as with the thumb, as much length as possible should be preserved.



must be taken with the skin which is often devitalised by pressure. Early elevation of the foot is essential to minimise swelling.

**Fractures of the Metatarsals.** The most commonly injured metatarsal is the fifth. It may be injured not only by direct violence but by inversion injuries of the foot, and fracture is thus encountered in association with ruptures of the fibular collateral ligament of the ankle, midtarsal sprains, and dislocations of the talus. The fracture is usually a transverse one, but the tip of the base may be avulsed. Fractures of the secondary centre for the epiphysis are also common.

Single or multiple fractures of the metatarsal shafts may be encountered. They can usually be manipulated into fairly good position and retained in this position by a plaster cast. Union is firm in four weeks. Weight-bearing in the plaster can be permitted at this time, but the plaster should be retained for another two weeks.

**March Fracture.** This is the commonest example of a fatigue fracture, and, as the name indicates, is found in young recruits undergoing heavy military training for the first time. The inorganic constituents of bone separate across a plane of stress in the bone, in a similar manner to the molecules of a metal after repeated stress. Some attempt is made to reinforce the weak place by deposition of subperiosteal bone. There is always radiological evidence of subperiosteal new bone formation. The fracture is seen as a fine irregular line across the centre of the expanded bone. The amount of pain before the fracture occurs is variable; sometimes it is trivial.

The fracture is an indication of deficient muscular control. If this were good the fracture would not occur. The fracture is undisplaced, and already undergoing repair when patients seek advice. It is illogical to fix it in plaster and thus provoke further muscular atrophy. The correct treatment is active exercises, graded according to the pain, and progressing to full activity.

**Fractures of the Phalanges.** Fractures of the proximal or distal phalanges of the great toe are of importance for they may give rise to pain on walking. Accurate reduction and adequate fixation of the fracture are necessary. The fractures resemble those encountered in the phalanges of the fingers. Crushing injuries are the most common, and are often associated with skin damage and loosening of the nail. The fractures encountered are: (a) Undisplaced fissure fractures; (b) fractures of the shoulders of the phalanges; (c) transverse fractures of the proximal phalanges; (d) avulsion fractures at the insertions of the collateral ligaments; (e) severely comminuted fractures. Elevation of the foot to avoid swelling is important. The immediate use of non-expandable dressings, such as collodion, is dangerous, as they may cause constriction. However, in undisplaced fractures, a firm supporting dressing made of collodion, or gauze and mastisol may be used after

the treatment appropriate to burns may be necessary (Chap. 107). Splinters and foreign bodies require careful removal after skin toilet. The subungual splinter which has broken off may be readily extracted by soaking the nail in warm water to soften it, and then paring a narrow strip from the nail over the splinter (Fig. 673). More extensive injuries, demanding suture and wound toilet, should be treated with full aseptic ritual in a "clean" theatre, where all the necessary facilities are available (Fig. 674). General anaesthesia, a bloodless field, efficient lighting, and a set of fine instruments are essential. Such injuries fall readily into two groups: (1) Those in which wound toilet and suture can be safely followed up in the out-patient department; (2) those in which the complicated nature of the injury demands admission to hospital.

**Severe Hand Injuries.** The first-aid treatment consists of stopping any arterial hæmorrhage, by clamping and ligating the vessel if possible, or by pressure. Care must be taken over the first-aid use of a tourniquet. A badly applied tourniquet is worse than none at all, for it causes venous bleeding. Tourniquets may only be left on for a limited time (twenty to thirty minutes) before release and if necessary re-application. The hand should be splinted with fluffed-out gauze in the resting position with the fingers flexed, and supported on a hand splint. The whole hand is then bandaged gently but securely. All cleaning and probing, other than removal of large and obvious foreign bodies, is best done at the time of definitive treatment. Full doses of antibiotics are given.

A full evaluation of the remaining function of the hand must be made before operation, as knowledge of the function of a divided tendon or nerve is of vital importance in planning surgery, and cannot always be assessed at operation. X-rays are essential to determine accurately any bone damage and the presence of radio-opaque foreign bodies.

Three main conditions are encountered: (1) Lacerations, clean and without skin loss, which if seen early can be treated as surgical incisions. (2) Lacerations for which treatment has been delayed and which are probably infected. Extensive surgery must be postponed, and the lacerations only are repaired. (3) Crush injuries, usually accompanied by skin loss or the threat of skin loss; they must be carefully débrided, although extensive surgery is avoided. Steps are taken to obtain skin cover.

Definitive treatment consists of thorough cleansing of the hand and forearm with a sterile detergent, as for compound fractures (see Chap. 93), removal of foreign material, and careful but conservative débridement of the wound. In suitable cases, repair of damaged structures may be carried out as follows: (a) Repair of all nerves including digital nerves (Chap. 61). (b) Suture of all tendons after careful squaring of the ends (fine silk or wire may be used as suture material). One possible exception to this rule exists. Thus, as suture of the extensor digitorum profundus and sublimis where they lie together in the digital tendon sheath is

## CHAPTER 106

### INJURIES AND INFECTIONS OF THE HAND

It is appropriate that these two subjects should be considered together, as two-thirds of the cases of infection of the hand have a traumatic origin. One of the surest methods to reduce the incidence of infection is the correct and early treatment of any injury of the hand which has breached the continuity of the skin, and it is important that the value of skin-toilet, wound cover and, if necessary, immobilisation should be recognised, even for trivial injuries. Such a discipline can be insisted on in enlightened industrial units, with consequent reduction in sepsis. In home accidents the care is apt to be inadequate until the onset of inflammation demands medical attention.

The treatment of hand infection has been dominated in the past by anticipatory incisions to relieve pressure, and the relationship of these incisions to the anatomical compartments of the hand. With the ready availability of effective antibiotics, the whole outlook has been altered, and the emphasis is now on adequate antibiotic therapy, adequate rest of the part, and minimal surgery after localisation. Infections of tendon sheaths and palmar spaces are now rareties.

Much of the credit for the advance in the treatment of hand injury and infection is due to the organisation of hand clinics, where the special experience gained has resulted in a better appreciation of the varieties of infection, the dangers of intermittent and inadequate care, and the value of conservative surgery. The infected finger is no longer the perquisite of the most junior member of the staff. All injuries, other than minor lacerations and abrasions which may be treated by the casualty staff, should be referred to a hand clinic. Here facilities for the sterile treatment of wounds and injuries should be available so that clean and infected cases can be treated without the risk of cross-infection, though this risk can be reduced by having clean and dirty cases treated at different times. The tremendous importance of the hand must be realised, and the need for admission to hospital and adequate bed rest in combating unsatisfactorily controlled infection must be accepted as a normal phase in treatment.

#### INJURIES OF THE HAND

Minor injuries of the hands are those in which the breach of skin continuity does not demand surgical closure to hasten healing, and avoid infection. Such small lacerations and abrasions should be cleansed with soap and water or other detergent, thoroughly dried, and covered with a non-airtight dressing to permit them to dry. In more extensive abrasions,

notoriously difficult, the sublimis may be sacrificed and the profundus alone repaired, or tendon repair may be left to a secondary reconstruction, or grafting. (c) Loose suture of severed muscle which is still actively contractile. (d) Repair of fascia and ligaments with fine silk. Absolute hæmostasis should be obtained. Small vessels are compressed till clotting occurs. Large vessels are ligated. The skin should be closed with fine non-absorbable sutures, e.g. black silk or stainless steel wire. Operation is followed by gauze pressure-dressing, "functional" splinting and elevation. When single fingers are injured, the other fingers may be left free and a finger wire used to immobilise the affected one. Never immobilise a finger unnecessarily.

**Bone Injury.** In many cases associated dislocations and fractures can be reduced, and they can be retained in position by packing with fluffed-out gauze and the use of a hand splint. In other cases, one or two fingers may be supported by means of a plaster of Paris forearm splint carrying a finger wire(s). Slings of strapping are applied across the finger wires and the affected fingers strapped or bandaged to them. Fingers and wires can then be bent together, for the correction of deformities. In a few instances in clean wounds internal fixation may be of value. Thus small screws may be inserted or wire sutures through bone and Kirschner wires may be used for intramedullary fixation; if necessary they may be passed across joints—with little damage to the joint—when fixation is important.

**Skin Loss.** If there is skin loss, immediate attention is given to this unless the area of ultimate loss is uncertain when the decision may be postponed for forty-eight to seventy-two hours, especially if the assistance of a plastic surgeon is required. Skin defects are closed most readily with split-skin grafts, and these can be replaced later by a full thickness graft if functionally unsatisfactory. Split-skin grafts do not take over bare bone or tendon, unless the area exposed is very small; when either of these is widely exposed, a full-thickness graft is needed. This may be obtained by advancement or rotation flaps, or by local or distant pedicle flaps (Fig. 675). Subsequent disturbance of dressings should be minimal, and



FIG. 675. A full thickness graft from the opposite arm being used to cover a skin defect over the dorsum of the right index finger. (Mr. Rainsford Moulem's case.)



FIG. 673. Paring a softened nail in order to remove a subungual splinter.

FIG. 674. A skin laceration of an index finger associated with division of the profundus tendon and a digital nerve. This type of wound is clean and a suitable one for immediate repair of both tendon and nerve.



not be so full or prolonged as with flexor tendons. In crush injuries across the fingers, adhesions of the extensor apparatus are almost inevitable with consequent restriction of function.

Tendons are not firmly united at the suture line until the end of about three weeks; immobilisation should therefore continue until then, and movements are allowed after this period. Tendon repair is carried out by a figure-of-eight suture, reinforced by finer approximation sutures



FIG. 676. Tendon graft. After division of the flexor profundus and sublimus the damaged tendons have been excised. A graft from the extensor tendons of the toes has been threaded along the digital sheath and is shown lying ready for suture to the stump of the flexor profundus at the base of the proximal phalanx and the flexor profundus close to a lumbrical in the palm. In each case the tendon has been buttonholed to facilitate suture.

where necessary. Atraumatic cutting-edge needles should be used ( $\frac{1}{8}$  inch curved). Fine waxed silk or stainless steel wire sutures are recommended.

#### INFECTIONS OF THE HAND

Control is obtained by a combination of antibiotic therapy, rest and conservative surgery. *Antibiotic Therapy*—This should be begun as early as possible. While the result of sensitivity tests is awaited penicillin is administered. Antibiotics are given until infection is controlled and localised. When bone is involved, they may have to be continued for a fortnight or more. *Rest*.—This is of fundamental importance, and in severe infections must include bed rest and elevation of the arm on pillows

re-dressing should always be carried out under strictly aseptic conditions—preferably in a theatre.

**Traumatic Amputations.** The aim is to save the greatest possible length of the digit, particularly of the thumb. Adequate skin cover must be obtained to avoid infection and contracture (*vide supra*). Stripping of the thumb needs a pedicle graft. Guillotine amputation through the pulp can usually be covered by a split-skin graft. Exposed bone can be nibbled back a little to expose cancellous bone and a split-skin graft applied. Wider loss of pulp is best made good by a small flap raised from the thenar eminence or on the volar surface of the pulp by a skin flap from the dorsum of a neighbouring finger. Split-skin grafts form the best immediate treatment in all cases of doubt.

**Tendon Repair. Flexor Tendons.**—The primary need for the successful return of function of a severed flexor tendon is the absence of adhesions after repair. These are inevitable if there is infection, and when there is any doubt about its presence or development, repair is left until a clean field is assured. Only two or three weeks' delay is required; no advantage is to be gained by further postponement of operation. Adhesions are also inevitable at the site of suture, and indeed it is by adhesions that the sutured tendon repairs itself. It is therefore essential that the sutures should be so placed that when adhesions form they produce the minimal interference with the function of the tendon. Ideally, the suture line should be at the point of attachment of a flexor tendon to the terminal phalanx, where there is minimal glide, and proximally, in the palm or wrist where there is sufficient mesotendon and tissue mobility for movement to re-develop rapidly, and where the suture line may be enclosed by healthy tissue such as the lumbrical muscles (Fig. 676).

In incised wounds, with no risk of infection, a single flexor tendon may be safely repaired. If adhesions form they will stretch, or can be freed by a subsequent operation (tenolysis). Repair of both flexor tendons together in the proximal part of the tendon sheath is, however, so productive of adhesions of tendon to tendon and tendon to sheath, that though it may be successful in the hands of the expert, it is best avoided by the less experienced. The profundus tendon only should be repaired and the sublimis tendon excised. When the sublimis is intact, however, it is a wise rule never to divide it during a primary repair. If there is any uncertainty of technique, or risk of infection, the restoration of function may be left to a subsequent grafting operation. With division of the tendon of the flexor pollicis longus, repair is possible at all levels.

In severe injuries across the wrist with multiple tendon division the sublimis may be sacrificed to facilitate repair and avoid massive adhesions in the carpal tunnel. The rules governing nerve suture are discussed under *The Peripheral Nerves* (Chap. 61).

**Extensor Tendons.** They can be repaired easily at all levels in clean wounds. Function rapidly returns, and immobilisation after suture need

tissue and any overhanging skin are removed. In basal infections, the base of the nail is cut across and removed, leaving the rest of the nail for protection of the nail bed.

*Subungual Infections.* Infection beneath the nail due to a splinter or a hematoma, usually results in the nail being floated off by pus. Removal of the nail is necessary. Involvement of the terminal phalanx in the infection is common. Lateral paronychia may spread to the pulp space.

*Pulp Space Infections.* The *felon*, or deep pulp space infection, is distinguished from other infections by the absence of an evident portal of entry. Infection arises in the deep fibrous septa of the pulp, usually following injury. The osteomyelitis which may follow is secondary in most cases. Localisation is awaited. Many cases present with a purulent blister or a sinus. Drainage is obtained by means of a small hockey stick incision. In many cases, probing reveals involvement of the terminal phalanx before radiological evidence of osteomyelitis. Sequestra should be removed; otherwise the bone is left untouched. Antibiotic therapy must be continued for at least a fortnight. Healing takes from ten to thirty days, depending on the occurrence of osteomyelitis. Tender scars and pulp deformities are a frequent end-result.

*Web Space Infections.* These are localised to the tissues between the metacarpals, but may spread to give more serious infections of the palmar spaces, if inadequately treated. Incisions for drainage are made longitudinally between the metacarpals. In the first interosseous space, direct approach to a localised infection is made if possible. If not, the line of incision is made along the curved edge of the first dorsal interosseous muscle.

*Tendon Sheath Infections.* Tendon sheath infections due directly to perforating injuries or hæmatogenous infection are rare. They are more commonly due to spread from local infection, when the tendon sheath infection is noted during exploration of the local lesion under a tourniquet. A limited exposure of the tendon should be carried out, for wide exposure results in spread of infection and sloughing of the tendon. Local instillation of penicillin or chloromycetin through a ureteric catheter placed in the depth of the wound may be of value.

*Palmar Space Infections.* Infection of the subcutaneous surface of the palm is common. Oedema may be marked and include the dorsum of the hand to such an extent that it may be thought to be the site of infection. Involvement of the deep palmar space is rare. The palmar compartments, deep to the palmar aponeurosis, are three: (1) Medial (or hypthenar); (2) intermediate (or deep palmar space); and (3) lateral (or thenar). Infection of the first *web space*, or of the subcutaneous palmar tissue, if extensive, may also be mistaken for deep infection. In infection of the deep palmar spaces, tenderness and swelling are more widespread, and there is a greater disturbance of pulse and temperature. In infections of the intermediate palmar space, the middle and ring fingers are flexed and



to reduce swelling. The hand is splinted in the position of rest, and the fingers packed with fluffed gauze which is controlled by a crepe bandage. Dressings should not be frequent; a single daily inspection often suffices, or in some cases it may be carried out every second day. *Surgery.*—With effective antibiotic treatment, resolution without pus formation is common. Surgery is only necessary when pus has formed. It should be carried out under general anaesthesia and in a bloodless field. Fine instruments and a gentle technique are necessary. Infection must not be spread by opening clean tissues. Nowadays incisions are made directly through the infected area. They should be short and if possible should not cross a skin crease. Drainage which has to be maintained by packing the wound is inadequate. When the skin is thick, a small ellipse of skin may be excised. Otherwise, the lips of the incision should remain open if superficially held apart by a strip of vaseline gauze.

Thiersch (split-skin) grafting or sometimes full-thickness grafting may be necessary to hasten healing when there is skin loss, but clean granulating areas often heal surprisingly rapidly and with little contracture.

**Special Sites of Infection.** *Subcuticular Infection.*—In this type of infection the epidermis is lifted off the dermis by a collection of pus, often originating in a blister. Occasionally this indicates a deeper infection and the formation of a collar-stud abscess. Then there is more diffuse tenderness around the area of infection. Careful excision of the overlying dead skin (without anaesthesia) results in rapid healing.

*Subcutaneous Infection.* As the dorsum of the hand and the proximal phalanges are the only hair-bearing areas in the hand, it is only at these sites that boils or carbuncles occur. The multiple pockets of a carbuncle should be incised only when drainage is unsatisfactory. Separation of sloughs may be hastened by the use of hypertonic saline dressings. Large areas of skin loss may later require grafting. Abscesses in other regions of the hand may follow penetrating injuries or retention of foreign bodies. Rest and antibiotics produce localisation of the pus in most instances. Small direct incisions are then made over the abscesses. Thick overhanging epidermal edges—common in the hands of manual labourers with thickened skins—are cut away. Spread of infection is nowadays uncommon in the hand with the use of antibiotics.

*Diffuse Cellulitis.* This may arise from infection of a haematoma, but is more commonly due to streptococcal infection, when it may be accompanied by lymphangitis and adenitis. Resolution without suppuration may take place. Localised pus is treated by local incision. Rarely an erysipeloid type of infection is seen.

*Infections around the Nail* (Basal and Lateral Paronychia). There is granulation tissue or a thin purulent discharge around the nail following a slight localised infection. At this stage free drainage is required, and this demands removal of part of or the whole of the nail. In chronic lateral infections, a narrow section of the side of the nail, the granulation

## CHAPTER 107

### BURNS

by

VALENTINE A. J. SWAIN

A **BURN** is the reaction of tissue to injury by heat. The local damage so produced affects the whole body and its extent determines the outcome. A superficial burn of the skin causes redness and blistering, whereas a deep burn destroys and chars the underlying tissue (some of the severest burns are seen in epileptics, who have fallen unconscious near an open fire). It is important to recognise whether the affected area of skin is totally or partially destroyed in order to plan the future local treatment. With superficial skin damage healing occurs without skin grafting, but with full-thickness skin damage grafting is required. At first, estimation of the depth of burning may be difficult, but it becomes easier a week or so after the injury. With full-thickness skin damage, the area remains white or charred, whereas, with superficial skin damage, redness and small red punctate areas are seen. The area and depth of a burn are estimated in order to gauge the fluid requirements of the patient. The percentage of the body surface involved can be estimated by "the rules of nine" (Fig. 677).

The general circulatory effects of a severe burn become apparent early, and increase in severity unless promptly treated. At the onset, increased capillary permeability at the burnt areas causes an exudate of fluid rich in proteins and salts; this is mostly visible in the blisters and in the tissue oedema around the burn. Much more fluid is lost from the circulation into the affected tissues soon after burning than can be returned by the damaged lymphatics and capillaries. The diminution of the circulating plasma leads to hæmoconcentration. The *fluid loss* into the burnt area continues for about forty-eight hours after burning, and is maximal in the first eight hours. Initially, with extensive untreated burns, there is withdrawal of fluid from the interstitial spaces leading in turn to depletion of water in the body cells. This may result in permanent kidney damage. Blood also is lost to the circulation by destruction of red blood corpuscles as the result of burning of the capillary network, and hæmolysis produced by the heat; in extensive burns, over a third of the blood volume may thus be lost. After about forty-eight hours the tissue oedema around the burnt area diminishes; dilution of the plasma results and is followed by diuresis.

painful to move. In thenar (lateral) space infections, the thumb is abducted and held flexed at the distal joint. The index finger may be flexed and a little painful to move. The thenar space is drained by extending the first web space incision towards the palm. The palmar space is incised through the distal crease of the palm. The medial palmar space is rarely infected.

*Joint Infections.* These must be treated by more radical incision. Penicillin instillation is useful. Healing is slow. A painless pseudarthrosis may form, but more commonly an arthrodesis in the semiflexed position will be needed.

*Burns of the Hands.* This subject is discussed under burns (Chap. 107).

TABLE I

Age	Weight		Satisfactory hours urine output (ml.)	Reductive dose of morphine	
	lb.	kg.		Grain	mg.
Birth-3 12	11	5	8-20	$\frac{1}{32}$	2
3-12-6 12	15	7	8-20	$\frac{1}{16}$	2
6-12-9 12	20	9	8-20	$\frac{1}{8}$	2.5
9-12-1	22	10	8-20	$\frac{1}{4}$	2.5
1-2	29	13	20-24	$\frac{1}{4}$	2.5
2-3	33	15	20-24	$\frac{1}{4}$	4
3-4	37	17	20-24	$\frac{1}{4}$	4
4-5	40	18	20-24	$\frac{1}{4}$	4
5-6	44	20	24-28	$\frac{1}{2}$	5
6-7	44	20	24-28	$\frac{1}{2}$	5
7-8	53	24	24-28	$\frac{1}{2}$	5
8-9	57	26	24-28	$\frac{1}{2}$	8
9-10	64	29	30-35		8
10-11	70	32	30-35		10
11-12	77	35	30-35		10
12-13	86	39	35-50		10
13-14	95	43	40-60		10
14-15	106	48	50-100		10
15-16	110	51	50-100		16
16-17	120	59	50-100		16
17-18	141	64	50-100		16
Adult	154+	70.4	50-100		16

(A. B. Wallace, 1933.)

**Treatment by Dressings.** After the initial cleaning the burnt area is covered with a layer of tulle gras (sterile vaselined gauze); next a thick layer of sterile gauze and wool is applied. The dressing is held in place by the even pressure of a crêpe bandage. It may not be possible to cover all areas by this method, owing to slipping of the bandage and its liability to constrict the underlying tissue, especially in the limbs. The dressing should be left undisturbed for as long as possible, although the outer layers may have to be changed if: (a) The bandage becomes too tight owing to œdema; (b) the dressing becomes soaked with exudate; (c) persistent pyrexia develops, indicating infection underneath the eschar; or (d) the dressing slips.

The time of the first change of dressing depends on the nature of the wound. Deep, localised burns requiring excision may be left undisturbed for five days. Superficial burns need not be disturbed until the tenth or fourteenth day, when signs of healing will be evident. Burns entailing full-thickness skin loss require redressing after seven to ten days; if they are infected, sloughs or dead skin, which are then well demarcated, should be excised and the resulting raw area is prepared for skin grafting. If dead skin is very adherent, frequent dressings with normal saline solution or sodium hypochlorite 5 per cent. assist separation of deep sloughs.

**Treatment by Exposure.** This method is becoming more popular, though few hospitals are suitably equipped for it. After the initial cleaning (*vide supra*), the burnt surface is left exposed, and the patient is

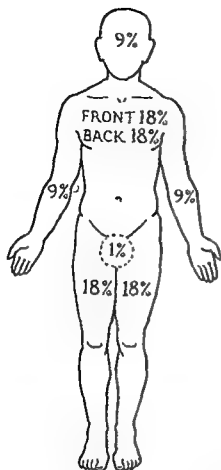


FIG. 677. For estimation of the area of a burn in an adult, the easily memorised "rules of nine" are based on the following scheme :

Head and neck . . . . .	9 per cent.
Whole of one upper limb . . . . .	9 "
Whole of one lower limb . . . . .	18 "
Whole of anterior aspect of trunk . . . . .	18 "
Whole of posterior aspect of trunk . . . . .	18 "
Perineum . . . . .	1 "

(A. B. Wallace, 1953.)

**Immediate Treatment.** A recent burn should be covered with a sterile sheet or towel. Local applications of ointments or oils are inadvisable, for they interfere with further treatment. Large burns should be treated in hospital as soon as possible. Pain is relieved by opiates. The doses of morphia required at the various ages are given in Table I. After assessment of the depth and extent of the burn, the fluid requirements are estimated (*vide infra*) ; if the burn affects over 10 per cent. of the body surface, intravenous fluids are required forthwith, in anticipation of oligæmic shock.

*Local treatment* aims at : (1) Minimising infection ; and (2) preserving function. *Strict asepsis* is maintained throughout. The burnt area is cleaned with warm sterile 1 per cent. cetrimide, under general anaesthesia or sedation. Loose skin tags are removed, and blisters are punctured, but are otherwise left intact to cover the underlying raw surface. The wounds are then dried with warm air from a hair drier, or with soft sterile towels. Subsequent treatment is then decided, and depends on the site of the burns and the facilities available. Two methods of treatment are in current use : (a) Application of dressings ; (b) the exposure technique. Either is used independently or both are used together according to circumstances.

have "taken." Later, the healed grafted areas can be made supple by repeated application of vaseline or lanolin cream. The above method of split-skin grafting is suitable for slightly infected areas.

If the area is sterile at the beginning, excision of the burn, followed by immediate skin graft is indicated. Haemostasis is necessary to ensure primary healing. A firm pressure dressing is applied and the area splinted for the first few days after operation. When the dressing is removed, seven to ten days later, vascularisation of the graft is evident. If, however, infection develops, the grafts cannot survive and should therefore be removed. The bare area is then prepared and delayed grafting carried out. Irrigation, or a moist dressing soaked in sodium hypochlorite is suitable to clean the surface.

### SPECIAL AREAS

**Hands, Wrists and Fingers.** The treatment of burns in these areas aims at maintaining the position of joints in the best functional position, and the prevention of œdema. Small surface burns on the back of the hand are treated by application of tulle gras, or penicillin cream, and a pressure dressing. After about ten days, the depth of the burn is apparent and indicates either further dressings until it is healed, or skin grafting after excision. In extensive burns involving the fingers it is most important to dress all the fingers separately with tulle gras. A roll of wool and gauze is then placed in the palm of the hand, and by bandaging over this the optimum flexion of all the joints of the hand and fingers is obtained. The hand is kept elevated for four to five days to prevent swelling. Movement of the fingers is carried out under the dressing. When the wound is redressed after about six days, the extent of the burn is more apparent; the more superficially affected areas can be allowed to heal, while the deeply affected areas are prepared for skin grafting. The eschar of deep burns can be softened by moist dressings and saline baths. Deep burns involving tendons and joints are covered by full thickness grafts.

**The Face.** Burns of the face are best treated by the exposure technique, after cleaning the area with 1 per cent. sterile cetrimide. In places, grafting may be required when areas of dead skin have separated. *The Eyes.*—Burns involving the eyes should be washed initially with normal sterile saline, and a search made for corneal abrasions, which can be made visible by the use of 2 per cent. fluorescein drops; subsequently 1 per cent. atropine eye drops are instilled daily and 10 per cent. sulphacetamide drops three times daily. Burns involving the eyelids should be grafted soon after burning, for neglect to do this is liable to cause exposure of the cornea due to shortening of the lids by scarring.

**The Perineum, Scrotum and Penis.** These regions are best treated by exposure methods; in children, the use of suspension by means of a gallow's splint is recommended; the areas are thus kept as clear as possible from soiling.

nursed in a warm air-conditioned \* room (65° to 70° F. or 18° to 20° C.); strict asepsis is maintained. *Staphylococcus* carriers should not be allowed to enter the room. The patient lies exposed on the bed, being covered only with a sheet over a cradle, so that there is no skin contact with the draping. This method aims at securing cover of the burns by natural coagulum; crusting usually occurs within forty-eight hours. The injured parts are immobilised as necessary to prevent cracking of the eschar. Subsequent treatment depends on the depth of the burn and its site. The exposure treatment is contraindicated in: (a) Burns of the hands; (b) burns which encircle the limbs, for the eschar produces a tourniquet effect causing pressure at the site and distal oedema (should any constriction develop inadvertently, multiple incisions through the eschar will bring relief); (c) those burns of the trunk which prevent patients lying on intact skin; and (d) burns on the neck as they are difficult to keep dry.

After about ten days, the crusts begin to separate, leaving a healing surface, and in deep burns, the line of demarcation between dead and live skin is apparent. Preliminary softening of the eschar for two or three days by the application of liquid paraffin facilitates removal of all dead tissue in preparation for skin grafting.

**Skin Grafting.** A suitable area on the thigh, back, lower abdomen or chest is prepared. After stretching the skin between the edges of two pieces of wood, a Blair's knife is used to cut an even partial skin thickness graft. Alternatively, a wide skin graft of measured, even thickness can be cut by means of Padgett's dermatome. With this instrument the layer of skin adheres to the drum as soon as it is sliced. If necessary, the free skin can be stored after laying it out on vaseline gauze and wrapping it in a moist normal saline pack. The graft is placed in a container and kept cool in a refrigerator at 32° to 34° F. (0° to 1° C.) where it can be kept for two to three weeks.

**Application of a Graft.** The raw area to be grafted should be bacteriologically as clean as possible, and free from excess discharge and tags of dead tissue. The granulating area should, if possible, be bacteria free, but more often than not organisms such as the haemolytic streptococcus, *Staphylococcus aureus* or Gram-negative bacilli (*pyocyaneus* or *proteus*) are present. In some instances, the flora can be suitably changed by the use of local or systemic antibiotics. Postage stamp patches of skin are generally suitable for grafting; with the skin placed on a layer of tulle gras, various sizes of graft can be cut with scissors; they are placed in position, side by side, to cover the prepared area, which has been previously washed with saline. Finally, a dressing of gauze and wool is applied and held in place by a crêpe bandage: splinting of the area is advisable to prevent movement of the grafts. The wounds are left undisturbed for seven to fourteen days, by which time the grafts should

\* Ideally the air should pass through an electric bacterial filter.

have "taken." Later, the healed grafted areas can be made supple by repeated application of vaseline or lanolin cream. The above method of split-skin grafting is suitable for slightly infected areas.

If the area is sterile at the beginning, excision of the burn, followed by immediate skin graft is indicated. Haemostasis is necessary to ensure primary healing. A firm pressure dressing is applied and the area splinted for the first few days after operation. When the dressing is removed, seven to ten days later, vascularisation of the graft is evident. If, however, infection develops, the grafts cannot survive and should therefore be removed. The bare area is then prepared and delayed grafting carried out. Irrigation, or a moist dressing soaked in sodium hypochlorite is suitable to clean the surface.

### SPECIAL AREAS

**Hands, Wrists and Fingers.** The treatment of burns in these areas aims at maintaining the position of joints in the best functional position, and the prevention of oedema. Small surface burns on the back of the hand are treated by application of tulle gras, or penicillin cream, and a pressure dressing. After about ten days, the depth of the burn is apparent and indicates either further dressings until it is healed, or skin grafting after excision. In extensive burns involving the fingers it is most important to dress all the fingers separately with tulle gras. A roll of wool and gauze is then placed in the palm of the hand, and by bandaging over this the optimum flexion of all the joints of the hand and fingers is obtained. The hand is kept elevated for four to five days to prevent swelling. Movement of the fingers is carried out under the dressing. When the wound is redressed after about six days, the extent of the burn is more apparent; the more superficially affected areas can be allowed to heal, while the deeply affected areas are prepared for skin grafting. The eschar of deep burns can be softened by moist dressings and saline baths. Deep burns involving tendons and joints are covered by full thickness grafts.

**The Face.** Burns of the face are best treated by the exposure technique, after cleaning the area with 1 per cent. sterile cetrimide. In places, grafting may be required when areas of dead skin have separated. *The Eyes.*—Burns involving the eyes should be washed initially with normal sterile saline, and a search made for corneal abrasions, which can be made visible by the use of 2 per cent. fluorescein drops; subsequently 1 per cent. atropine eye drops are instilled daily and 10 per cent. sulphacetamide drops three times daily. Burns involving the eyelids should be grafted soon after burning, for neglect to do this is liable to cause exposure of the cornea due to shortening of the lids by scarring.

**The Perineum, Scrotum and Penis.** These regions are best treated by exposure methods; in children, the use of suspension by means of a gallow's splint is recommended; the areas are thus kept as clear as possible from soiling.



## SPECIAL BURNS

**Acid and Alkali Burns.** Acids are neutralised with 2 per cent. sodium bicarbonate solution, and alkalis with 1 per cent. acetic acid. Recently, a buffer solution has been recommended for burns produced by acids or alkalis. It consists of monobasic potassium phosphate (70 gm.), dibasic sodium phosphate (180 gm.) and water (850 ml.). The burnt areas are washed with this solution, and, if necessary compresses soaked in it are frequently applied. Subsequent treatment depends on the extent of the burning, being carried out according to the principles already enunciated for other types of burns.

## FLUID REPLACEMENT IN BURNS

In all cases of burns it is important to : (1) Restore the fluid loss from the circulation, and (2) establish and maintain good renal function. The amount and nature of the fluid requirement, and whether it is given by mouth and/or intravenously, depends on the age of the patient and the extent of the burns. Various formulæ and charts are available for calculating the amount, but these should be regarded as rough guides only, for each case should be treated individually, as circumstances vary considerably. In practice, the fluid requirements can be roughly assessed as follows : 3 ml. of fluid are required for each 1 per cent. area of burn per 1 kg. body-weight in the first forty-eight hours ; this is *in addition* to the normal metabolic requirements. The initial fluid replacement should be given as soon as possible after the injury, when the fluid loss is greatest. After the amount required for the first forty-eight hours has been calculated, this total is divided by three ; a third is given within the first eight hours from the time of burning, another third is given in the next period of sixteen hours (i.e. eight to twenty-four hours after burning), and the final third is administered during the next period of twenty-four to forty-eight hours, that is, throughout the day after the accident. The above formula holds good for burns up to 30 per cent. of the body surface ; beyond this percentage, fluid equivalent to 10 per cent. of the body-weight is given.

Estimation of the area burnt can be roughly assessed in an adult with the aid of Fig. 677. In children, adjustments to the calculation are made in accordance with Fig. 678. In minor burns, i.e. of less than 15 per cent. of the adult body surface (10 per cent. in children), the total fluid requirements can be given by mouth. Glucose water is most suitable. In more severe cases, oral and parenteral fluid are necessary. The nature of the intravenous fluid depends on the severity of the burn. In superficial burns of less than 25 per cent. of the body surface, colloid (e.g. Dextran or plasma) and electrolyte solutions are given in the proportion 1 : 1, whereas in more extensive damage, especially if the burn is deep, blood, plasma and electrolytes are given in the proportion of 2 : 1½ : ¼ (Wallace,

1955). Early blood replacement is essential in deep burns in order to balance the blood volume deficit, and prevent anoxia. In severe cases, administration of oxygen may be life-saving, and is especially necessary in cases in which the air passages are burnt or the movements of the chest wall are restricted by muscle damage. Throughout treatment, regular assessment of the clinical condition is made. The urinary output is probably the best guide as to whether the correct amount of fluid has been given, for the aim should be to maintain a steady flow of urine ;

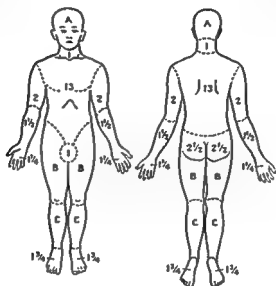


FIG. 678. *Percentage Surface Area at Various Ages* (modified from Lund and Browder). The areas A, B and C are estimated according to the age of the patient as follows :

Area in diagram	Age (years)					
	0	1	5	10	15	Adult
A (= $\frac{1}{3}$ of head)	$9\frac{1}{2}$	$8\frac{1}{2}$	$6\frac{1}{2}$	$5\frac{1}{2}$	$4\frac{1}{2}$	$3\frac{1}{2}$
B (= $\frac{1}{3}$ of one thigh)	$2\frac{1}{2}$	$3\frac{1}{2}$	4	$4\frac{1}{2}$	$4\frac{1}{2}$	$4\frac{1}{2}$
C (= $\frac{1}{3}$ of one lower leg).	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	3	3	3

(A. B. Wallace, 1953.)

this is checked by having a catheter in the bladder and measuring the urinary output every hour. The average output to be maintained is shown in Table I. In adults especially, there may be delay in reaching the required volume owing to the antidiuretic response following injury (see Chap. 108). Also, sedation by morphia tends to diminish urinary excretion. Other methods of assessing the progress can be used, such as periodic hæmoglobin estimation, hæmatocrit readings, and estimation of the electrolytes. Adjustments of the fluid requirements demand particular care in the extreme years of life. See also *Reactions to Injury*, Chap. 108,

and *Fluid Requirements for Infants and Children Undergoing Surgical Treatment*, Chap. 23.

**General Management.** It is important to maintain good nutrition and to prevent anæmia, so that any associated infection can be better resisted and healing hastened. A high protein and a high calorie diet is necessary, for there is an increase in the basal requirements of protein, and this remains so for several weeks after burning. The normal daily protein requirement for a child is 4 gm. per kg., whereas that of an adult is 1.5 gm. per kg. For a patient suffering from moderately severe burns, the normal protein requirements should be increased 50 per cent. in children and 100 per cent. in adults; this is proportionately increased with more severe burns. A normal infant requires about 100 calories per kg. per day, whereas a normal adult requires 45 calories per kg. body-weight per day; these amounts should be increased by at least a third for the burnt patient. Vitamins and iron should also be prescribed. If after the first few days of burning the patient is anæmic, blood transfusion is required. Measures, as outlined above, which hasten the covering and healing of the burned areas, shorten convalescence. Control of infection by antibiotics is also important.

Restoration of the full function of a burned limb is best attained by early skin cover and gradual movement of the joints. Deep burns at the flexures may cause marked contractures. To prevent this, grafting of the areas, preferably with full thickness skin, should be carried out. In some cases hypertrophy of scar tissue (keloid) may be unsightly, and require excision and regrafting later; in these circumstances, superficial irradiation of the wound with X-rays before and after grafting helps to prevent recurrence of excessive scar tissue.

In the writing of this section great help has been obtained from the publication of the *British Association of Plastic Surgeons* entitled "Management of Burns," *The Lancet*, 1954.

## SECTION XIII

### REACTIONS TO INJURY. BLOOD TRANSFUSION SURGERY IN DIABETICS. ANTIBIOTIC THERAPY

by

A. A. G. LEWIS

#### CHAPTER 108

### REACTIONS TO INJURY. SURGERY IN DIABETICS

#### REACTIONS TO INJURY

THE immediate reactions of conscious patients to injury vary from transient emotional disturbances to collapse and unconsciousness. Certain injuries may have immediate consequences which dominate the clinical picture ; among these are head injuries, sucking chest wounds, injuries to the heart and pericardium with cardiac tamponade and hæmorrhage from a divided artery. Two types of general reaction, however, are common after severe trauma ; the *vasovagal*, with pallor, sweating, bradycardia, low blood pressure and sighing respirations, and the *hypertensive*, in which the blood pressure is raised, while colour and pulse rate may be normal. Either may be followed by recovery, or may be succeeded by shock. At the same time a complex metabolic response to injury is initiated, which may continue for days or weeks. These reactions may be intensified by incidental factors, such as exposure, starvation, dehydration, fat embolism or infection, or may themselves give rise to complications, such as acute renal failure, which may determine the outcome.

These are reactions to fear, pain, hæmorrhage and perhaps to massive afferent nerve stimulation. They are mediated by :

(1) The *autonomic nervous system*, modifying pulse rate, cardiac output and peripheral vascular tone.

(2) *Hormones*, of which the actions may be summarised as follows :

(a) From the *adrenal medulla*, stimulated via the splanchnic nerves, *adrenaline* (causing tachycardia, increase of cardiac output and systolic blood pressure, constriction of splanchnic and skin arterioles with relaxation of those in muscle and conversion of hepatic glycogen into glucose) and *noradrenaline* (raising diastolic blood pressure).

(b) From the *anterior pituitary*—possibly as a result of the action of adrenaline on the hypothalamus—*adrenocorticotrophic hormone* (ACTH) and, possibly, *thyrotrophic hormone* (TSH).

(c) From the *adrenal cortex*—from stimulation by ACTH—17-hydroxy-corticosterone (cortisol, compound F.) which leads to the increased formation of glucose from amino-acids and of hepatic glycogen, and causes eosinopenia and lymphopenia; and *aldosterone* (bringing about increased sodium reabsorption and potassium secretion by the renal tubules).

(d) From the *posterior pituitary*—after emotion, and also after a fall in left auricular pressure or rise in blood sodium concentration—*vasopressin* (which stimulates renal tubular water reabsorption, so that the urine volume is reduced and its specific gravity raised, and in large quantities causes constriction of cutaneous and hepatic blood vessels with reduction of blood flow through the skin and liver).

(3) Other hypothetical factors—for example, *serotonin* liberated from clotting blood may cause local vasoconstriction, while the vasotropic factors VEM and VDM (vaso-excitor and vasodepressor material), said to be formed by the kidney and liver, may modify vascular tone (in particular VDM, increased in hepatic anoxia, may depress it). In addition, toxins formed in severe infections, particularly by gas-forming organisms, may cause vasomotor failure within a few hours of injury.

The reactions may, therefore, be exceedingly complex, and it is not surprising that phases of slow or rapid heart action and of high or low blood pressure may sometimes occur and may follow one another.

**The Immediate Reaction to Injury.**—This may be dramatic, but is often very transient. But shock may supervene, even after a normal interval, especially when hæmorrhage persists from a wound. It is, therefore, essential to arrest bleeding at the earliest possible moment. In this stage, the blood pressure is not a reliable guide to the extent of blood loss, and if this is thought to have amounted to 10 per cent. or more of the blood volume intravenous replacement should be given at the first opportunity, especially if the injury is severe or involves much soft tissue, or if the abdomen and chest have been penetrated. A sucking chest wound must immediately be closed. The patient should be laid flat and kept warm (too much heating, by a heat cradle, for example, leads to sweating, vasodilatation and reduction of effective blood volume). Fluids should be given if the patient can swallow. Fractured limbs must be splinted, but severe manipulations must be avoided as they produce shock. Pain should be relieved by morphine, given intravenously if necessary, though in many injuries pain is not great. The injection of anti-tetanic serum must not be forgotten, and as many people have now been immunised against tetanus, a boosting dose of toxoid should be given as well. Broad spectrum antibiotic therapy should be begun at once. A record of pulse rate, blood pressure, general appearance and state of consciousness and of urine volume should be made at regular intervals.

**Traumatic Shock.** This is a life-menacing clinical condition, occurring within a few hours of injury, associated with a reduction of effective blood volume (that is, in relation to the vascular bed) and characterised by

reduction of the systolic blood pressure. This is a working definition, which is not invalidated by the fact that a phase of normal or even raised blood pressure may precede or, in a few less severe cases, entirely replace this hypotensive state.

While the nervous and humoral influences initiated by an immediate reaction to injury may persist and be partly responsible for some of the features of this condition, by far the most important factor is loss of blood volume. This may occur from hæmorrhage, from loss of plasma from burnt areas or into crushed or lacerated tissue, or from loss of fluid by diarrhoea and vomiting or into obstructed or paralysed bowel. In this stage, the systolic blood pressure gives some indication of the deficit in blood volume: when above 100 mm. Hg this is not likely to exceed 10 per cent. unless there is a pulse rate above 100; when below 100 mm. Hg it may be as much as 30 per cent., and with a very low pressure and a pulse rate above 140 it may be 50 per cent. The patient is usually pale and apathetic, with cold clammy extremities. But in severe cases there may be restlessness, air hunger, profuse sweating and empty, constricted veins.

The treatment consists in early and adequate transfusion, sufficient to raise the blood volume to at least 90 per cent. of the estimated normal, and given as rapidly as possible, especially in the early stages in severe cases. This involves applying positive pressure to the transfusion bottle, using a device such as a Higginson's syringe; the usual precautions are taken to avoid air embolism, i.e. by discontinuing the transfusion before the bottle is empty of fluid. In very urgent cases, transfusion can be given into a peripheral artery or directly into the aorta. Since the immediate need is to restore the blood volume, the fluid transfused need not be blood itself, but may be plasma or a plasma substitute, such as Dextran (see the section on blood transfusion). But there is no doubt that injured patients do better if the hæmoglobin is simultaneously restored, and a figure of 70 per cent. should be aimed at, allowing for the dilution which will occur in the hours following recovery. Arrangements can, therefore, be made for grouping the patient while a plasma substitute is being given. Once adequate restoration of blood volume has been achieved the patient is ready for surgery, and it must be realised that this is an indispensable step in treatment. Hæmorrhage must be completely and finally arrested, dead tissue must be removed and, in the case of abdominal wounds, perforated or damaged viscera must be appropriately treated.

**Irreversible Shock.** It is probable that if early, rapid and adequate transfusion is given, few cases of shock will fail to respond. Cases in which the blood pressure fails to rise, or in which it falls after being restored to normal, are almost certainly complicated by continuing hæmorrhage, which may be internal or may be severe oozing from lacerated surfaces; the volume thus lost is not appreciated. In either

case, it may be necessary to operate before the condition is fully restored. It is often said, however, that there are cases in which the shock runs a progressive and irreversible course, irrespective of adequate transfusion. When this occurs some hours after injury, it is probable that overwhelming infection, particularly with gas-forming organisms (with circulation of toxins, or septicæmia), is responsible. It is also possible that the liver under conditions of reduced oxygen tension may form VDM, which profoundly depresses vasomotor tone, and which cannot be inactivated under the conditions of depressed circulation prevailing, so that a vicious circle, with deepening shock, is the result. This hypothesis, however, remains open to doubt.

*Effect of Anæsthetics.* Many anæsthetics, particularly ether and cyclopropane, and (when the blood volume is already low) thiopentone, may cause some vasodilatation and fall in blood pressure. The temperature and colour of the extremities may be good. In the injured patient, therefore, whose blood volume has not been adequately restored, a further condition of shock, in which the appearance of the patient is misleading, may develop. This has been termed "warm hypotension."

*The Metabolic Response to Trauma.* This apparently follows a wide variety of injuries, including surgical operations, fractures, burns and other forms of stress. There is an increased urinary excretion of potassium and nitrogen, with reduced output of sodium and water. After severe trauma, the rate of nitrogen excretion is greater than normal, even when there is no intake, representing a loss of tissue that may continue for days or even weeks. For several days the urinary potassium excretion exceeds the amount predictable from the loss of tissue indicated by the nitrogen loss. Sodium excretion is greatly reduced even when intake is maintained. The volume of urine is usually low for the first twenty-four hours, with a high specific gravity; after this, larger volumes can be formed so that the sodium and chloride concentrations may fall to low figures.

The extent and duration of these changes (which closely resemble the effects produced by ACTH) tend to be greater after more severe trauma. It has been shown that the blood level of 17-hydroxycorticoids rises after operations, and the depression of liver function by anæsthetics reduces the rate of conjugation and of removal of the adrenal steroids. They may also be utilised at an increased rate by the tissues. The low urine volume of the first twenty-four hours is probably the result of the release of antidiuretic hormone (ADH, vasopressin). The factors responsible for this release are complex, and may include dehydration (which is unlikely to be an important one except in battle casualties) and a fall in left auricular pressure. Emotional stimuli may also cause a release of this hormone. Occasionally this release lasts for two or more days in spite of adequate hydration.

It is possible that a similar mechanism leads to the secretion of aldosterone, reducing the output of sodium and increasing that of potassium

by the kidney. In this way, auricular pressure receptors may act as "volume-receptors" which initiate changes tending to maintain the blood volume. This again emphasises the importance of early and adequate replacement of blood loss after trauma; indeed it has recently been shown that the changes in water and electrolyte excretion after burns are least in those patients given early, adequate replacement therapy. On the other hand, it is quite possible to produce large positive balances of water and sodium after operations by giving intravenous fluids too rapidly. If the antidiuretic response is prolonged for two or three days, continuous infusion of 5 per cent. glucose, in the mistaken belief that the small volume of concentrated urine indicates dehydration, may lead to water intoxication, with headache, vomiting, coma and convulsions. Prolonged infusion of normal saline, given on account of the low urinary sodium concentration, can cause systemic and pulmonary oedema.

Very severe trauma produces responses which may last for more than a week. In those who are already ill or who are recovering from a previous operation, the changes may be less intense. In the more severely ill, low extracellular sodium levels are particularly likely to be found, in spite of a positive sodium balance. This phenomenon may occur shortly after operation if intravenous infusions of glucose are given when water cannot be freely excreted.

**The General Principles of Treatment.** These may be summarised as follows: (1) After any injury, effective circulating blood volume must be maintained. The level of the systolic blood pressure and, to a lesser extent, the pulse rate, are a guide to this. The use of pressor drugs is a mistake (except in special circumstances such as adrenalectomy or spinal anaesthesia or to combat a sudden collapse), for if not actually ineffective they may mask the true state of affairs (e.g. continued bleeding). (2) The calorie intake must be as high as possible. Protein is poorly utilised for a time; if adequate calories are not given it will be used to provide them. (3) The water intake should equal the volume of the urine added to that of insensible losses and of sweat (the latter resembles fifth-normal saline). With modern technique, insensible losses in the operating theatre are not great; an adult should not lose much more than the normal twenty-four-hourly loss of about 1 litre by this route. Thirst is usually a safe guide to water requirements; a dry tongue is often misleading; skin elasticity is helpful if nutrition is good. *Over-hydration is very unlikely to occur if fluids are being given entirely by mouth or by rectum.*

**Potassium Deficiency.** The normal intake of potassium is about 100 mEq. a day, of which 90 mEq. are excreted in the urine. For several days after trauma the urine may contain almost as much as this even if the intake is zero. A negative balance of the order of 200 mEq. is of little significance in a previously healthy patient, provided that the intake can soon be resumed. But potassium losses may be considerable from vomiting (up to 35 mEq. per litre of vomit), diarrhoea (up to 70 mEq. per day),



ileostomy or biliary fistula, and then if intake is low a significant depletion can soon occur. This may lead to apathy, anorexia, muscular weakness (especially of the legs), ileus and abdominal distension (it has been suggested that this may be one cause of paralytic ileus). Sodium retention and œdema may occur. The possibility of potassium deficiency should always be borne in mind under these circumstances of increased loss, or in any patient with the above symptoms. Trauma, surgical operations and prolonged steroid hormone therapy should be remembered as possible causes of increased potassium loss.

In diagnosis, a plasma potassium level below 4.0 mEq./litre is suggestive, while the ECG may show ST depression, low or inverted T waves, QT prolongation and prominent U waves. But normal ECG and plasma levels do not exclude deficiency, especially in dehydrated patients. Whenever possible, cumulative potassium deficiency should be prevented by increasing the intake of patients in danger of it. Potassium chloride can be given by mouth in tablet form; if this is impossible, it may be necessary to give the ion intravenously. This should never be done unless renal function is good, so that any excess can be rapidly excreted, for a rise of plasma potassium to about double its normal level carries a risk of cardiac standstill. The plasma level should be checked regularly, and the infusion, not exceeding 40 mEq./litre in concentration, should not be given faster than 20 mEq./hour. Patients who are to be maintained on intravenous fluids alone for several days should probably be given a moderate intake of potassium (between 50 and 100 mEq./day), so that, should any complication arise which suddenly increases losses, they are less likely to develop a significant deficiency at a time when intake cannot be raised.

**Regime for Patients Maintained on Intravenous Fluids.** Once the blood volume has been restored and any other estimated losses replaced, the following routine is suggested: (1) Water (as 5 per cent. glucose) should be given with care until the urine volume is rising. Two litres (approx. 4, 1-pint bottles) in twenty-four hours should be ample until then; 3 litres may be given later. (2) One pint of normal saline every twenty-four hours. (3) Six grams potassium chloride (80 mEq.) in 2 litres of water (to be included in reckoning (1) above). When the urine volume is low immediately after trauma, this should be halved. Whenever abnormal losses are occurring, twenty-four hour balances should be compiled.

Vomit and liquid faeces can be pooled for estimations of volume, sodium and potassium (normal formed stools can be neglected). An attempt should be made to estimate water loss by the skin: when water replacement is difficult to judge, the patient should be weighed daily, for sudden gross fluctuations in weight are due to changes in body water. Estimations of plasma levels are of little help in management, for these may be kept within normal limits by variations in water balance in spite of deficits, and plasma sodium levels are often low in very sick patients and

cannot be corrected. Once previous losses have been made good, treatment should aim at maintaining intake equal to output every twenty-four hours.

**Other Complications of Injury.** *Fat embolism* is commonly found at autopsy after severe injuries. It is doubtful if this is a contributory cause of death. However, massive infarction of the lungs (as with emboli from veins) is more likely to occur when the pulmonary circulation is already failing and therefore fat embolism may be an added danger to the seriously ill patient.

*Acute renal failure*, from necrosis of tubular epithelium, is likely to occur if shock, with severe circulatory collapse, persists for many hours, for the renal circulation is then reduced to a small fraction of the low cardiac output, and thus severe renal ischæmia results. The principal causes are: (1) Shock, when transfusion has been delayed or inadequate; (2) mismatched transfusion, with intravascular hæmolysis; (3) abortion; (4) circulatory collapse associated with severe diarrhœa and vomiting, or with hepatic failure. Anuria is rare; usually about 200 c.c. of urine are formed a day, but this is quite different from the highly concentrated urine passed in dehydration, having a specific gravity little different from that of plasma, with a low urea content. The blood urea, therefore, rises rapidly.

The essential treatment is to replace previous losses without overloading, and to maintain this equilibrium until function returns, as it will do in the majority of cases in two to four weeks. Once the estimated losses have been made good and blood volume has been fully restored, no further loads of fluid or electrolyte must be given to try to promote water or osmotic diuresis, for pulmonary œdema may be induced. A strictly accurate balance of all intake and output and daily weighings are essential. The water intake should be slightly less than the loss by all routes, so that a slight daily weight loss occurs as tissue is consumed. This tissue breakdown must be minimised by giving as much glucose as possible—if necessary, by inserting a polythene catheter far enough into a vein to reach the vena cava, and dripping in 50 per cent. glucose. Little is achieved by adding fat to oral glucose, for it often causes diarrhœa and vomiting and thus further upsets electrolyte balance. It does not further reduce protein breakdown, and its place as a source of calories can be taken by body fat. With this therapy, blood urea and potassium slowly rise as endogenous protein is consumed, and a fatal outcome is often caused by potassium intoxication. Injections of insulin will temporarily lower the level, by causing its deposition with intracellular glycogen. Potassium may also be removed by enemas of ion-exchange resin.

When diuresis commences it may soon reach a considerable degree, with a rapid renal loss of water and electrolyte until normal tubular reabsorption is established again; careful balance estimations and replacement are essential.

ileostomy or biliary fistula, and then if intake is low a significant depletion can soon occur. This may lead to apathy, anorexia, muscular weakness (especially of the legs), ileus and abdominal distension (it has been suggested that this may be one cause of paralytic ileus). Sodium retention and œdema may occur. The possibility of potassium deficiency should always be borne in mind under these circumstances of increased loss, or in any patient with the above symptoms. Trauma, surgical operations and prolonged steroid hormone therapy should be remembered as possible causes of increased potassium loss.

In diagnosis, a plasma potassium level below 4.0 mEq./litre is suggestive, while the ECG may show ST depression, low or inverted T waves, QT prolongation and prominent U waves. But normal ECG and plasma levels do not exclude deficiency, especially in dehydrated patients. Whenever possible, cumulative potassium deficiency should be prevented by increasing the intake of patients in danger of it. Potassium chloride can be given *by mouth* in tablet form; if this is impossible, it may be necessary to give the ion intravenously. This should never be done unless renal function is good, so that any excess can be rapidly excreted, for a rise of plasma potassium to about double its normal level carries a risk of cardiac standstill. The plasma level should be checked regularly, and the infusion, not exceeding 40 mEq./litre in concentration, should not be given faster than 20 mEq./hour. Patients who are to be maintained on intravenous fluids alone for several days should probably be given a moderate intake of potassium (between 50 and 100 mEq./day), so that, should any complication arise which suddenly increases losses, they are less likely to develop a significant deficiency at a time when intake cannot be raised.

**Regime for Patients Maintained on Intravenous Fluids.** Once the blood volume has been restored and any other estimated losses replaced, the following routine is suggested: (1) Water (as 5 per cent. glucose) should be given with care until the urine volume is rising. Two litres (approx. 4, 1-pint bottles) in twenty-four hours should be ample until then; 3 litres may be given later. (2) One pint of normal saline every twenty-four hours. (3) Six grams potassium chloride (80 mEq.) in 2 litres of water (to be included in reckoning (1) above). When the urine volume is low immediately after trauma, this should be halved. Whenever abnormal losses are occurring, twenty-four hour balances should be compiled.

Vomit and liquid faeces can be pooled for estimations of volume, sodium and potassium (normal formed stools can be neglected). An attempt should be made to estimate water loss by the skin: when water replacement is difficult to judge, the patient should be weighed daily, for sudden gross fluctuations in weight are due to changes in body water. Estimations of plasma levels are of little help in management, for these may be kept within normal limits by variations in water balance in spite of deficits, and plasma sodium levels are often low in very sick patients and

not been stabilised should be postponed for two or three days, to allow control of the hyperglycæmia. If immediate surgery is necessary, an intravenous infusion of 10 per cent. glucose should be started at once and 20 units of soluble insulin given subcutaneously. This dose can be increased six hours later if the glycosuria is still very heavy. If there is ketosis or precoma, the initial dose can be 40 or 50 units, and large doses must be given six-hourly until the ketosis is abolished. Particular care must be taken to restore normal fluid and electrolyte balance, for these patients may be very dehydrated, with low blood volume and blood pressure. Large doses of glucose and insulin may precipitate hypokalaemia, so that potassium should be given intravenously. The management of these patients is made easier if twenty-four-hour balances of fluid and electrolyte exchanges are compiled.

*Only strictly emergency procedures should be carried out on a patient in coma, for which the usual medical routine treatment is given.*

## NORMAL VALUES

Blood volume	. . .	8- 9.5 per cent. of body-weight
Extracellular fluid volume	. . .	18-25 " " "
Total body water	. . .	55-65 " "
Plasma sodium	. . .	135-145 mEq./litre "
" potassium	. . .	4- 5 " "
" bicarbonate	. . .	25- 28 " "
" chloride	. . .	100-105 " "
" protein	. . .	6.0-7.5 gm. per cent.
" albumin	. . .	4.0-5.5 " "

1 gm. NaCl = 17 mEq. of Na

1 gm. KCl = 13 mEq. of K

1 litre normal saline = 145 mEq. Na

= 8.5 gm. NaCl

*Average Composition of Secretions of Alimentary Tract*

	Na mEq./l.	K	Cl
Stomach . . . . .	60	9	84
Ileostomy (recent) . . .	130	11	116
Ileostomy (adapted) . . .	46	3	21
Bile . . . . .	150	5	100
Pancreatic juice . . . .	140	5	77

## SURGERY IN DIABETICS

Little change in the routine of a diabetic patient, already stabilised on insulin, is necessary when surgery is contemplated. Starvation, dehydration and electrolyte depletion must be avoided. The anaesthetic may be local, spinal, or general (provided that chloroform or ethyl chloride are not used).

Minor procedures can be carried out without alteration of the normal diet and insulin. In preparation for a major procedure, an injection of soluble insulin, equal to one-quarter of the total daily insulin dosage, should be given six hours preoperatively, followed by a light carbohydrate meal and a glucose drink. This injection is repeated just before the operation, when an intravenous infusion of 10 per cent. glucose should be started and continued at the rate of a litre every six hours. Injections of soluble insulin are repeated six-hourly, the quantity being increased according to the degree of glycosuria. As soon as the patient can take food, meals of roughly equal calorie value can be given at regular three- or six-hourly intervals throughout the day, and the six-hourly injections can be replaced by the normal schedule when the need for increased insulin has disappeared.

Wherever possible, surgical procedures in the diabetic patient who has

cells are agglutinated by the serum of rabbits which have been immunised against the Rhesus monkey. An Rh. negative patient, if given Rh. positive blood, will be immunised and may react against this if it is given again (even the first transfusion may be incompatible). An Rh. negative mother may be sensitised by an Rh. positive foetus (the antigen being derived from the father) or she may have formed the antibody after a previous transfusion. In the former case, the first child often escapes, but there is an increasing risk with successive pregnancies of the offspring being born with hæmolytic disease.

A number of sub-groups of the Rh. system have been found, and also a number of rarer blood groups. These are of little importance to the clinician unless the patient is likely to receive a large number of transfusions. Otherwise, the A B O and Rh. grouping of the patient is sufficient. But wherever possible, a technique for cross matching, which will detect incompatibility even with the rarer antigens, should be used (*vide infra*). There are probably few instances in which this cannot be done.

**Cross Matching.** The cells from a few drops of blood from the sampling tube attached to the bottle of blood are washed in a little saline and spun down. The saline is pipetted off; the procedure is repeated and then the cells are suspended in about twenty times their volume of saline. To a drop of this suspension, in each of two small tubes, is added a drop of the recipient's serum. After gentle mixing one tube is allowed to stand and the other incubated at 37° C. for one and half hours. The supernatant fluid in the latter tube is withdrawn and a drop of 20 per cent. bovine albumin added without disturbing the cells. The tube is then incubated for another half an hour. The cells at the bottom of both tubes are now spread on two slides and examined under the microscope for agglutination. If none is seen, the compatibility should be written on the label of the bottle at once, and the patient's name and ward are stated. The nursing staff must never use a bottle of blood without checking this record on the label. Blood should not be used if it has been frozen; and on being brought from the refrigerator the blood should be warmed gently in water at body temperature. Hæmolysis obvious to the naked eye should lead to the rejection of the bottle. Once used, the bottle should be kept without rinsing for twenty-four hours, as the blood left in it can be used for investigation of any reaction that occurs.

**Technique of Transfusion.** Forearm veins, away from joints, should be used whenever possible, and the tubing strapped to the arm so that the patient can move the limb. A blunt-ended needle with a sharp stilette, such as the West Middlesex needle, should be inserted directly into the vein and, after the stilette has been withdrawn, the needle is pushed well along the vein. The drip tubing, previously filled by running blood through, with the needle level with the drip chamber, to exclude air, is attached and the flow regulated by a screw clamp placed at the lower end of the tubing close to the arm. This minimises the risk of air being sucked

## CHAPTER 109

### BLOOD TRANSFUSION

THE technique of transfusion has been so greatly simplified in the last few years and the Blood Transfusion Service is so efficient that there is a tendency to transfuse patients too readily. It is tempting to try and hasten the recovery and discharge of a patient with a moderately severe anaemia by a blood transfusion, but before doing so one should ask "Is this transfusion really necessary?" For there is a definite risk attached to this procedure, and to allow it to become a substitute for the investigation and correct treatment of anaemia is bad practice.

After acute blood loss, the immediate need of the patient is the restoration of blood volume. Plasma or a plasma substitute can be given for this purpose, while arrangements for careful grouping and cross matching are made. There are very few occasions when time does not allow for the technique of direct matching described below.

Chronic anaemia rarely calls for transfusion—aplastic and hæmolytic anaemia apart. The patient with pernicious anaemia or severe hypochromic anaemia already has a high plasma volume and may have myocardial deterioration; then the rapid infusion of blood may cause overloading and acute pulmonary oedema. If such patients appear to be in immediate need of more hæmoglobin, then a slow transfusion of packed cells is given.

On the other hand, there are occasions in surgical practice when the value of transfusion is greater than that of the increased oxygen-carrying capacity it confers. Examples are the treatment of chronic wound sepsis and the preparation of a severe case of ulcerative colitis for colectomy. Again, there is increasing evidence that early and adequate replacement of blood lost after injuries and during major operations minimises the metabolic disturbance that follows.

**The Blood Groups.** People can be divided into four groups according to the presence or absence of specific antigens in the cells (known as A and B), and of anti-A and anti-B antibodies in the plasma. If the cells of a patient contain an antigen, the corresponding antibody is absent from the plasma; if an antigen is absent the antibody is present. The groups are named according to the antigens present in the cells: A, B, AB and O. Forty-five per cent. of Europeans fall into the first and the last group. Group O people are the "universal donors" whose cells contain neither antigen (both antibodies being present in the plasma).

Other antigens and antibodies exist, capable of producing agglutination. Eighty-five per cent. of people are said to be Rh, positive, for their red





in anywhere. With the average drip chamber, forty drips per minute corresponds to a pint every four hours. The bottle must be changed before air enters the tubing, or the level of blood in the drip chamber will fall. This is absolutely imperative if positive pressure is applied to the bottle with a Higginson's syringe, or fatal air embolism may occur.

*Transfusion Reactions.* The patient should always be watched for a time when a new bottle of blood has been started. A B O incompatibility usually produces a severe, acute reaction with lumbar pain and often a rigor. Reactions from other incompatibilities may be less dramatic. In an anaesthetised patient, a rise in pulse rate or fall of blood pressure may be the only signs, so that both should be recorded every five minutes for fifteen minutes.

Rarely, a reaction is due to destruction of the patient's cells by the donor's plasma, either when the latter contains very high titres of anti-A or anti-B antibodies (such donors should be known in advance, but sometimes these titres have been raised by recent inoculations containing horse serum), or when massive transfusions are being given.

*In vivo* agglutination is followed by hæmolysis and jaundice. The most serious complication is *acute renal tubular necrosis*. There may be a short period of falling urine volume and hæmoglobinuria, after which oliguria and rising blood urea are evident. Anuria is rare. It is customary, as soon as it is realised that a hæmolytic transfusion reaction has occurred, to give a large alkali load (e.g. 300 ml. of 3 per cent. sodium citrate i.v.) to make the urine alkaline. It is doubtful whether this does anything to prevent the development of acute tubular necrosis and it has the disadvantage of producing an abnormality in the composition of extra-cellular fluid at a time when the essential treatment is to try and keep it as normal as possible.

*Sensitisation* to the Rh. antigen may occur if Rh. positive blood is given to an Rh. negative patient, though there is a good deal of variation in the response. Patients with acquired hæmolytic anæmia, especially those who have often been transfused, may be extremely difficult to match. This task should be left to the Blood Transfusion Service, for very many bottles may have to be tested by a scrupulous technique if reactions are to be avoided.

*Febrile reactions* are common. Many are due to faulty cleaning of the apparatus and the use of old tubing. They are less common when transfusion is given slowly and the patient is kept warm.

*Allergic reactions*, with urticaria, angioneurotic oedema, asthma, vomiting, and even a shock-like state may occur. These may be mitigated by adding an ampoule of an intravenous antihistamine (e.g. Piriton) to the bottle of blood.

*Diseases transmitted by transfusion* include hepatitis, syphilis and malaria. The virus of hepatitis may persist in the blood stream for years after an attack of jaundice; since it is not known what the maximum

time may be, it is wise to exclude anyone who has ever had jaundice from giving blood. The chances of transmitting syphilis are very small, but a routine W.R. should be done on prospective donors and positive reactors excluded. Malaria can be transmitted for many years after an attack, though modern therapy has greatly reduced the chances of this occurrence. This is still a problem in the tropics however.

**The Use of Plasma.** During the last war, the use of dried plasma saved countless lives. Unfortunately, when plasma from a large number of donors is mixed, there is a real possibility of every bottle from such a large pool carrying the virus of serum hepatitis, if only one of the donors was infected. Plasma from small pools of, say, half a dozen selected donors is much safer. But there is no known way to inactivate the virus, so the risk remains a real one. The use of human albumin solution, which can be heat treated, is free from this danger. Prepared from Red Cross donors, this is available in convenient packs in the U.S.A., each containing 1 unit of 25 gm. albumin.

Substitutes for plasma include polyvinylpyrrolidone (Plasmosan), gum acacia and Dextran. The first is, unfortunately, taken up by the reticulo-endothelial system with uncertain results and is, therefore, better avoided. Dextran is prepared from the bacterial breakdown of high molecular weight polysaccharides. It contains molecules similar in size to those of plasma proteins, but they are not uniform. The smaller molecules are excreted by the kidneys, but the substance is a convenient, safe plasma-expander for use in an emergency.

## CHAPTER 110

### ANTIBIOTIC THERAPY

AMONG the infections which are of practical importance to surgeons, there are a few for which sulphonamide therapy is convenient and effective. *Sulphonamide powder*, with or without penicillin, is a convenient dry local application for an infected surface, but carries a real risk of sensitisation, especially if the patient is exposed to sunlight. Its use should, therefore, be restricted to a few days.

*Urinary tract infections* often respond rapidly to sulphonamides, which may also be used when therapy with other antibiotics has led to the emergence of resistant strains (especially Gram-negative ones). Since some interstitial infections of the kidney, bladder or prostate probably always complicates urinary infections, the dosage used should be the same as that for general ones, namely, 2 to 4 gm. followed by 1 gm. four-hourly for seven days. There is little advantage to be gained by using drugs other than those which are so soluble that urinary complications are rare, namely, Sulphamethazine, Gantrisin, or a mixture such as Sulphatriad. Occasionally, a chronic urinary infection associated with hydronephrosis or an obstruction which cannot be relieved, and which occurs after each course of treatment with antibiotics, may be controlled by regular small doses of sulphonamide.

The prophylactic use of these drugs has been recommended when any instrumentation of the urinary tract is necessary, but this practice has little to commend it as a routine. The main protections against infection are absolute sterility and the avoidance of any instrumentation that is not absolutely necessary; nothing should be encouraged which might allow a relaxation of these standards or a false sense of security.

Sulphonamides may also be used to reduce the bacterial flora of the bowel before operations. Sulphasuxidine or sulphathalidine may be given in doses of 0.25 gm./kg. daily, but a much greater effect is obtained by giving Neomycin (0.05 gm./kg. daily) orally as well. This should be combined with castor oil, enemata and a low-residue diet for forty-eight hours before operation, so that resistant flora have little time in which to multiply.

Penicillin is the safest and most reliable antibacterial agent available. It is a bactericidal drug, active against most Gram-positive organisms (including hæmolytic streptococci and many non-hæmolytic strains, many staphylococci, pneumococci and some clostridia), meningococci, gonococci and *B. anthracis*, nearly all leptospira, most strains of actinomyces, and *Treponema pallidum*. Unlike the sulphonamides, it is active in the

presence of pus. After injection, little reaches the cerebrospinal fluid or pleural space, but it penetrates peritoneal and synovial exudates. Dry crystalline sodium penicillin (penicillin G) is stable for three years (though solutions deteriorate in a few hours at room temperature). The principal preparations available are :

*Penicillin G*, for intermittent intramuscular injection. A single large dose produces a high blood concentration which ensures the greatest possible penetration of the infected tissues. This should be repeated when the bacteria are again multiplying rapidly, after the level of penicillin in the tissues has fallen. A dosage of 500,000 units twelve-hourly is highly effective.

*Long-acting preparations*, maintaining effective concentrations for twenty-four hours or more after a single injection. These include procaine penicillin, effective for twenty-four hours, and benzathine penicillin, effective for several days. Mixtures of two or more varieties of penicillin are available.

*Oral Preparations.* These include benzathine penicillin, penicillin G with probenecil (which, by blocking the renal excretion of the drug, prolongs the period of effectiveness) and phenoxymethyl-penicillin (penicillin V). These preparations produce effective blood levels if given half an hour before meals.

*Reactions to penicillin* may occur even in patients receiving the drug for the first time, and are not uncommon after repeated courses. Urticaria is the most common, and may be widespread. Severe anaphylactic reactions may occur and prove fatal, though they are very rare after oral penicillin or penicillin G. Adrenaline should, therefore, always be available for immediate use, and an inquiry should be made as to asthma, allergy and abnormal sensitivity to drugs before therapy is begun.

*Principles of Therapy.* Wherever possible, the sensitivity of the infecting organism should be determined in the laboratory. No resistant strains of hæmolytic streptococci, pneumococci, gonococci or *Treponema pallidum* have been reported. An increasing number of strains of staphylococci—as many as 50 per cent. of those found in infections in some general hospitals—are being found to be resistant. Mild and moderately severe infections with sensitive organisms can be effectively treated by oral penicillin. Penicillin V is said to cause few reactions and is available in tablet or capsule form. Adults may be given 60 mg. four-hourly by day, with a double dose in the evening, half an hour before meals. Children should be given one-half, and infants one-quarter of this dosage. Therapy should be given for a minimum of five days. It has been shown that streptococci may not be eliminated from the upper respiratory tract for ten days.

Severe infections are most effectively treated by twelve-hourly injections of penicillin G. Not only is this probably the most efficient way of using a bactericidal drug, but it is the one least likely to be followed by

## CHAPTER 110

### ANTIBIOTIC THERAPY

AMONG the infections which are of practical importance to surgeons there are a few for which sulphonamide therapy is convenient and effective. *Sulphonamide powder*, with or without penicillin, is a convenient dry local application for an infected surface, but carries a real risk of sensitisation, especially if the patient is exposed to sunlight. Its use should therefore, be restricted to a few days.

*Urinary tract infections* often respond rapidly to sulphonamides, which may also be used when therapy with other antibiotics has led to the emergence of resistant strains (especially Gram-negative ones). Since some interstitial infections of the kidney, bladder or prostate probably always complicates urinary infections, the dosage used should be the same as that for general ones, namely, 2 to 4 gm. followed by 1 gm. four-hourly for seven days. There is little advantage to be gained by using drugs other than those which are so soluble that urinary complications are rare, namely, Sulphamethazine, Gantrisin, or a mixture such as Sulphatriad. Occasionally, a chronic urinary infection associated with hydronephrosis or an obstruction which cannot be relieved, and which occurs after each course of treatment with antibiotics, may be controlled by regular small doses of sulphonamide.

The prophylactic use of these drugs has been recommended when any instrumentation of the urinary tract is necessary, but this practice has little to commend it as a routine. The main protections against infection are absolute sterility and the avoidance of any instrumentation that is not absolutely necessary; nothing should be encouraged which might allow a relaxation of these standards or a false sense of security.

Sulphonamides may also be used to reduce the bacterial flora of the bowel before operations. Sulphasuxidine or sulphathalidine may be given in doses of 0.25 gm./kg. daily, but a much greater effect is obtained by giving Neomycin (0.05 gm./kg. daily) orally as well. This should be combined with castor oil, enemata and a low-residue diet for forty-eight hours before operation, so that resistant flora have little time in which to multiply.

Penicillin is the safest and most reliable antibacterial agent available. It is a bactericidal drug, active against most Gram-positive organisms (including hæmolytic streptococci and many non-hæmolytic strains, many staphylococci, pneumococci and some clostridia), meningococci, gonococci and *B. anthracis*, nearly all leptospira, most strains of actinomyces, and *Treponema pallidum*. Unlike the sulphonamides, it is active in the

intestinal disturbance, but diarrhoea and, less often, vomiting may occur with any of these drugs. The dosage is 250 mg. in a capsule, given with milk after a meal, four times daily. (For children, 4 mg. per kg. body-weight, q.i.d.) Twice this amount can be given in very severe infections, but little increase in the blood concentration is found after larger quantities. Special preparations of tetracycline are available for intravenous injection (500 mg. twelve-hourly, given in twenty-five minutes or in an intravenous drip) or intramuscular injection (100 mg. from two to six injections in twenty-four hours).

Erythromycin has an antibacterial activity similar to that of penicillin, and may be given by mouth (0.5 gm. six-hourly). It may cause vomiting and diarrhoea. Many staphylococci resistant to penicillin are sensitive to erythromycin.

**Complications of Antibiotic Therapy.** Apart from the toxic effects mentioned above, there are two important considerations to be borne in mind when using antibiotics, namely, the occurrence of drug resistance and of superinfections. As a result of the increasing use of antibiotics during the last few years, more strains of pathogenic bacteria are being found to be resistant to their action, particularly in hospital practice. The most striking example is the rise in the number of penicillin-resistant strains of staphylococci, due to the increase in the number able to form penicillinase. These strains may be sensitive to the broad spectrum antibiotics or to erythromycin (the latter is so valuable for these infections that its use is best confined to them). No strains of *Strep. pyogenes*, *pneumococci* or *gonococci* have been found to be resistant to penicillin.

All bacteria can become resistant to streptomycin, and this can occur with remarkable rapidity during treatment. In the therapy of tuberculosis, the development of resistant strains can be largely prevented by giving P.A.S. at the same time, but in other infections resistant strains can develop so rapidly that the drug should never be given without a clear indication, and then only for a few days. Prolonged therapy not only leads to the development of a resistant infection, but may also cause resistant bacilli to develop in an unsuspected tuberculous focus. Strains of organisms resistant to one member of the tetracycline group are usually resistant to all of them. Gram-negative bacilli resistant to the tetracyclines are usually resistant to chloramphenicol; this does not apply to Gram-positive organisms.

*Superinfections* may occur during broad spectrum antibiotic therapy and prove to be highly dangerous complications. The normal flora of the alimentary tract may be replaced by freely growing fungi or resistant bacteria; the most important is *Candida albicans* which may multiply in the mouth and throat (spreading to the lungs) and in the vagina. A severe, often fatal, staphylococcal enteritis may develop (these complications are much less common after chloramphenicol, which produces much less change in the normal flora of the bowel). The stools should, therefore,

severe reactions. Infections with moderately resistant organisms may be treated by larger doses, given more frequently. It must be remembered that it is often more effective to use large doses of a bactericidal drug than a bacteriostatic one to which the organism may be more sensitive on laboratory testing.

Streptomycin is a bactericidal drug, stable in solution, and is mainly excreted in the urine. Almost none is absorbed from the intestine. It is active against *Mycobacterium tuberculosis* and an important range of Gram-positive and Gram-negative bacilli including some strains of *Proteus* and *Pseudomonas*.

There is some synergism with penicillin and with the sulphonamides in many infections. Many bacteria become resistant to streptomycin very rapidly, so that it should rarely be given for more than five days, except in the treatment of tuberculosis. There is little risk of any toxic effect in this time, though urticaria and drug fever may occur, and nurses handling the drug may become sensitised. Albuminuria and cylindruria are common, but are not thought to be of any serious significance.

For urinary infections, intramuscular injection of 1 gm. daily for five days is recommended though resistant strains of *Ps. aeruginosa* may require twice this dose. The urine should be kept alkaline by giving potassium citrate, as the action of the drug is then more efficient. For intestinal infections the drug may be given by mouth (0.25 gm. q.i.d.) with a sulphonamide.

*Polymyxins B. and E.* are bactericidal drugs, active against Gram-negative organisms, particularly *Ps. pyocyanea*. An average dose is 2 mg./kg. body-weight daily for one to two weeks, given by intramuscular injection. Occasionally there is albuminuria and reduction of renal function. These drugs form a very safe local application, with little risk of sensitisation, and can be applied to burns as a 0.1 per cent. cream. This can be combined with Neomycin or Bacitracin when activity will be extended against a wide range of organisms.

**Broad Spectrum Antibiotics.** *Chloramphenicol* has the advantages of being very stable and of causing little change in the intestinal flora. The palmitate is agreeable to children. But it cannot be given parenterally, and its use is occasionally followed by the development of aplastic anaemia. It is largely excreted in the urine and is effective in a wide range of urinary infections. It is the only effective drug against *S. typhi*.

**The Tetracyclines.** *Chlortetracycline* (Aureomycin), *Oxytetracycline* (Terramycin) and *Tetracycline* (Achromycin, Tetracyclon) are all active against most pathogenic cocci and many bacilli, whether Gram-positive or negative. On the whole, there are few differences between the antibacterial activities of these drugs (which are bacteriostatic in action), but some variations have been reported. Tetracycline itself is found in higher concentration in the C.S.F. than the other two drugs, and appears in pleural and peritoneal exudates. It tends to cause the least gastro-

## SECTION XIV

### CHAPTER III

# THE PRINCIPLES AND SURGICAL APPLICATIONS OF RADIOTHERAPY

by

H. CLIVE SIMS

**Physical Principles.** Radiotherapy is treatment by X-rays or radioactive substances. *X-rays*, discovered by Roentgen in 1895, are electromagnetic waves, like light rays or wireless waves. Their wavelength is extremely short, varying between 1.0 and  $0.001 \times 10^8$  cm. They are invisible to the eye and have the power of penetrating matter, the shorter the wavelength, the greater the penetrating power. X-rays are produced in a high-voltage vacuum tube, by accelerating a stream of electrons against a metal target. When these high-speed electrons strike the atoms of the target, part of their kinetic energy is transformed and emitted as X-rays. The higher the accelerating voltage, the shorter the wavelength, and therefore the greater the penetrating power of the X-rays produced.

For the treatment of superficial lesions, e.g. of the skin, "*superficial X-ray therapy*," i.e. X-rays generated at a comparatively low voltage of 50,000 to 100,000 volts are used, as deep penetration is neither required nor desirable. For lesions situated deep in the human body, "*deep X-ray therapy*," i.e. X-rays generated at 200 to 300 kv. are generally used. In recent years "supervoltage" X-ray apparatus of 1 to 2 million volts has been constructed, and X-ray therapy machines of novel design and capable of producing X-rays of 10, 20 and over 100 million volts are now being developed, such as the *betatron* and the *linear accelerator*. These new developments are still in the experimental stage, and it is not yet possible to assess their scope in clinical radiotherapy.

**Radioactive Substances.** Radioactivity was first discovered by Becquerel in 1896 in uranium ore, from which, in 1898, Pierre and Marie Curie isolated the first radioactive element, which they called *radium*. A radioactive element is one whose atoms are unstable, i.e. gradually they "break up" spontaneously, in contrast with the majority of the natural elements, whose atoms are stable. When breaking up, the atoms of radioactive elements emit part of their nuclear energy as three main types of radiation: (a) Alpha rays (positively charged particles); (b) beta rays (negatively charged particles); (c) gamma rays (electromagnetic waves corresponding to X-rays of a million volts or more). The rays mainly



be cultured if severe diarrhoea occurs during oral antibiotic therapy, and the latter stopped at once if a pure growth of resistant staphylococci is reported. The sensitivity of the organisms must be determined: frequently erythromycin is effective. The interference with the normal synthesis of vitamins of the B group, when the normal flora are suppressed, must also be remembered.

**General Principles of Antibiotic Therapy.** Antibiotics should never be given indiscriminately. Wherever possible, the sensitivity of the infecting organism should be determined: this is especially true of urinary and wound infections, in which immediate therapy is rarely imperative. Once the decision has been made to administer an antibiotic, it must be given in really effective dosage. The bactericidal drugs are probably most effective when given intermittently in large doses, for their action is greatest when the bacteria are actively multiplying. For this reason, to combine them with a bacteriostatic drug may actually reduce their efficiency. They may be more effective than bacteriostatic drugs even when the infecting organisms are more sensitive to one of the latter *in vitro* (the best example of this is the treatment of subacute bacterial endocarditis, in which penicillin is far superior in its results to the broad spectrum antibiotics). Other things being equal, penicillin is always to be preferred to other antibiotics, for the complications of its use (especially crystalline or oral penicillin) are less dangerous.

Chloramphenicol is probably best avoided except for infections with *S. typhi*, or when it is particularly important to avoid gross alteration in the bowel flora. Erythromycin is best reserved for use against penicillin-resistant staphylococci (this advantage would soon disappear if it were to be used indiscriminately). The enthusiastic use of any new antibiotic is to be deprecated before careful laboratory and clinical trial have defined its sphere of greatest usefulness.

## SECTION XIV

### CHAPTER 111

# THE PRINCIPLES AND SURGICAL APPLICATIONS OF RADIOTHERAPY

by

H. CLIVE SIMS

**Physical Principles.** Radiotherapy is treatment by X-rays or radioactive substances. X-rays, discovered by Roentgen in 1895, are electromagnetic waves, like light rays or wireless waves. Their wavelength is extremely short, varying between 1.0 and  $0.001 \times 10^8$  cm. They are invisible to the eye and have the power of penetrating matter, the shorter the wavelength, the greater the penetrating power. X-rays are produced in a high-voltage vacuum tube, by accelerating a stream of electrons against a metal target. When these high-speed electrons strike the atoms of the target, part of their kinetic energy is transformed and emitted as X-rays. The higher the accelerating voltage, the shorter the wavelength, and therefore the greater the penetrating power of the X-rays produced.

For the treatment of superficial lesions, e.g. of the skin, "*superficial X-ray therapy*," i.e. X-rays generated at a comparatively low voltage of 50,000 to 100,000 volts are used, as deep penetration is neither required nor desirable. For lesions situated deep in the human body, "*deep X-ray therapy*," i.e. X-rays generated at 200 to 300 kv. are generally used. In recent years "*supervoltage*" X-ray apparatus of 1 to 2 million volts has been constructed, and X-ray therapy machines of novel design and capable of producing X-rays of 10, 20 and over 100 million volts are now being developed, such as the *betatron* and the *linear accelerator*. These new developments are still in the experimental stage, and it is not yet possible to assess their scope in clinical radiotherapy.

**Radioactive Substances.** Radioactivity was first discovered by Becquerel in 1896 in uranium ore, from which, in 1898, Pierre and Marie Curie isolated the first radioactive element, which they called *radium*. A radioactive element is one whose atoms are unstable, i.e. gradually they "break up" spontaneously, in contrast with the majority of the natural elements, whose atoms are stable. When breaking up, the atoms of radioactive elements emit part of their nuclear energy as three main types of radiation: (a) Alpha rays (positively charged particles); (b) beta rays (negatively charged particles); (c) gamma rays (electromagnetic waves corresponding to X-rays of a million volts or more). The rays mainly

be cultured if severe diarrhoea occurs during oral antibiotic therapy, and the latter stopped at once if a pure growth of resistant staphylococci is reported. The sensitivity of the organisms must be determined: frequently erythromycin is effective. The interference with the normal synthesis of vitamins of the B group, when the normal flora are suppressed, must also be remembered.

**General Principles of Antibiotic Therapy.** Antibiotics should never be given indiscriminately. Wherever possible, the sensitivity of the infecting organism should be determined: this is especially true of urinary and wound infections, in which immediate therapy is rarely imperative. Once the decision has been made to administer an antibiotic, it must be given in really effective dosage. The bactericidal drugs are probably most effective when given intermittently in large doses, for their action is greatest when the bacteria are actively multiplying. For this reason, to combine them with a bacteriostatic drug may actually reduce their efficiency. They may be more effective than bacteriostatic drugs even when the infecting organisms are more sensitive to one of the latter *in vitro* (the best example of this is the treatment of subacute bacterial endocarditis, in which penicillin is far superior in its results to the broad spectrum antibiotics). Other things being equal, penicillin is always to be preferred to other antibiotics, for the complications of its use (especially crystalline or oral penicillin) are less dangerous.

Chloramphenicol is probably best avoided except for infections with *S. typhi*, or when it is particularly important to avoid gross alteration in the bowel flora. Erythromycin is best reserved for use against penicillin-resistant staphylococci (this advantage would soon disappear if it were to be used indiscriminately). The enthusiastic use of any new antibiotic is to be deprecated before careful laboratory and clinical trial have defined its sphere of greatest usefulness.

cylindrical applicator used for the treatment of carcinoma of the penis ; (d) *radium beam therapy* (syn. "telecurie therapy," "radium bomb") by means of an apparatus containing a large amount (10 to 20 gm.) of radium used for external radiation of deeply situated lesions. This method is similar in principle to X-ray therapy. *Radon*, a gaseous element, is produced from radium and can be put in small sealed capillary gold tubes, known as radon "seeds." Their use is similar to that of radium needles, e.g. insertion into a carcinoma of the bladder.

*Radioactive isotopes* may be used as "tracers" after introducing them in small quantities into the body. Their presence, metabolism, and "decay" can be measured for physiological, clinical, and other studies, by sensitive radiation detectors, e.g. a Geiger counter. Radioactive isotopes behave physiologically like their non-radioactive counterparts. For instance, radioactive iodine is taken up by the thyroid and may therefore be used in the study of thyroid function (Chap. 69). Similarly, as radioactive iodine, or phosphorus is taken up selectively by brain tumours, the latter can often be localised by a radiation detector after intravenous injection of a radioactive iodine or phosphorus compound.

Isotopes of comparatively long half-life, e.g. cobalt or caesium, are now being used for treatment in a way similar to that of radium therapy, as, for instance, in a "cobalt bomb," for they can be produced in comparatively large quantities in an atomic pile. Another example is radioactive tantalum wire, used for insertion—like radium needles or radon seeds—into a tumour. Other isotopes of comparatively short half-life can be introduced in liquid form into the body for treatment. The beta and gamma radiation is used in these instances. For example, radioactive iodine is used in the treatment of hyperthyroidism or thyroid carcinoma, phosphorus in polycythaemia, and colloidal gold in bladder carcinoma or in the palliative treatment of ascites caused by carcinomatous peritoneal metastasis.

*Dosage.* Individual treatment planning has now become the basis of radiotherapy. As X-rays and the gamma rays are essentially the same, the choice between X-rays and radioactive substances is to a large extent dependent on "technical" considerations, and not on any possible "specific" difference in biological effect between radium and X-rays. The physical dosage unit of radiation is the "roentgen"—"r" for short—or more recently the very slightly different "rad" unit.\* The following examples give an idea of the clinical significance of the "r" unit: 800 "r" applied to a limited area of the skin with a single dose of deep X-rays will produce a definite erythema. The X-ray dose for a small rodent

\* The definition of the "roentgen" is: "The quantity of X-ray or gamma radiation, such that the associated corpuscular emission per 0.001293 gm. of air produces, in air, ions carrying one electrostatic unit of quantity of electricity of either sign."

The definition of the "rad" is: "The absorbed dose of any ionising radiation is the amount of energy imparted to matter by ionising particles per unit mass of irradiated material at the place of interest. The rad is the unit of absorbed dose and is 100 ergs per gramme."

used in radium therapy are the gamma rays, but with radioactive isotopes (*vide infra*) beta rays are also used.

An atom in its simplified form can be regarded as consisting of a nucleus, composed of positively charged particles (protons) and neutral particles (neutrons) and surrounded by a "planetary system" of negatively charged particles (electrons) revolving around the nucleus and counterbalancing the positive charge of the protons. The number of protons in the nucleus of an element determines its "atomic number" in the periodic system of elements, and therefore the chemical character of the element. The combined number of protons and neutrons determines its "atomic weight." It is therefore possible for chemical elements, defined by the number of protons in their atomic nuclei, to exist in several forms of different atomic weights, according to the number of additional neutrons in the nucleus. In other words, elements may exist as various "isotopes." An isotope therefore indicates a variety of a given chemical element, possessing the same "atomic number" and therefore the same chemical characteristics, but different atomic weight. An isotope is not necessarily radioactive. However, if the number of the additional neutrons in the nucleus of an isotope element is disproportionate, the nucleus becomes unstable, and therefore has a tendency to break up, i.e. the isotope is radioactive. With the development of atomic science, several hundred isotope elements have become known, mostly radioactive isotopes of natural elements. The majority of these are produced artificially, in the "atomic pile."

The spontaneous "decay" of radioactive elements obeys definite physical laws. It is generally expressed in "half-life time," i.e. the time in which the strength of a given quantity of the radioactive element falls to half its value. This varies widely, according to the element, from a fraction of a second to thousands of years, but it is fixed for a given element. Thus among radioactive elements used in clinical surgery, the half-life of radium is 1,600 years, of radon 3.85 days, of radioactive iodine 8 days, of phosphorus 14 days, of gold 2.7 days, of tantalum 120 days, of cobalt 5.3 years, of caesium 33 years. Radium, a metallic element, is for clinical purposes generally used in the form of radium sulphate, a powder, which is put in sealed containers—usually hollow platinum needles or tubes—of various amounts of radium, expressed in milligrams. The platinum absorbs the unwanted alpha and beta rays, and radium therapy is therefore essentially gamma ray therapy. There are four main methods of radium treatment: (a) *Interstitial radium implantation*, in which radium needles are inserted into the tissues, e.g. in the treatment of carcinoma of the tongue; (b) *intracavitary treatment*, in which radium containers are inserted into a cavity, e.g. into the uterine canal and vagina in the treatment of carcinoma of the cervix; (c) *radium mould treatment*, consisting of the use of individually constructed surface applicators, usually of plastic material, loaded with radium tubes, e.g. ■

dangers of radiation and the importance of adequate protection for radiation workers. This is comparatively easy with X-ray therapy by the use of protective walls, of lead or barium bricks. The handling of radium requires special skill: the radium storage safe, the manipulation benches, the theatre radium trolley, etc., must have adequate lead protection. The handling of radioactive isotopes, especially in liquid form, with its danger of being splashed or inhaled, requires, in addition to lead protection, the use of a chemical laboratory type "fume cupboard." The disposal of radioactive urine and faeces of patients treated by radioactive isotopes also requires special precautions. Regular safety checks are carried out routinely in all radiotherapy departments. The maximum permissible dose of radiation, which radiation workers may receive without any ill effects, has been internationally agreed as 0.3 roentgen per week.

**Applications in Surgery.** Although the main use of radiotherapy in surgery is in malignant disease, it has important applications in the treatment of various *non-malignant conditions* (Fig. 679). In the treatment of chronic inflammatory diseases radiotherapy has been largely superseded by antibiotics, but it is still of value, e.g. in tuberculous adenitis, actinomycosis and chronic parotitis. Other amenable conditions are *keloid scars*, the larger "strawberry-type" of *haemangioma* so frequent

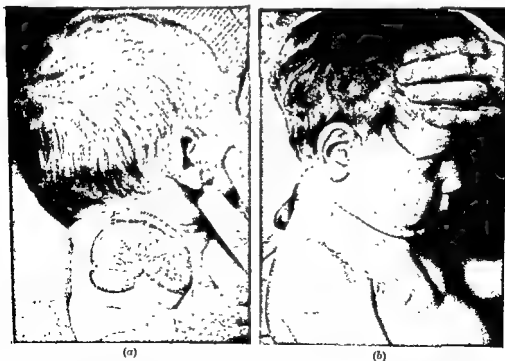


FIG 679. (a) Large haemangioma of the right shoulder. Male baby, three months old. Lesion present at birth, growing rapidly, becoming ulcerated and bleeding in centre. (b) After X-ray Therapy. Two years later.

ulcer may be 2,000 "r" in a single exposure. The radium dose for a carcinoma of the tongue may be 7,000 "r" in one week. The X-ray dose given post-operatively for a carcinoma of the breast may be 4,000 "r" in four weeks.

**Biological Effects of Radiations.** The detailed mechanism of the effect of radiations on the living cells and tissues of the body is not yet fully understood. It is generally accepted that radiation produces ionisation in the cell, i.e. the "knocking-out" of electrons from the atomic structure. This, in turn, causes a disruption of the complicated molecular structure of the various components of the living cell, and, if severe enough, leads to the death of the cell. If the radiobiological damage is only slight, the cell will gradually recover. The chromosomal structure of the nucleus is particularly vulnerable to radiation. Müller, in 1928, showed that irradiation of germ cells could produce an alteration in the small hereditary particles, genes, of the chromosomes and cause a sudden heritable change—a gene "*mutation*." *Radiation genetics* has become an important field of research in recent years, especially in connection with radiation from atomic energy. The direct radiation effect on the cells is probably reinforced by an indirect radiation effect, perhaps through radiobiological damage to the small blood vessels, supplying the irradiated tissues. It can easily be understood therefore that, with the doses used in radiotherapy, there is a latent period between radiation and its visible effect. There is no evidence that radiation can stimulate a malignant tumour to increased activity or cause more rapid metastasis.

The various cells and tissues of the body vary considerably in their sensitivity to radiation and certain tumour cells are more radiosensitive than the surrounding normal cells. Modern radiotherapy is based on these facts. Radiotherapy is not a form of "radio-cautery," but a method of "selective" radiobiological destruction by making use of the differential radiosensitivity between normal and tumour cells. The aim is to deliver a lethal dose to the tumour cells, without killing the normal tissues in the vicinity. The following list, modified from Desjardins, classifies the main types of *normal cells and tissues* according to their *radiosensitivity*, beginning with the most radiosensitive: (a) Lymphocytes and leucocytes, in lymphoid tissue, bone marrow, etc.; (b) spermatogonial cells of the testis and follicular epithelium of the ovary; (c) epithelial cells of the skin and mucous membranes, in particular certain glandular epithelium, such as the salivary glands, the epithelium of the small intestine, the alveolar epithelium of the lungs, the epithelium of the liver, the epithelium of the renal tubules; (d) endothelial cells, of blood vessels, pleura and peritoneum; (e) nervous tissue cells (previously thought to be rather radioresistant); (f) connective tissue cells; (g) muscle cells; (h) bone cells.

**Radiation Protection.** The radiosensitivity of certain cells, e.g. white blood cells, gonads, epithelium of the skin, etc., indicates the potential

dangers of radiation and the importance of adequate protection for radiation workers. This is comparatively easy with X ray therapy by the use of protective walls, of lead or barium bricks. The handling of radium requires special skill; the radium storage safe, the manipulation benches, the theatre radium trolley, etc., must have adequate lead protection. The handling of radioactive isotopes, especially in liquid form, with its danger of being splashed or inhaled, requires, in addition to lead protection, the use of a chemical laboratory type "fume cupboard." The disposal of radioactive urine and faeces of patients treated by radioactive isotopes also requires special precautions. Regular safety checks are carried out routinely in all radiotherapy departments. The maximum permissible dose of radiation, which radiation workers may receive without any ill effects, has been internationally agreed as 0.3 roentgen per week.

**Applications in Surgery.** Although the main use of radiotherapy in surgery is in malignant disease, it has important applications in the treatment of various *non-malignant conditions* (Fig. 679). In the treatment of chronic inflammatory diseases radiotherapy has been largely superseded by antibiotics, but it is still of value, e.g. in tuberculous adenitis, actinomycosis and chronic parotitis. Other amenable conditions are *keloid scars*, the larger "strawberry-type" of *hemangioma* so frequent



FIG. 679 (a) Large hemangioma of the right shoulder. Male baby, three months old. Lesion present at birth, growing rapidly, becoming ulcerated and bleeding in centre. (b) After X-ray Therapy. Two years later.



in babies (the smaller ones often regress spontaneously), and it is at present the most effective treatment for *ankylosing spondylitis*.\* Radiotherapy is of value in the treatment of some endocrine conditions, e.g. radioactive iodine in *hyperthyroidism*.

In the treatment of *malignant disease*, a distinction must be made between curative and palliative treatment. In curative therapy the intention is to eradicate the disease completely, whereas in palliative therapy the aim is to obtain temporary improvement, when the disease is considered to be incurable. Successful palliation means: (a) Relief of distressing symptoms, such as pain, discharge and bleeding; (b) temporary arrest or regression of growth; (c) prolongation of life in a more or less symptom free condition. In a considerable number of these distressing conditions, so frequent in advanced malignant disease, radiotherapy is often the only effective palliative treatment. It must, however, be emphasised that there are occasions when the patient's condition is "beyond radiation." At present there are only two effective methods for the radical treatment of malignant disease, surgery and radiotherapy. Chemotherapy and hormone therapy, so far, have only achieved palliative results. Surgery and radiotherapy are complementary methods of treatment.

Whereas the suitability of a tumour for radiotherapy depends to a large extent on its radiosensitivity, curability by radiotherapy, and radiosensitivity are by no means synonymous. Thus a very radiosensitive tumour which responds rapidly to radiation, may recur and metastasise more rapidly, and therefore be less curable than a less radiosensitive tumour, which responds slowly. The following table, modified from Desjardins and Paterson, shows the relative radiosensitivity of tumours. There is no hard and fast line between the various groups, and the term "radiosensitive" or "radioresistant" does not necessarily imply that a tumour is suitable or unsuitable for radiotherapy.

(1) *Very Radiosensitive Tumours*. Radiotherapy is the main treatment, but is sometimes combined with surgery.

(a) *Reticulo-endothelial Tumours*

Reticulosarcoma.  
Lymphosarcoma.  
Hodgkin's disease.  
Thymoma.  
Ewing's tumour.  
Multiple myeloma.  
Leukæmias.

(b) *Embryonal Tumours*

Seminoma.

\* Recently a few cases of leukæmia, following radiotherapy for ankylosing spondylitis have been reported. Further investigations are, however, necessary before any definite causal connection can be assumed.

Dysgerminoma of ovary.

Wilms' tumour.

Medulloblastoma and neuroepithelioma.

(2) *Moderately Radiosensitive Tumours.* Radiotherapy or surgery are alternative forms of treatment, or are used together.

(a) *Diseases Treated usually by Radiotherapy, sometimes combined with Surgery*

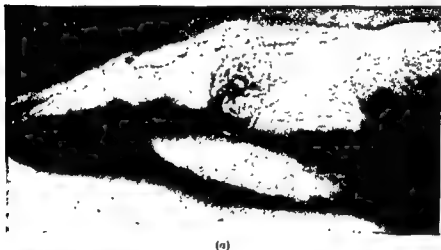
Carcinoma of skin (Fig. 680).

Epithelioma of lip, mouth, tongue, nasal sinuses, pharynx and larynx.

Carcinoma of cervix and vagina.

Hamangioma.

Osteoclastoma.



(a)



(b)

FIG. 680. (a) *Epithelioma of hand.* Male, sixty years old. History of two months, growing rapidly, with raised, hard edge and ulcerated centre. Histology: squamous-celled epithelioma. (b) *After X-ray Therapy.* Four months later.

in babies (the smaller ones often regress spontaneously), and it is at present the most effective treatment for *ankylosing spondylitis*.\* Radiotherapy is of value in the treatment of some endocrine conditions, e.g. radioactive iodine in *hyperthyroidism*.

In the treatment of *malignant disease*, a distinction must be made between curative and palliative treatment. In curative therapy the intention is to eradicate the disease completely, whereas in palliative therapy the aim is to obtain *temporary improvement*, when the disease is considered to be incurable. Successful palliation means: (a) Relief of distressing symptoms, such as pain, discharge and bleeding; (b) temporary arrest or regression of growth; (c) prolongation of life in a more or less symptom free condition. In a considerable number of these distressing conditions, so frequent in advanced malignant disease, radiotherapy is often the only effective palliative treatment. It must, however, be emphasised that there are occasions when the patient's condition is "beyond radiation." At present there are only two effective methods for the radical treatment of malignant disease, surgery and radiotherapy. Chemotherapy and hormone therapy, so far, have only achieved palliative results. Surgery and radiotherapy are complementary methods of treatment.

Whereas the suitability of a tumour for radiotherapy depends to a large extent on its radiosensitivity, curability by radiotherapy, and radiosensitivity are by no means synonymous. Thus a very radiosensitive tumour which responds rapidly to radiation, may recur and metastasise more rapidly, and therefore be less curable than a less radiosensitive tumour, which responds slowly. The following table, modified from Desjardins and Paterson, shows the relative radiosensitivity of tumours. There is no hard and fast line between the various groups, and the term "radiosensitive" or "radioresistant" does not necessarily imply that a tumour is suitable or unsuitable for radiotherapy.

(1) *Very Radiosensitive Tumours*. Radiotherapy is the main treatment, but is sometimes combined with surgery.

(a) *Reticulo-endothelial Tumours*

Reticulosarcoma.  
Lymphosarcoma.  
Hodgkin's disease.  
Thymoma.  
Ewing's tumour.  
Multiple myeloma.  
Leukæmias.

(b) *Embryonal Tumours*  
Seminoma.

\* Recently a few cases of leukæmia, following radiotherapy for ankylosing spondylitis have been reported. Further investigations are, however, necessary before any definite causal connection can be assumed.

Dysgerminoma of ovary.

Wilms' tumour.

Medulloblastoma and neuroepithelioma.

(2) *Moderately Radiosensitive Tumours.* Radiotherapy or surgery are alternative forms of treatment, or are used together.

(a) *Diseases Treated usually by Radiotherapy, sometimes combined with Surgery*

Carcinoma of skin (Fig. 680).

Epithelioma of lip, mouth, tongue, nasal sinuses, pharynx and larynx.

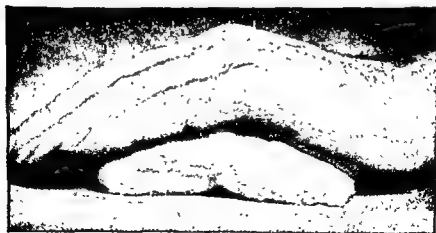
Carcinoma of cervix and vagina.

Hamangioma.

Osteoclastoma.



(a)



(b)

FIG. 680. (a) *Epithelioma* of hand. Male, sixty years old. History of two months, growing rapidly, with raised, hard edge and ulcerated centre. Histology: squamous-celled epithelioma. (b) *After X-ray Therapy.* Four months later.

(b) *Diseases Treated Essentially by Surgery, often combined with Radiotherapy*

Carcinoma of parotid.

Carcinoma of thyroid.

Carcinoma of bronchus.

Carcinoma of breast.

Carcinoma of corpus uteri.

Carcinoma of ovary.

Carcinoma of œsophagus.

Carcinoma of bladder.

Glioma (some, including ependymoma and oligodendroglioma).

Pituitary adenoma.

(3) *Radioreistant Tumours.* Tumours of very low radiosensitivity; radiotherapy is of very little or no value.

Fibrosarcoma and myosarcoma.

Osteogenic sarcoma.

Malignant melanoma (with some exceptions).

Meningioma.

Adenocarcinoma of stomach, intestine, or rectum.

Carcinoma of the prostate.

True teratoma.

**Radiation Reactions.** A knowledge of the normal radiation reactions is important. They are divided into local and general reactions. *Local Reactions.*—When a lesion is irradiated, the normal skin, mucosa and other tissues in the vicinity inevitably receive a certain amount of radiation. The ensuing reaction which is almost imperceptible with the small doses used in the treatment of benign conditions, is quite pronounced with the doses used in the treatment of malignant diseases. These are anticipated reactions and should not be referred to as radiation "burns." The usual skin reaction is an erythema which develops one to two weeks after radiation, and is sometimes followed by temporary epilation. The skin, which returns to normal in about a month, should be kept dry by applications of plain starch powder. Any irritation, e.g. the application of heat, ointments, or adhesive plaster dressings should be avoided. When a high radiation dose is given a patchy moist desquamation, with discrete blisters here and there, may develop in the erythematous area. One per cent. aqueous gentian violet solution is applied to the blisters and the skin protected by a plain dressing. The skin heals rapidly and returns to normal in about a month, but a slight permanent pigmentation or epilation may remain.

A true radiation necrosis—now very rare—is always a sign of an over-dose. It is usually characterised by a necrotic and painful ulcer which may develop quickly, or sometimes a considerable time after irradiation. The ulcer takes a long time to heal, and may sometimes require surgical excision, especially when involving underlying tissues such as bone or

cartilage. It is important not to mistake radiation necrosis for recurrent malignancy. Therefore biopsy may be necessary to avoid further radiotherapy being given in error. Telangiectatic patches, or very rarely a late epithelioma can develop in skin severely damaged by excessive radiation.

The radiation reaction of the mucosa, of the mouth or the intestine, follows a similar pattern. Frequent mouth washes with a mild antiseptic, the prescription of semiliquid food, and the symptomatic treatment of diarrhoea may be indicated during the height of the reaction. *General Reactions*.—These vary with the dose, and the region treated, and may show themselves in the form of general lassitude, loss of appetite, and sometimes "radiation sickness"—a gastro-intestinal sickness, occasionally accompanied by vomiting. The cause of radiation sickness is not quite known. General reactions rarely require treatment and clear up within a few days after completion of radiotherapy, but occasionally a troublesome radiation sickness may require symptomatic treatment by drugs such as pyridoxine (vitamin B<sub>6</sub>), Dramamine, or Avomine.

# INDEX

## A

- Abdomen, acute, in children, 287-296  
 burst, 33, 193-196  
 mass in, in appendicitis, 4, 6  
 in gastric carcinoma, 16  
 pain in, differential diagnosis, 6  
 Abdominal incisions, 191-196  
 wall, agenesis of muscles, 237  
 anterior, abnormalities of, 234-240  
 development of, 234  
 Abscess, amebic, 184  
 anorectal, 163-164  
 apical, 856  
 appendiceal. *See* Appendix, abscess of.  
 areola, 944  
 brain, 659-664  
     bacteriology of, 660  
     diagnosis of, 660-661, 712  
     epilepsy and, 761  
     treatment of, 661-664  
 breast, 944-945  
 Brodie's, 709, 1044-1045, 1050, 1078  
 cold, 874, 1025, 1046-1047, 1093-1094  
 collar stud, 871  
 epidural, 787  
 extradural, 653  
 hepatic, 82  
     diagnosis from hydatid cyst, 84  
 horseshoe, 184  
 ischiorectal, 163  
 mesenteric, 107  
 pelvic, 36, 104-105  
 pelvirectal, 164  
 perianal, 163  
 perinephric, 325-327  
 perineurethral, 416  
 peons, diagnosis from hernia, 208, 216  
 pulmonary, 437-473. *See also* Lung  
     abscess.  
 pyogenic, 118, 163-164  
 retropharyngeal, acute, 863  
     chronic, 863-864  
 subdural, 658-657  
 subphrenic, 103-104  
     diagnosis from hydatid cyst, 84  
     empyema and, 476-477  
     in cholecystitis, 50  
     in peptic ulcer, 36, 38  
     treatment of, 104  
     tuberculous, of neck, 868  
 Acetabulum, fractures of, 1234  
 Achondroplasia, 1033  
 Achromycin, 1316. *See also* Tetracycline.  
 Acid burns, 1298  
 Acoustic tumour, 690  
 Acrocyanosis, 799  
     operative treatment, 796-797  
 Acromegaly, 727, 859, 1068  
 Acromioclavicular joint, arthritis of, 1096  
     dislocations of, 1164  
 ACTH, 724, 725, 930  
     in acquired hemolytic anemia, 97  
     in hypopituitarism, 731  
     in idiopathic thrombocytopenic purpura, 96  
     in pseudomembranous enterocolitis, 116  
 Actinomycosis, diagnosis from carcinoma of  
     colon, 150  
     Crohn's disease, 115  
     malignancy in neck, 1029  
     of face, 828  
     of jaw, 854-856  
     of liver, 82  
     of lung, 474  
     of neck, 864, 870  
     rectal stricture in, 163  
 Adamantinoma, 728  
 Addison's disease, adrenal androgens and  
     corticoids in, 1068  
 Adenitis, gummatous, 1031  
     mesenteric, in children, 290  
     diagnosis, from appendicitis, 288  
     tuberculous, 518, 526, 868, 871-875  
     treatment of, 873-875  
 Adenocarcinoma of appendix, 11  
     of stomach, 14  
 Adenoidectomy, in tuberculous cervical  
     adenitis, 875  
 ADH, 724, 725  
 Adrenal cortex, in metabolic disturbances  
     of bone, 1068  
     glands, 930  
     medulla, tumours of, 936  
 Adrenalectomy, in adrenocortical syn-  
     dromes, 933-934  
     in cancer of breast, 937-938  
     management of, 933-936  
     results of, 936  
     technique of, 934-935  
 Adrenaline, 930  
 Adrenocortical syndromes, investigations,  
     933  
 Adrenocorticotrophic hormone. *See* ACTH.  
 Adrenogenital syndrome, 930-931  
     investigations, 933  
     treatment of, 933-934  
 Aterocoele of neck, 868-869  
 Agnesia, renal, 302  
     urteric, 306  
 Ageusia, 684  
 Agnosia, in parieto-occipital syndrome,  
     715, 716  
 Air embolism, 1014  
 Air-encephalography. *See* Encephalo-  
     graphy, air.  
 Air insufflation, peri-nephric, 325  
 Aldosterone, 725, 930  
 Aldosteronism, 923, 934  
 Alkali burns, 1294  
 Alkaline phosphatase test, of liver function,  
     78

- Alkalosis, respiratory, in traumatic coma, 639-639
- Alveolus, cysts of, acquired, 513-517
- Amigen solutions, 301
- Amnesia, post-traumatic, 635  
retrograde, 635
- Amoebiasis, cutaneous, 183  
hepatic, 184-190  
clinical picture, 185  
complications of, 186  
differential diagnosis of, 186  
investigations, 185-186  
treatment of, 189
- Intestinal, 182-184  
clinical manifestations, 183  
investigations, 183-184  
treatment of, 184  
treatment of, 186-190
- Amoeboma, diagnosis from carcinoma, 150, 176
- Amputation, amniotic, 1036  
in gangrene, 976-977  
in lymphoedema, 1032  
in osteogenic sarcoma, 1073  
in tuberculosis of bone, 1050  
traumatic, of hand, 1289
- Anemia, hemolytic, 96-97  
in gastric carcinoma, 13, 16, 18
- Anesthetics, effect of, in shock, 1304
- Anal canal, anatomy of, 156-157  
carcinoma of, 175-176  
fissure, 162-163  
hematoma, 161  
stenosis, diagnosis from Hirschsprung's disease, 273
- Anderson-Hynes pyeloplasty, for hydro-nephrosis, 342
- Androgens, 725, 1069
- Aneurysms, abdominal, diagnosis from perforated peptic ulcer, 33  
arterial, 984-990  
etiology of, 984-985  
clinical features, 986-987  
complications of, 987  
differential diagnosis of, 987-988  
investigations, 988  
treatment of, 988-990
- arteriovenous, papilloedema and, 671  
atherosclerotic, 983  
berry, 745  
carotid, 746, 747, 749-750, 863  
cirsoid, 984  
congenital, 984  
intracranial, 745-748  
management of, 748-752  
diagnosis from mediastinal cysts and tumours, 518  
dissecting, 984, 985, 988  
diagnosis from pancreatitis, 68  
false, 984  
femoral, diagnosis from hernia, 216  
fusiform, 984  
in arteriosclerosis, 753  
intracranial, 745-752  
surgical treatment, 749-752  
operative exposures, 751-752  
mycotic, 752, 963, 985
- Aneurysms, syphilitic, 985  
varicose, 990
- Angina, Ludwig's, 862  
pectoris, 799  
operative treatment of, 796-797, 800
- Angiography, of aneurysms, 749  
of cerebral vessels, 697  
of epilepsy, 759  
of meningiomas, 709, 710  
of parietal lobe, 717  
of temporal lobe, 712-713  
of tumours of frontal lobe, 707  
vertebral, 699-700
- Angioma, 1018, 1022. *See also* under names of organs.
- Angiorectuloma, of posterior fossa, 721
- Angiosarcoma, 1018
- Ankle joint, abduction injuries of, 1276-1277  
epiphyseal injuries of, 1278  
external rotation injuries of, 1275-1276  
fractures and fracture-dislocations of, 1269-1278  
compression, 1277-1278  
ligamentous injury of, 1274-1275
- Ankyloglossia, congenital, 849
- Anosmia, causes of, 666
- Anterior tibial syndrome, 821-822
- Antibiotic therapy, 1314-1318  
bowel sterilization, 150-151  
complications of, 1317-1318  
general principles of, 1318  
of actinomycosis, 855  
of acute infective arthritis, 1115  
of appendicitis, 8-9  
of biliary disease, 62-63  
of bronchiectasis, 404  
of cerebral abscess, 661-662, 663  
of cholecystitis, 51  
of epidural abscess, 787  
of extradural abscess, 655  
of fractures, 1199, 1247  
of hand infections, 1289, 1291  
of hand injuries, 1285  
of lateral sinus thrombosis, 657  
of meningitis, 659  
of osteomyelitis, 664, 1041  
of pancreatitis, 69  
of parotitis, 838  
of perforated ulcer, 35  
of perinephric abscess, 337  
of peritonitis, 101, 137, 289  
of pulmonary abscess, 465, 470, 473  
of pyelonephritis, 400  
of pyogenic infection of liver, 81, 82  
of soft chancre, 419  
of spinal injuries, 780  
of subdural abscess, 656, 657  
of subphrenic abscess, 104  
of urethritis, 410, 411  
of urinary tract infections, 1314
- Anticoagulant therapy, 1015-1017  
in arterial grafting, 995  
in embolectomy, 984  
of atherosclerosis, 973  
of frost-bite and frozen foot, 981  
of iliofemoral thrombosis, 998, 999



# INDEX

## A

Abdomen, acute, in children, 287-296

burst, 38, 195-196

mass in, in appendicitis, 4, 6

in gastric carcinoma, 16

pain in, differential diagnosis, 6

Abdominal incisions, 191-196

wall, agenesis of muscles, 237

anterior, abnormalities of, 234-240

development of, 234

Abscess, amœbic, 184

anorectal, 163-164

apical, 856

appendiceal. *See* Appendix, abscess of.

areola, 944

brain, 659-664

bacteriology of, 660

diagnosis of, 660-661, 712

epilepsy and, 761

treatment of, 661-664

breast, 944-945

Brodie's, 769, 1044-1045, 1050, 1078

cold, 874, 1025, 1046-1047, 1093-1094

collar stud, 871

epidural, 787

extradural, 635

hepatic, 82

diagnosis from hydatid cyst, 84

horseshoe, 164

ischio-rectal, 163

mesenteric, 107

pelvic, 36, 104-105

perirectal, 164

perianal, 163

perinephric, 335-337

periurethral, 416

psoas, diagnosis from hernia, 208, 216

pulmonary, 457-475. *See also* Lung

abscess.

pyogenic, 118, 163-164

retropharyngeal, acute, 863

chronic, 863-864

subdural, 656-657

subphrenic, 103-104

diagnosis from hydatid cyst, 84

empyema and, 476-477

in cholecystitis, 50

in peptic ulcer, 36, 33

treatment of, 104

tuberculous, of neck, 868

Acetabulum, fractures of, 1234

Achondroplasia, 1033

Achromycin, 1316. *See also* Tetracycline.

Acid burn, 1298

Acoustic tumour, 690

Acrocyanosis, 979

operative treatment, 796-797

Acromegaly, 727, 859, 1068

Acromioclavicular joint, arthritis of, 1096

dislocations of, 1164

ACTH, 724, 725, 930

in acquired hæmolytic anæmia, 97

in hypopituitarism, 731

in idiopathic thrombocytopenic purpura,

96

in pseudomembranous enterocolitis, 116

Actinomyces, diagnosis from carcinoma of

colon, 150

Crohn's disease, 115

malignancy in neck, 1029

of face, 828

of jaw, 854-856

of liver, 82

of lung, 474

of neck, 864, 870

rectal stricture in, 168

Adamantinoma, 723

Addison's disease, adrenal androgens and

corticoids in, 1068

Adenitis, gummatous, 1031

mesenteric, in children, 290

diagnosis, from appendicitis, 288

tuberculous, 518, 526, 868, 871-875

treatment of, 873-875

Adenocarcinoma of appendix, 11

of stomach, 14

Adenoidectomy, in tuberculous cervical

adenitis, 875

ADH, 724, 725

Adrenal cortex, in metabolic disturbances

of bone, 1068

glands, 930

medulla, tumours of, 936

Adrenalectomy, in adrenocortical syn-

dromes, 933-934

in cancer of breast, 937-938

management of, 935-936

results of, 936

technique of, 934-935

Adrenaline, 930

Adrenocortical syndromes, investigations,

933

Adrenocorticotrophic hormone. *See* ACTH.

Adrenogenital syndrome, 930-931

investigations, 933

treatment of, 933-934

Aerocoe of neck, 868-869

Agenesis, renal, 302

ureteric, 306

Ageusia, 684

Agnosia, in parieto-occipital syndrome,

715, 716

Air embolism, 1014

Air-encephalography. *See* Encephalo-

graphy, air.

Air insufflation, peri-nephric, 325

Aldosterone, 725, 930

Aldosteronism, 933, 934

Alkali burns, 1298

Alkaline phosphatase test, of liver function,

78

- Alkalosis, respiratory, in traumatic coma, 634-639
- Alveolus, cysts of, acquired, 513-517
- Amigen solutions, 301
- Amnesia, post-traumatic, 635  
retrograde, 635
- Amoebiasis, cutaneous, 183  
hepatic, 184-190  
clinical picture, 185  
complications of, 186  
differential diagnosis of, 186  
investigations, 185-186  
treatment of, 189
- Intestinal, 182-184  
clinical manifestations, 183  
investigations, 183-184  
treatment of, 184  
treatment of, 186-190
- Amerboina, diagnosis from carcinoma, 150, 170
- Amputation, amniotic, 1036  
in gangrene, 976-977  
in lymphoedema, 1032  
in osteogenic sarcoma, 1073  
in tuberculosis of bone, 1050  
traumatic, of hand, 1288
- Anemia, hemolytic, 96-97  
in gastric carcinoma, 13, 16, 18
- Anæsthetics, effect of, in shock, 1304
- Anal canal, anatomy of, 156-157  
carcinoma of, 175-176  
fissure, 162-163  
hematoma, 161  
stenosis, diagnosis from Hirschsprung's disease, 273
- Anderson-Hynes pyeloplasty, for hydro-nephrosis, 342
- Androgens, 725, 1068
- Aneurysms, abdominal, diagnosis from perforated peptic ulcer, 33  
arterial, 984-990  
etiology of, 984-985  
clinical features, 986-987  
complications of, 987  
differential diagnosis of, 987-988  
investigations, 988  
treatment of, 988-990
- arteriovenous, papilloedema and, 671  
atherosclerotic, 985  
berry, 745  
carotid, 746, 747, 749-750, 863  
cirsoid, 984  
congenital, 984  
intracranial, 745-748  
management of, 748-752  
diagnosis from mediastinal cysts and tumours, 518  
dissecting, 984, 985, 988  
diagnosis from pancreatitis, 68  
false, 984  
femoral, diagnosis from hernia, 216  
fusiform, 984  
in arteriosclerosis, 753  
intracranial, 745-752  
surgical treatment, 749-752  
operative exposures, 751-752  
mycotic, 752, 963, 985
- Aneurysms, syphilitic, 985  
varicose, 990
- Angina, Ludwig's, 862  
pectoralis, 799  
operative treatment of, 796-797, 800
- Angiography, of aneurysms, 749  
of cerebral vessels, 697  
of epilepsy, 759  
of meningiomas, 709, 710  
of parietal lobe, 717  
of temporal lobe, 712-713  
of tumours of frontal lobe, 707  
vertebral, 699-700
- Angioma, 1018, 1022. *See also* under names of organs.
- Angiorectuloma, of posterior fossa, 721
- Angiosarcoma, 1018
- Ankle joint, abduction injuries of, 1276-1277  
epiphyseal injuries of, 1278  
external rotation injuries of, 1275-1276  
fractures and fracture-dislocations of, 1269-1278  
compression, 1277-1278  
ligamentous injury of, 1274-1275
- Ankyloglossia, congenital, 849
- Anosmia, causes of, 666
- Anterior tibial syndrome, 821-822
- Antibiotic therapy, 1314-1318  
bowel sterilization, 150-151  
complications of, 1317-1318  
general principles of, 1318  
of actinomycosis, 853  
of acute infective arthritis, 1115  
of appendicitis, 8-9  
of biliary disease, 62-63  
of bronchiectasis, 494  
of cerebral abscess, 661-662, 663  
of cholecystitis, 51  
of epipituitary abscess, 787  
of extradural abscess, 655  
of fractures, 1199, 1247  
of hand infections, 1289, 1291  
of hand injuries, 1285  
of lateral sinus thrombosis, 657  
of meningitis, 659  
of osteomyelitis, 664, 1041  
of pancreatitis, 69  
of parotitis, 838  
of perforated ulcer, 35  
of perinephric abscess, 337  
of peritonitis, 101, 137, 289  
of pulmonary abscess, 465, 470, 473  
of pyelonephritis, 400  
of pyogenic infection of liver, 81, 82  
of soft chancre, 419  
of spinal injuries, 780  
of subdural abscess, 656, 657  
of subphrenic abscess, 104  
of urethritis, 410, 411  
of urinary tract infections, 1314
- Anticoagulant therapy, 1015-1017  
in arterial grafting, 995  
in embolectomy, 984  
of atherosclerosis, 973  
of frost-bite and trench foot, 981  
of iliofemoral thrombosis, 998, 999

# INDEX

## A

- Abdomen, acute, in children, 287-296  
 burst, 38, 195-196  
 mass in, in appendicitis, 4, 6  
 in gastric carcinoma, 16  
 pain in, differential diagnosis, 11  
 Abdominal incisions, 191-196  
 wall, agenesis of muscles, 237  
 anterior, abnormalities of, 234-240  
 development of, 234  
 Abscess, amoebic, 184  
 anorectal, 163-164  
 apical, 856  
 appendiceal. *See* Appendix, abscess of.  
 areola, 944  
 brain, 659-664  
 bacteriology of, 660  
 diagnosis of, 660-661, 712  
 epilepsy and, 761  
 treatment of, 661-664  
 breast, 944-945  
 Brodie's, 769, 1044-1045, 1050, 1078  
 cold, 874, 1025, 1046-1047, 1093-1094  
 collar stud, 871  
 epidural, 787  
 extradural, 655  
 hepatic, 82  
 diagnosis from hydatid cyst, 84  
 horseshoe, 164  
 ischiorectal, 163  
 mesenteric, 107  
 pelvic, 36, 104-105  
 polviorectal, 164  
 perianal, 163  
 perinephric, 335-337  
 periurethral, 418  
 psoas, diagnosis from hernia, 209, 216  
 pulmonary, 457-475. *See also* Lung  
 abscess,  
 pyogenic, 118, 163-164  
 retropharyngeal, acute, 863  
 chronic, 863-864  
 subdural, 656-657  
 subphrenic, 103-104  
 diagnosis from hydatid cyst, 84  
 empyema and, 476-477  
 in cholecystitis, 50  
 in peptic ulcer, 36, 38  
 treatment of, 104  
 tuberculous, of neck, 868  
 Acetabulum, fractures of, 1234  
 Achondroplasia, 1033  
 Achromycin, 1316. *See also* Tetracycline.  
 Acid burns, 1298  
 Acoustic tumour, 690  
 Acrocyanosis, 979  
 operative treatment, 796-797  
 Acromegaly, 727, 859, 1068  
 Acromioclavicular joint, arthritis of, 1096  
 dislocations of, 1164  
 ACTH, 724, 725, 930  
 in acquired haemolytic anaemia, 97  
 in hypopituitarism, 731  
 in idiopathic thrombocytopenic purpura,  
 96  
 in pseudomembranous enterocolitis, 116  
 Actinomycosis, diagnosis from carcinoma of  
 colon, 150  
 Crohn's disease, 115  
 malignancy in neck, 1020  
 of face, 828  
 of jaw, 854-856  
 of liver, 82  
 of lung, 474  
 of neck, 864, 870  
 rectal stricture in, 168  
 Adamantinoma, 728  
 Addison's disease, adrenal androgens and  
 corticoids in, 1068  
 Adenitis, gummatous, 1031  
 mesenteric, in children, 290  
 diagnosis, from appendicitis, 288  
 tuberculous, 518, 526, 868, 871-875  
 treatment of, 873-875  
 Adenocarcinoma of appendix, 11  
 of stomach, 14  
 Adenoidectomy, in tuberculous cervical  
 adenitis, 875  
 ADH, 724, 725  
 Adrenal cortex, in metabolic disturbances  
 of bone, 1068  
 glands, 930  
 medulla, tumours of, 936  
 Adrenalectomy, in adrenocortical syn-  
 dromes, 933-934  
 in cancer of breast, 937-938  
 management of, 933-936  
 results of, 936  
 technique of, 934-935  
 Adrenaline, 930  
 Adrenocortical syndromes, investigations,  
 933  
 Adrenocorticotrophic hormone. *See* ACTH.  
 Adrenogenital syndrome, 930-931  
 investigations, 933  
 treatment of, 933-934  
 Aterocoele of neck, 868-869  
 Agensis, renal, 302  
 ureteric, 306  
 Ageusia, 684  
 Agnosia, in parieto-occipital syndrome,  
 715, 716  
 Air embolism, 1014  
 Air-encephalography. *See* Encephalo-  
 graphy, air.  
 Air insufflation, peri-nephric, 325  
 Aldosterone, 723, 930  
 Aldosteronism, 933, 934  
 Alkali burns, 1298  
 Alkaline phosphatase test, of liver function,  
 78

- Alkalosis, respiratory, in traumatic coma, 638-639
- Alveolus, cysts of, acquired, 513-517
- Amigen solutions, 301
- Amnesia, post-traumatic, 635
- retrograde, 635
- Amoebiasis, cutaneous, 183
- hepatic, 184-190
- clinical picture, 185
- complications of, 186
- differential diagnosis of, 186
- investigations, 185-186
- treatment of, 189
- intestinal, 182-184
- clinical manifestations, 183
- investigations, 183-184
- treatment of, 184
- treatment of, 186-190
- Anaerobiosis, diagnosis from carcinoma, 150, 176
- Amputation, amniotic, 1036
- in gangrene, 976-977
- in lymphoedema, 1032
- in osteogenic sarcoma, 1073
- in tuberculosis of bone, 1050
- traumatic, of hand, 1288
- Anæmia, hæmolytic, 96-97
- in gastric carcinoma, 13, 16, 18
- Anæsthetics, effect of, in shock, 1304
- Anal canal, anatomy of, 156-157
- carcinoma of, 175-176
- fissure, 162-163
- hematoma, 161
- stenosis, diagnosis from Hirschsprung's disease, 273
- Anderson-Hynes pyeloplasty, for hydro-nephrosis, 342
- Androgens, 725, 1068
- Aneurysms, abdominal, diagnosis from perforated peptic ulcer, 33
- arterial, 984-990
- etiology of, 984-985
- clinical features, 986-987
- complications of, 987
- differential diagnosis of, 987-988
- investigations, 988
- treatment of, 988-990
- arteriovenous, papilloedema and, 671
- atherosclerotic, 985
- berry, 745
- carotid, 746, 747, 749-750, 865
- cirsoid, 984
- congenital, 984
- intracranial, 745-748
- management of, 748-753
- diagnosis from mediastinal cysts and tumours, 518
- dissecting, 984, 985, 988
- diagnosis from pancreatitis, 88
- false, 984
- femoral, diagnosis from hernia, 216
- fusiform, 984
- in arteriosclerosis, 753
- intracranial, 745-752
- surgical treatment, 749-752
- operative exposures, 751-752
- mycotic, 752, 963, 985
- Aneurysms, syphilitic, 985
- varicose, 990
- Angina, Ludwig's, 862
- pectoris, 709
- operative treatment of, 796-707, 800
- Angiography, of aneurysms, 749
- of cerebral vessels, 697
- of epilepsy, 759
- of meningiomas, 709, 710
- of parietal lobe, 717
- of temporal lobe, 712-713
- of tumours of frontal lobe, 707
- vertebral, 699-700
- Angioma, 1018, 1022. *See also* under names of organs.
- Angioreticuloma, of posterior fossa, 721
- Angiosarcoma, 1018
- Ankle joint, abduction injuries of, 1276-1277
- epiphyseal injuries of, 1278
- external rotation injuries of, 1275-1276
- fractures and fracture-dislocations of, 1269-1278
- compression, 1277-1278
- ligamentous injury of, 1274-1275
- Ankyloglossia, congenital, 840
- Anosmia, causes of, 666
- Anterior tibial syndrome, 821-822
- Antibiotic therapy, 1314-1318
- bowel sterilization, 150-151
- complications of, 1317-1318
- general principles of, 1318
- of actinomycosis, 855
- of acute infective arthritis, 1115
- of appendicitis, 8-9
- of biliary disease, 62-63
- of bronchiectasis, 494
- of cerebral abscess, 661-662, 663
- of cholecystitis, 51
- of epidural abscess, 787
- of extradural abscess, 653
- of fractures, 1199, 1247
- of hand infections, 1289, 1291
- of hand injuries, 1285
- of lateral sinus thrombosis, 657
- of meningitis, 658
- of osteomyelitis, 664, 1041
- of pancreatitis, 69
- of parotitis, 838
- of perforated ulcer, 35
- of perinephric abscess, 337
- of peritonitis, 101, 137, 289
- of pulmonary abscess, 465, 470, 473
- of pyelonephritis, 400
- of pyogenic infection of liver, 81, 82
- of soft chancre, 419
- of spinal injuries, 780
- of subdural abscess, 656, 657
- of subphrenic abscess, 104
- of urethritis, 410, 411
- of urinary tract infections, 1314
- Anticoagulant therapy, 1015-1017
- in arterial grafting, 995
- in embolectomy, 984
- of atherosclerosis, 973
- of frost-bite and trench foot, 981
- of iliofemoral thrombosis, 998, 999

# INDEX

## A

- Abdomen, acute, in children, 287-296  
burst, 38, 193-196  
mass in, in appendicitis, 4, 6  
in gastric carcinoma, 16  
pain in, differential diagnosis, 6  
Abdominal incisions, 191-196  
wall, agenesis of muscles, 237  
anterior, abnormalities of, 234-240  
development of, 234  
Abscess, amœbic, 184  
anorectal, 163-164  
apical, 850  
appendiceal. *See* Appendix, abscess of.  
areola, 944  
brain, 659-664  
bacteriology of, 660  
diagnosis of, 660-661, 712  
epilepsy and, 761  
treatment of, 661-664  
breast, 944-945  
Brodie's, 769, 1044-1045, 1050, 1078  
cold, 874, 1025, 1048-1047, 1093-1094  
collar stud, 871  
epidural, 787  
extradural, 655  
hepatic, 82  
diagnosis from hydatid cyst, 84  
horseshoe, 164  
ischio-rectal, 163  
mesenteric, 107  
pelvic, 36, 104-105  
pelvirectal, 164  
perianal, 163  
perinephric, 335-337  
periurethral, 416  
psoas, diagnosis from hernia, 208, 216  
pulmonary, 457-475. *See also* Lung  
abscess.  
pyogenic, 118, 163-164  
retropharyngeal, acute, 863  
chronic, 863-864  
subdural, 650-657  
subphrenic, 103-104  
diagnosis from hydatid cyst, 84  
empyema and, 476-477  
in cholecystitis, 50  
in peptic ulcer, 36, 38  
treatment of, 104  
tuberculous, of neck, 868  
Acetabulum, fractures of, 1234  
Achondroplasia, 1033  
Achromycin, 1316. *See also* Tetracycline.  
Acid burns, 1298  
Acoustic tumour, 690  
Acrocyanosis, 979  
operative treatment, 796-797  
Acromegaly, 727, 859, 1068  
Acromioclavicular joint, arthritis of, 1096  
dislocations of, 1164  
ACTH, 724, 725, 930  
in acquired hæmolytic anæmia, 97  
in hypopituitarism, 731  
in idiopathic thrombocytopenic purpura, 96  
in pseudomembranous enterocolitis, 116  
Actinomyces, diagnosis from carcinoma of  
colon, 150  
Crohn's disease, 115  
malignancy in neck, 1029  
of face, 828  
of jaw, 854-856  
of liver, 82  
of lung, 474  
of neck, 864, 870  
rectal stricture in, 168  
Adamantinoma, 728  
Addison's disease, adrenal androgens and  
corticoids in, 1068  
Adenitis, gummatous, 1031  
mesenteric, in children, 290  
diagnosis, from appendicitis, 288  
tuberculous, 518, 526, 868, 871-875  
treatment of, 873-875  
Adenocarcinoma of appendix, 11  
of stomach, 14  
Adenoidectomy, in tuberculous cervical  
adenitis, 875  
ADH, 724, 725  
Adrenal cortex, in metabolic disturbances  
of bone, 1068  
glands, 930  
medulla, tumours of, 936  
Adrenalectomy, in adrenocortical syn-  
dromes, 933-934  
in cancer of breast, 937-938  
management of, 935-936  
results of, 936  
technique of, 934-935  
Adrenaline, 930  
Adrenocortical syndromes, investigations,  
933  
Adrenocorticotrophic hormone. *See* ACTH.  
Adrenogenital syndrome, 930-931  
investigations, 933  
treatment of, 933-934  
Aerocele of neck, 868-869  
Agenesis, renal, 302  
ureteric, 306  
Ageusia, 684  
Agnosia, in parieto-occipital syndrome,  
715, 716  
Air embolism, 1014  
Air-encephalography. *See* Encephalo-  
graphy, air.  
Air insufflation, peri-nephric, 325  
Aldosterone, 723, 930  
Aldosteronism, 933, 934  
Alkali burns, 1298  
Alkaline phosphatase test, of liver function,  
78

Atherosclerosis, 963, 965-966, 983  
 occlusion of internal carotid in, 754  
 treatment of, with anticoagulants, 973  
 with reconstructive surgery, 974-976  
 Atresia, pulmonary and tricuspid, 614  
 Atrial defects, 624-625  
 Aureomycin, 1316  
 in Friedlander's bacillus infection, 473  
 in lymphogranuloma inguinale, 1025  
 in soft chanere, 419  
 in urethritis, non-gonococcal, 411  
 Auricle, congenital lesions of, 826  
 Auricular fibrillation, pre- and post-operative, 904, 909  
 Automatism, post-traumatic, 635  
 Autocopy, 750  
 Axonotmesis, 803, 808

## B

Babinski reflex, 605-606  
 Badenoch's pull-through operation, 415  
 Balanitis, 314, 419-420  
 Bankart's operation, 1163  
 Banti's disease, 97  
 Barrier cream, 239  
 Basal metabolic rate, 886  
 Basophilism, pituitary, 728. *See also*  
 Cushing's disease or syndrome.  
 Bassini's operation, 211-212  
 Bat's ears, 826-827  
 Behcet's disease, 845  
 Bell's palsy, 694  
 Bennett's stove fracture, 1167, 1217-1218  
 Betatron, 1319  
 Bier's spots, 982  
 Bile duct, common, cystic dilatation of, 262-263  
 obstruction of, 81  
 Bile ducts, carcinoma of, 58  
 hypertonia of, 47  
 stricture of, 63-64  
 Bilharziasis, 108, 109, 366  
 Biliary tract, abnormalities of, congenital, 261-263  
 adenoma of, 57  
 antibiotic therapy of, 62-63  
 atresia of, congenital, 261-262  
 obstruction of, due to biliary sand, 262  
 operations, 58-62  
 papilloma of, 57  
 tumours of, 57-58  
 Bilirubin, use of, 48  
 Bilirubin serum test, of liver function, 77  
 Billroth I operation, 42-43  
 Billroth II operation, 42  
 Birthmarks, 1018  
 Bladder, abnormalities of, congenital, 365  
 automatic, 386  
 autonomous, 386  
 cauda equina, 386, 387  
 diversion of urine from, by operation, 363-364  
 diverticulum of, 372-373, 389  
 dysfunction, neurogenic, 385-388  
 exstrophy of, 238, 309

Bladder, foreign bodies in, 370  
 Hunner's ulcer of, 367  
 injuries of, 365-366  
 in spinal cord lesions, 386, 776-778  
 neck, 393, 398  
 obstruction, 398-399, 393-406  
 carcinoma of prostate causing, 403  
 congenital, 309  
 in prostatitis, 404  
 without prostatic enlargement, 398-399  
 with retention of urine, 399  
 papilloma, 375  
 paralytic, 396, 397  
 reflex, 396  
 rupture of, complicating pelvic fracture, 1237  
 schistosomiasis of, 366  
 trabeculation of, 389  
 tuberculosis of, 440-442  
 tumours of, 373-379  
 treatment of, 377-379  
 Blalock's operation for Fallot's tetralogy, post-operative care, 623  
 technique of, 615-617  
 Blindness, 666  
 Blood, cells, tumours of, 1075-1076  
 coagulation of, 1015  
 cross matching, 1311  
 diseases, splenectomy in, 95-98  
 groups, 1310-1311  
 incompatibility of, 1312  
 occult, test in gastric carcinoma, 17-18  
 plasma and substitutes, 1313  
 transfusion, 1310-1313  
 in acquired hæmolytic anæmia, 97  
 in burns, 1300  
 in carcinoma of colon, 151  
 in coarctation of aorta, 631-632  
 in pulmonary resection, 563, 564  
 in rupture of spleen, 95  
 in shock, 1303  
 in thoracoplasty, 540  
 reactions, 1312-1313  
 technique of, 1311-1312  
 urea, estimation of, as test of renal function, 326  
 Boeck's sarcoid, Crohn's disease and, 113  
 Böhler-Braun splint, 1184  
 Boils, of face, 657, 827  
 of neck, 861  
 Bones, cysts of, 1077-1078  
 diseases of, causing pathological fractures, 1178  
 fractures of. *See* Fractures and under names of bones.  
 grafts, 1198, 1209  
 gumma of, 1078  
 injuries of, complications, 1200-1210  
 malunion of, 1209-1210  
 traumatic atrophy of, 1203-1204  
 tuberculosis of, 1078  
 tumours of, 1069-1077  
 chondromas, 1069-1070, 1077-1078  
 chondrosarcoma, 1073, 1074  
 classification of, 1069  
 enchondromas, 1070-1071, 1077

- Anticoagulant therapy, of mesenteric vascular occlusion, 108  
 of post-operative thrombosis, 1000
- Anticonvulsant drugs, dosages of, 759-760
- Antrum, maxillary, carcinoma of, 680
- Anus, carcinoma of, diagnosis from anal fissure, 162  
 covered, 276, 277-278, 312  
 ectopic, 278  
 imperforate, 277  
 malformations of. *See* Rectum and anus.
- Anxiety states, surgery of, 763
- Aorta, abdominal, aneurysm of, 985  
 coarctation of, 630-634  
 rupture of, 630
- Aortic incompetence, 625  
 stenosis, 625
- Aortography, 325
- Aphasia, in head injuries, 654
- Apoplexy, pituitary, 728
- Appendicectomy, ancillary care, 8  
 complications of, 9-10  
 indications and contra-indications for, ■  
 in infancy and childhood, 289  
 interval, 7  
 technique of, 7-8
- Appendicitis, acute, 1-10  
 aetiology of, 2  
 amoebic, 183  
 in infancy and childhood, 287-289  
 pathology, 2-3, 287  
 portal pyæmia and thrombophlebitis in, 80  
 position of appendix in, 1, 5  
 treatment of, 6-9, 288-289
- chronic, 11  
 pelvic abscess and, 104  
 recurrent subacute, 10-11  
 subacute, simulating duodenal ulcer, 25-26  
 unusual types of, 4-5
- Appendix, abscess of, 5, 6, 118  
 diagnosis from cholecystitis, 51  
 adenocarcinoma of, 11  
 anatomy of, 1-2  
 carcinoid, 11  
 congenital absence of, 1  
 fecolitis in, 2, 11  
 foreign bodies in, 11  
 mucocele of, 12  
 neoplasms of, secondary, 12  
 parasites in, 11  
 perforated, 3  
 position of, in appendicitis, 1, ■  
 tumours of, 11-12
- Apraxia, in parieto-occipital syndrome, 716
- Arachnoiditis, non-specific, optic atrophy and, 669
- Argentaffin tumour, of small gut, 111
- Argyll-Robertson pupil, 667
- Arnold-Chiari deformity, 743
- Arteries, disobliteration of, 974  
 grafting of, 974-976, 994-995  
 injuries of, 992-994  
 occlusion of, 964-965  
 peripheral, 963-995  
 chronic trauma of, 967
- Arteries, peripheral, excision and end-to-end anastomosis, 974  
 grafting of, 974-976  
 ischaemia of, chronic, 967-972  
 treatment of, 972-974  
 occlusion of, acute, 981-984  
 treatment of, 983  
 chronic, 965-972  
 treatment of, 972-974  
 post-traumatic, 967  
 reconstructive surgery for, 974-976  
 trauma of, 992-994  
 treatment of, 993-994
- Arteriography, of aneurysms, 988  
 of occlusive disease, 971
- Arteriosclerosis, aneurysms in, 752  
 medial, 963
- Arteriosclerotic degeneration, types of, 963
- Arteritis, 963-964  
 temporal, 667
- Artery, carotid, common, internal, ligation of, 750, 751  
 internal, occlusion of, 754  
 cerebellar, posterior inferior, occlusion of, 680, 690  
 popliteal, occlusion of, chronic, 967  
 vertebral, occlusion of, 680
- Arthritis, acute, diagnosis from hæmatogenous osteomyelitis, 1040  
 infective, of hip, 1114-1115  
 chronic, 1052-1063  
 gonorrhoeal, in hip, 1110  
 infective, of hip, 1114-1115  
 of wrist joint, 1106  
 mid-tarsal, 1148-1149  
 neurotrophic, 1062-1063  
 of acromioclavicular joint, 1006  
 psoriatic, 1061  
 purulent, 1042  
 rheumatoid, 1058-1062  
 clinical features, 1059-1060  
 of knee, 1131-1132, 1135  
 of wrist joint, 1106  
 pathology of, 1058-1059  
 radiology of, 1060  
 tenosynovitis in, 1104  
 treatment of, 1060-1061
- subtaloid, 1146-1147  
 traumatic, 1203  
 tuberculous, 1040, 1106, 1118-1119
- Arthrodesis, in hallux valgus, 1152  
 in osteo-arthritis, 1057-1058, 1121  
 of foot, 1144  
 of knee joint, 1135  
 of shoulder joint, 1099  
 of toe, 1153
- Arthroplasty, in osteo-arthritis, 1057, 1058, 1121-1122  
 Keller's, 1152
- Arthrosis deformans, 1052-1058. *See also* Osteo-arthritis.
- Ascites, 88, 102, 105  
 chyloous, 1032  
 operations for relief of, 93
- Astrocytoma, and glioblastoma, 709  
 of posterior fossa, 720-721
- Atelectasis, bronchoscopy for, 454-455

Atherosclerosis, 963, 965-966, 995  
 occlusion of internal carotid in, 754  
 treatment of, with anticoagulants, 973  
 with reconstructive surgery, 974-976  
 Atresia, pulmonary and tricuspid, 614  
 Atrial defects, 624-625  
 Aureomycin, 1316  
 in Friedlander's bacillus infection, 473  
 in lymphogranuloma inguinale, 1025  
 in soft chancre, 410  
 in urethritis, non-gonococcal, 411  
 Auricle, congenital lesions of, 826  
 Auricular fibrillation, pre- and post-operative, 904, 909  
 Automatism, post-traumatic, 635  
 Autoscopy, 756  
 Axonotmesis, 803, 809

## B

Babinski reflex, 695-696  
 Badenoch's pull-through operation, 415  
 Balanitis, 314, 419-420  
 Bankart's operation, 1163  
 Banti's disease, 97  
 Barrier cream, 239  
 Basal metabolic rate, 896  
 Basophilism, pituitary, 729. *See also*  
     Cushing's disease or syndrome.  
 Bassini's operation, 211-212  
 Bat's ears, 820-827  
 Bechet's disease, 845  
 Bell's palsy, 694  
 Bennett's stove fracture, 1167, 1217-1218  
 Betatron, 1310  
 Bier's spots, 992  
 Bile duct, common, cystic dilatation of, 262-263  
     obstruction of, 81  
 Bile ducts, carcinoma of, 59  
     hypertonia of, 47  
     stricture of, 63-64  
 Bilharziasis, 168, 169, 366  
 Biliary tract, abnormalities of, congenital, 261-263  
     adenoma of, 57  
     antibiotic therapy of, 62-63  
     atresia of, congenital, 261-262  
     obstruction of, due to biliary sand, 262  
     operations, 58-62  
     papilloma of, 57  
     tumours of, 57-58  
 Bilgravin, use of, 48  
 Bilirubin serum test, of liver function, 77  
 Billroth I operation, 42-43  
 Billroth II operation, 42  
 Birthmarks, 1018  
 Bladder, abnormalities of, congenital, 365  
     automatic, 386  
     autonomous, 386  
     cauda equina, 386, 387  
     diversion of urine from, by operation, 363-364  
     diverticulum of, 372-373, 389  
     dysfunction, neurogenic, 385-388  
     exstrophy of, 238, 309

Bladder, foreign bodies in, 370  
 Hunner's ulcer of, 367  
 injuries of, 365-366  
 in spinal cord lesions, 386, 776-778  
 neck, 393, 399  
     obstruction, 398-399, 393-406  
     carcinoma of prostate causing, 403  
     congenital, 399  
     in prostatitis, 404  
     without prostatic enlargement, 398-399  
     with retention of urine, 399  
 papilloma, 375  
 paralytic, 396, 397  
 reflex, 396  
 rupture of, complicating pelvic fracture, 1237  
 schistosomiasis of, 366  
 tralaculation of, 399  
 tuberculosis of, 440-442  
 tumours of, 373-379  
     treatment of, 377-379  
 Blalock's operation for Fallot's tetralogy, post-operative care, 623  
     technique of, 615-617  
 Blindness, 666  
 Blood, cells, tumours of, 1075-1076  
     coagulation of, 1015  
     cross matching, 1311  
     diseases, splenectomy in, 95-99  
     groups, 1310-1311  
     incompatibility of, 1312  
     occult, test in gastric carcinoma, 17-18  
     plasma and substitutes, 1313  
     transfusion, 1310-1313  
     in acquired hemolytic anemia, 97  
     in burns, 1300  
     in carcinoma of colon, 151  
     in coarctation of aorta, 631-632  
     in pulmonary reaction, 563, 564  
     in rupture of spleen, 95  
     in shock, 1303  
     in thoracoplasty, 540  
     reactions, 1312-1313  
     technique of, 1311-1312  
     urea, estimation of, as test of renal function, 326  
 Boeck's sarcoid, Crohn's disease and, 113  
 Bohler-Braun splint, 1184  
 Boils, of face, 637, 827  
     of neck, 861  
 Bones, cysts of, 1077-1078  
     diseases of, causing pathological fractures, 1178  
     fractures of. *See Fractures and under names of bones.*  
     grafts, 1198, 1209  
     gumma of, 1078  
     injuries of, complications, 1200-1210  
     malunion of, 1209-1210  
     traumatic atrophy of, 1203-1204  
     tuberculosis of, 1078  
     tumours of, 1069-1077  
     chondromas, 1069-1070, 1077-1078  
     chondrosarcoma, 1073, 1074  
     classification of, 1069  
     enchondromas, 1070-1071, 1077



- Bones, tumours of, fibromas, 1071  
 fibrosarcoma, 1074  
 malignant, 1071-1077  
 osteochondromas, 1069-1070  
 osteomas, 1069-1070  
 sarcoma, 1071-1073, 1074  
 secondary malignant, 1076-1077
- Bougies, injuries by, 412  
 urethral, 409
- Bow-legs, 1136, 1137
- Brachial plexus, lesions of, 811-813
- Bradford frame, in apinal fractures, 1231
- Brain, angiography of, 697-701  
 cystic tumour of, angiogram of, 701  
 cysts of, 708  
 embolism of, 1015  
 infarction of, 748  
 injuries of, 639-641, 648  
 susceptibility to epilepsy, 757  
 swelling of, 737  
 syphilis of, diagnosis from frontal lobe  
 tumours, 707  
 tumours of, epilepsy and, 761  
 frontal region, 706-707
- Branchial cysts, 867-868
- Branchiogenic carcinoma, 869
- Brandon's modification of Bassini's operation, 212
- Breast, 939-940  
 abscess of, 944-945  
 adenocarcinoma of, 950  
 adenofibroma of, 949  
 carcinoma of, 945, 950-958  
 cysts of, 947-948  
 ectopic tissue, 941  
 examination of, 940-941  
 fat necrosis of, 948  
 fibro-adenoma of, 946  
 infective conditions of, 944-948  
 lump in, diagnosis and investigation of,  
 941, 953  
 male, carcinomas of, 959  
 disorders of, 958-960  
 tumours of, benign, 948-949  
 malignant, 949-958
- Broek's valvotomy, 619
- Brodie's abscess, 769, 1044-1045, 1050, 1078
- Serocystic tumour, 950
- Bronchial tree, anatomy of, 447-450
- Bronchiectasis, 489-495  
 chronic, 476  
 carcinoma and, 498  
 congenital cysts and, 512  
 papillœdema in, 673  
 lobectomy for, 558  
 post-operative complications and sequelæ,  
 494-495  
 tuberculous, excision for, 544  
 types of, 489-490
- Bronchoscopy, 445-456  
 aspiration by, 455  
 indications for, 455-456  
 post-operative use of, 563  
 technique of, 445-450
- Bronchostenosis, tuberculous, 544
- Bronchus, carcinoma of, 448, 456, 496-507,  
 527  
 incidence of, 496-498  
 intracranial metastasis, 697, 710, 768  
 investigations, 500-504  
 lung abscess and, 457  
 neuropathy and, 819-820  
 pathology of, 498  
 treatment of, 504-507  
 cysts of, 475, 527-528  
 foreign bodies in, 450-454, 476  
 stricture of, 455
- Brown-Séquard syndrome, 771-772, 774
- Browne (Denis) expression of tuberculous  
 cervical glands, 875  
 operation, for cleft lip, 248  
 for hypospadias, 311-312, 313  
 for post-traumatic stricture, 415  
 splint, for club-foot, 1140
- Brudzinski's sign, 658
- Bruises of the scalp, 636
- Bryant's method of treatment, for fractures  
 of shaft of femur, 1252
- Bubonocœle, 204
- Buerger's disease, 966-967  
 exercises, 973  
 test, 969
- Buffalo hump, in Cushing's syndrome,  
 931
- Buie's operation, for pilonidal sinus, 181
- Bunion, 1145, 1151-1152
- Burns, 1293-1300  
 acid and alkali, 1298  
 estimation of area of, 1294  
 fluid replacement in, 1298-1300  
 treatment of, by dressings, 1295  
 by exposure, 1295-1298  
 by skin grafting, 1296  
 immediate, 1294
- Buritis, acute, 1096-1097  
 olecranon, 1101  
 prepatellar, 1133-1134  
 subacromial, 1096-1097  
 tuberculous, 1050-1051
- Butazolidin, in osteoarthritis, 1054, 1135  
 in rheumatoid arthritis, 1061  
 in spondylitis ankylopoetica, 1059  
 in tendinitis, 1098

## C

- Cæcum, appendix in relation to, 1-2  
 diverticulum of, diagnosis from appen-  
 dicitis, 5  
 volvulus of, 133-134
- Calcaneus, fractures of, 1280-1281
- Calciferol, in parathyroid tetany, 929
- Calculi, parotid, 849  
 prostatic, 405  
 renal, 342-347  
 salivary, 838-840  
 submandibular, 839  
 ureteric, 358-361  
 urethral, 417  
 vesical, 368-370
- Calf muscle, pulled, 1147-1148

- Caloric tests, in neurosurgery, 088  
 Cancerum oris, 815  
 Capitellum, fractures of, 1223-1224  
 Caruncle of face, 657, 827  
   renal, 334-335  
 Cardiospasm, 580-583  
   diagnosis from mediastinal tumour, 518  
 Carotid body tumour, 870  
 Carotid ligation, 750, 751  
 Carotid sinus syndrome, sympathectomy and, 709  
 Carpal cysts, 1078  
 Carpal tunnel syndrome, 816  
 Carpus, fractures and dislocations of, 1215-1219  
 Cartilage of knee, injuries of, 1128-1129, 1200  
   loose, in joints, 1124-1125  
 Casoni test, in hydatid disease, 83  
 Catheter, bicoudé, 391  
   Foley, 380, 381, 382, 392  
   gauges, 404  
   Hickes', 392  
   Tiemann, 392  
   whistle-tip, 391  
 Catheterisation, 325, 390-392, 391, 415  
 Cauda equina, ependymomas of, 789-790  
   lesions, bladder in, 779-777  
   syndromes, 775-776, 783  
   tumours of, sciatica caused by, 786  
 Causalgia, 806  
 Cavernositis, fibrous, 421  
 Cavernous sinus, thrombosis, 657  
 Cellulitis, diagnosis from osteomyelitis, 1040  
   of hand, 1290  
   of neck, 802-803  
   of orbit, diagnosis from osteitis, 854  
 Cephalohematoma, 647, 769  
 Cerebello-pontine angle, tumours of, 680  
 Cerebral atrophy, 760-761  
   fungus, 652-653  
 Cerebrospinal fluid, normal, 658  
 Cervical disc protrusion, 781  
   ganglion, superior, resection of, 796  
   lymph glands, neoplasms of, 875-877  
   tuberculous, 871-875  
   rib, 790, 813-814  
   spinal cord, tumours of, 818  
   syndromes, 774-775  
 Chancre, soft, 419-420  
   syphilitic, 419  
 Chancroid, 419-420  
 Charcot's disease of joints, 1062-1063, 1119, 1136  
 Cherubism, 859  
 Cheyne-Stokes' breathing, 639  
 Chiasm, optic, glioma of, 667  
 Chickenpox, diagnosis from appendicitis, 8  
 Chlbblain, 979-980  
 Chimney sweeps' cancer, 433  
 Chloramphenicol, 1316, 1318  
   in *Haemophilus influenzae* meningitis, 659.  
   *See also* Chloromycetin.  
 Chloride test, in tuberculous meningitis, 658-659  
 Chloromycetin, in Friedlander's bacillus infection, 437. *See also* Chloramphenicol.  
 Chloroquine, in amebiasis, 188, 189  
 Chlorotetracycline, 1316. *See also* Aureomycin.  
 Cholangiography, use of, on operating table, 61  
 Cholangioma, 85  
 Cholangitis, in cholecystitis, 50  
   suppurative, acute, 81  
 Cholecystectomy, emergency, 51  
   technique of, 58-59  
 Cholecystitis, 48-49  
   acute, 49-51  
     diagnosis from perforated peptic ulcer 33  
     treatment of, 51  
   chronic, 51-54  
     stones and, treatment of, 50-57  
     diagnosis from appendicitis, 5  
       duodenal ulcer, 25  
       hydatid cyst, 84  
       omental torsion, 107  
       pancreatitis, 68  
 Cholecystostomy, technique of, 50-52  
 Choledochoduodenostomy, 62, 111  
 Choledocholithotomy, 62  
 Choledochostomy, 60  
 Cholecholestomy, 60  
 Cholesteatoma, 605, 710, 723, 766  
 Cholesterol gall-stones, 55  
 Cholesterofosis, of gall bladder, 54  
 Cholinergic drugs, in myasthenia, 914, 919  
 Chondro-malacia patellae, 1127  
 Chondroma, 1069-1070, 1077-1078  
 Chondrosarcoma, 1073-1074  
 Chordoma, 285  
 Christmas factor, 1015  
 Chromaffin tissue tumour, 936  
 Chromophobe adenoma, 725, 726-772  
 Chvostek's sign, 929  
 Chylothorax, 1032  
 Cino-radiography, in study of micturition, 334  
 Circulation, peripheral, 961-963  
 Circumcision, 312-313, 315  
   operative technique of, 316  
   in adults, 421-422  
 Cirrhosis hepatis, 87-93  
 Cisternal puncture, technique of, 772-773  
 Cisternography, for pituitary tumours, 729  
 Clavicle, dislocations of, 1163-1165  
   fractures of, 1226-1227  
 Claw hand, 816  
 Cleft lip and palate, 241-249  
 Cleidocranial dysostosis, 1034  
 Club-foot, 1138-1141  
 Clutton's joints, 1037, 1135  
 Coccydynia, 1238  
 Coccyx, fractures of, 1238  
 Cold abscess, 874, 1025, 1046-1047, 1093-1094  
 Colectomy, for carcinoma, 150  
   for ulcerative colitis, 142  
   total, technique of, 155  
 Colic, biliary, diagnosis from appendicitis, 11  
   cholecystitis, 50  
   intestinal obstruction, 125  
   pancreatitis, 68  
   mechanism of, 47

- Bones, tumours of, fibromas, 1071**  
 fibrosarcoma, 1074  
 malignant, 1071-1077  
 osteochondromas, 1069-1070  
 osteomas, 1069-1070  
 sarcoma, 1071-1073, 1074  
 secondary malignant, 1076-1077
- Bougies, injuries by, 412**  
 urethral, 409
- Bow-legs, 1136, 1137**
- Brachial plexus, lesions of, 811-813**
- Bradford frame, in spinal fractures, 1231**
- Brain, angiography of, 697-701**  
 cystic tumour of, angiogram of, 701  
 cysts of, 708  
 embolism of, 1015  
 infarction of, 748  
 injuries of, 639-641, 648  
 susceptibility to epilepsy, 757  
 swelling of, 737  
 syphilis of, diagnosis from frontal lobe tumours, 707  
 tumours of, epilepsy and, 761  
 frontal region, 706-707
- Branchial cysts, 867-868**
- Branchiogenic carcinoma, 869**
- Brandon's modification of Bassini's operation, 212**
- Breast, 939-940**  
 abscess of, 944-945  
 adenocarcinoma of, 950  
 adenofibroma of, 949  
 carcinoma of, 945, 950-958  
 cysts of, 947-948  
 ectopic tissue, 941  
 examination of, 940-941  
 fat necrosis of, 948  
 fibro-adenoma of, 948  
 infective conditions of, 944-948  
 lump in, diagnosis and investigation of, 941, 953  
 male, carcinoma of, 950  
 disorders of, 958-960  
 tumours of, benign, 948-949  
 malignant, 949-958
- Brock's valvotomy, 619**
- Brodie's abscess, 769, 1044-1045, 1050, 1078**  
 serocystic tumour, 950
- Bronchial tree, anatomy of, 447-450**
- Bronchiectasis, 489-495**  
 chronic, 476  
 carcinoma and, 498  
 congenital cysts and, 512  
 papilloedema in, 672  
 lobectomy for, 558  
 post-operative complications and sequelæ, 494-495  
 tuberculous, excision for, 544  
 types of, 489-490
- Bronchoscopy, 445-456**  
 aspiration by, 455  
 indications for, 455-456  
 post-operative use of, 563  
 technique of, 445-450
- Bronchostenosis, tuberculous, 544**
- Bronchus, carcinoma of, 448, 456, 496-507, 527**
- Bronchus, carcinoma of, diagnosis of, 498, 524**  
 incidence of, 496-498  
 intracranial metastasis, 697, 710, 768  
 investigations, 500-504  
 lung abscess and, 457  
 neuropathy and, 819-820  
 pathology of, 498  
 treatment of, 504-507  
 cysts of, 475, 527-528  
 foreign bodies in, 450-454, 476  
 stricture of, 455
- Brown-Séquard syndrome, 771-772, 774**
- Browne (Denis) expression of tuberculous cervical glands, 875**  
 operation, for cleft lip, 248  
 for hypospadias, 311-312, 313  
 for post-traumatic stricture, 415  
 splint, for club-foot, 1140
- Brudzinski's sign, 658**
- Bruises of the scalp, 636**
- Bryant's method of treatment, for fractures of shaft of femur, 1252**
- Bubonocoele, 204**
- Buerger's disease, 966-967**  
 exercises, 973  
 test, 969
- Buffalo hump, in Cushing's syndrome, 931**
- Buie's operation, for pilonidal sinus, 181**
- Bunion, 1145, 1151-1152**
- Burns, 1293-1300**  
 acid and alkali, 1298  
 estimation of area of, 1294  
 fluid replacement in, 1298-1300  
 treatment of, by dressings, 1295  
 by exposure, 1295-1296  
 by skin grafting, 1296  
 immediate, 1294
- Burnitis, acute, 1096-1097**  
 olecranon, 1101  
 prepatellar, 1133-1134  
 subacromial, 1096-1097  
 tuberculous, 1050-1051
- Butazolidin, in osteoarthritis, 1054, 1135**  
 in rheumatoid arthritis, 1061  
 in spondylitis ankylopoetica, 1059  
 in tendinitis, 1098
- 
- Cæcæm, appendix in relation to, 1-2**  
 diverticulum of, diagnosis from appendicitis, 5  
 volvulus of, 133-134
- Calcaneus, fractures of, 1280-1281**
- Calciferol, in parathyroid tetany, 929**
- Calculi, parotid, 840**  
 prostatic, 405  
 renal, 342-347  
 salivary, 838-840  
 submandibular, 839  
 ureteric, 358-361  
 urethral, 417  
 vesical, 368-370
- Calf muscle, pulled, 1147-1148**

Decortication, in empyema, 484  
 Deformities, congenital, 1033-1037  
 Déjà vu phenomenon, 756  
 Dentigerous cyst, 837-839  
 Deoxycorticosterone, in hypopituitarism, 731  
   in traumatic coma, 834  
 De Quervain's stenosing tenosynovitis, 1101  
 Dermatitis, ammoniacal, 314, 315  
 Dermatomes, 770  
 Dermoid cysts, 791  
 Devic's disease, 667  
 De Vulpis rongeur, 731, 735  
 Diabetes, in pancreatitis, 69, 70  
   insipidus, 726, 727, 729, 731  
   surgery in, 1304-1309  
 Diaphragm, congenital abnormalities of, 234  
   eventration of, 254, 369, 811  
 Diaphylal acalasia, 1033-1034  
 Diathermy, in intracranial surgery, 732  
 Dindevan, 1016, 1017  
 Diodone, 697, 703  
 Diplopia in head injuries, 653  
 Dislocations, classification of, 1157  
   complications of, 1160  
   *See also under names of joints.*  
 Diverticula, of colon, 134-137. *See also*  
   Diverticulosis,  
   of small gut, 109-111  
 Diverticulitis, 135, 137, 271  
   carcinoma in, 145, 148  
   coli, pelvic abscess and, 104  
   portal pyemia and thrombophlebitis  
   in, 80  
   venous occlusion and, 108  
 Meckel's, 109-110  
 Diverticulosis, 134-137  
 Diverticulum, duodenal, 110-111  
   jejunal and ileal, 111  
   Meckel's, 209-271  
   of bladder, 372-373  
   traction, of small gut, 111  
 DOCA, in adrenalectomy, 935  
 Dolichocolon, diagnosis from Hirschsprung's  
   disease, 273  
 DOPA reaction, 833  
 Drusen, simulating papilloedema, 672  
 Ductus arteriosus, patent, 626-629  
   operative technique for, 627-629  
 Dugas' sign, 1161  
 Dukes and Masina's classification of bladder  
   tumours, 376  
 Dumb-bell ganglioneuroma, 802  
   tumours, 789  
 Duodenal ulcer, diagnosis from cholecystitis,  
   50  
   simulating gastric carcinoma, 18. *See*  
   *also* Peptic ulcer.  
 Duodenum, atresia of, 258-259  
   diverticulum of, 110-111  
   obstruction of, extrinsic, 259  
   intrinsic, 258-259  
   stenosis of, 258-259  
 Dupuytren's contracture, 1107-1108  
   fracture, 1266-1268, 1271  
 Durham's tracheotomy outfit, 879  
 Duval forceps, 552

Dwarfism, pituitary, 725, 729  
 Dysentery, amebic, 183  
 Dyskinesia, bulary, 55  
 Dysphasia, in head injuries, 654  
   in temporal lobe syndrome, 711  
 Dysplasia of bone, fibrous, 1063-1066

**E**

Ectopia vesicae, 309  
   and epispadias, 238-240  
 EEG. *See* Electroencephalography.  
 Egger's plates, 1197-1198  
 Eisenmenger's complex, 610  
 Elastoplast treatment, of varicose ulcer,  
   1013  
 Elbow-joint, dislocations of, 1163-1166  
   fractures around, 1222-1223  
   osteoarthritis of, 1100  
 Electrocardiography, 762  
 Electroencephalography, in brain abscess,  
   661  
   in epilepsy, 758, 761  
   in grand mal and small grand mal, 757,  
   758  
   in intracranial tumours, 704-705, 758  
   in Jacksonian seizures, 756, 758  
   in juvenile petit mal, 756, 758  
 Electromyography, in amyotrophic lateral  
   sclerosis, 819  
   in peripheral nerve injury, 806, 808  
 Electrotome, McCarthy's "resectoscope,"  
   397, 398  
 Elephantiasis, 1029-1031  
 Emblectomy, technique of, 983-984  
 Embolism, 961-962, 981  
   air, 1014-1015  
   cerebral, 1015  
   diagnosis from thrombosis, 982  
   fat, 1015, 1201, 1307  
   from thyrotoxic auricular fibrillation, 809  
   mesenteric arterial, intestinal obstruction  
   and, 121  
   paradoxical, 962  
   pulmonary, 962, 996, 998, 1015  
   complicating appendicectomy, 10  
   cholecystectomy, 59  
   varicose veins, 1006, 1003, 1009  
 Embolus, cardiac, causing mesenteric  
   arterial occlusion, 107, 108  
   mesenteric, causing arterial occlusion, 107  
 Embryological maldevelopments, 1035-1037  
 Embryoma, renal, 304-306  
 Emetine in amebiasis, 184, 188, 189  
 Emphysema, bronchiectasis and, 492-493  
   obstructive, 452, 453  
   papilloedema and, 672  
 Empyema, chronic, 486-488  
   decortication in, 488, 516-518  
   resection and unroofing operations, 488  
   diagnosis from mediastinal tumour, 518  
   in amebiasis, 186  
   localised, 472  
   necessitatis, 485  
   non-tuberculous, 476-488  
   of appendix, 3

- Colic, renal, 344
- Colitis, mucous, diagnosis from ulcerative colitis, 139
- ulcerative, 137-143
- clinical features, 139
- differential diagnosis, 139
- from Crohn's disease, 114-115, 138, 139
- fistula-in-ano, 163
- pathology of, 138
- prognosis of, 142-143
- rectal stricture in, 168
- treatment of, 139-142, 155
- Collar stud abscess, 871
- Colles' fracture, 1211-1214
- reversed, 1214
- Colloid cysts, 710
- Colon, ameboma of, 183
- carcinoma of, 145
- diverticula of, 134-137. *See also* Diverticulosis.
- exteriorization of, technique of, 153
- inertia of, diagnosis from Hirschsprung's disease, 273
- operations on, 152-156
- perforation of, 135, 137
- resection of, technique of, 153-156
- sigmoid, carcinoma of, 145
- volvulus of, 132-133
- tumours of, 143-145
- benign, 143
- malignancy and, 144, 150
- Colostomy, for carcinoma of colon, 151
- for intestinal obstruction, 129
- for rectal carcinoma, 173-174
- malformation, 282, 283
- prolapse, 168
- stricture, 169
- technique of, 152-153
- Colotomy, for tumours, 144
- Coma, hypopituitary and hypothalamic, 731
- in hyperparathyroidism, 925
- traumatic, metabolism in, 637-639
- Complement fixation test, in hydatid disease, 83
- Compound F, 725
- Compression syndrome of spinal cord, 770-773
- Concussion, 635
- Condyles, femoral, fractures of, 1254-1255
- fractures of, 1223
- tibial, fractures of, 1255-1256
- Condylomata acuminata, 419
- Conn's disease, 933
- Contusion of brain, 635
- Coombs's reaction, 96-97
- Corns, 1146
- Corpora amylacea, 405
- Corticosteroids, estimation of, 720
- Corticotrophin, 930
- Cortisol, 725
- Cortisone, 930
- adrenal deficiency following, 724
- in adrenalectomy, 935
- in coma, traumatic, 638
- in haemolytic anaemia, 97
- in haemorrhagic pancreatitis, 69
- Cortisone, in hypopituitarism, 731
- in idiopathic thrombocytopenic purpura, 96
- in pseudomembranous enterocolitis, 116
- in rheumatoid arthritis, 1061
- in spondylitis ankylopoetica, 1089
- Costotransversectomy, for Pott's disease, 787
- Councill's (basket) extractor, 361
- Coxa vara, adolescent, 1116-1118
- Craniopharyngioma, 725, 728-729
- Craniotabes, 1067
- Crepitus, 1174
- Cribs' operation for block dissection of neck, 876-877
- Crohn's disease, 113-115
- diagnosis from carcinoma, 150
- ulcerative colitis, 114-115, 138, 139
- fistula and, 9, 118, 120, 165, 371
- rectal stricture in, 168
- Cruveilhier, pearly tumour of, 710, 723, 766, 790
- Cuff, short rotator, degenerative tendinitis of, 1007-1008
- of humerus, 1225-1226
- rupture of, 1096-1097
- Curetting, of tuberculous glands, 875
- Cushing's disease or syndrome, 728, 931, 933, 1068
- Cystadenoma, of liver, 85
- of pancreas, 71
- Cystectomy, for bladder tumours, 377-378
- Cystinuria, 342
- Cystitis, 366-368
- acute, 366
- chronic, 366-368
- cystica, 368
- haematuria in, 322
- tuberculous, 440
- with metaplasia, 368
- Cystogram, micturating, 384
- Cystometrography, 382-384
- Cystometry, 317
- Cystoprostatectomy, for carcinoma of prostate, 402
- Cystoscopy, 325-326
- for bladder tumours, 377
- for pyonephrosis, 334
- for schistosomiasis, 366
- Cystostomy, for retention of urine, 777-778
- prostatectomy and, 399
- suprapubic, 380, 391
- Cystourethrogram, 384
- D**
- Dactylitis, tuberculous, 1047
- Dandy's operation, for tic douloureux, 678
- Davis and Ritchies' classification of cleft lip and cleft palate, 242
- Deafness, audiometry in, 686
- causes of, 686-687, 722
- congenital, 683
- cranial nerve lesions and, 683
- head injuries complicated by, 653
- in chronic otitis media, 663

- Decortication, in empyema, 489  
 Deformities, congenital, 1033-1037  
 DeJà vu phenomenon, 758  
 Dentigerous cyst, 857-859  
 Deoxycorticosterone, in hypopituitarism, 731  
 in traumatic coma, 638  
 De Quervain's stenosing tenosynovitis, 1104  
 Dermatitis, ammoniacal, 314, 315  
 Dermatomes, 770  
 Dermoid cysts, 701  
 Devic's disease, 667  
 De Villiers rongeur, 734, 735  
 Diabetes, in pancreatitis, 69, 70  
 insipidus, 726, 727, 729, 731  
 surgery in, 1304-1309  
 Diaphragm, congenital abnormalities of, 254  
 eventration of, 254, 269, 811  
 Diaphragmal hernia, 1033-1034  
 Diathermy, in intracranial surgery, 732  
 Dindevan, 1016, 1017  
 Diiodone, 697, 705  
 Diplopia in head injuries, 653  
 Dislocations, classification of, 1157  
 complications of, 1160  
*See also under names of joints.*  
 Diverticula, of colon, 134-137. *See also*  
 Diverticulosis,  
 of small gut, 109-111  
 Diverticulitis, 135, 137, 271  
 carcinoma in, 145, 148  
 coli, pelvic abscess and, 104  
 portal pyemia and thrombophlebitis  
 in, 80  
 venous occlusion and, 109  
 Meckel's, 109-110  
 Diverticulosis, 134-137  
 Diverticulum, duodenal, 110-111  
 jejunal and ileal, 111  
 Meckel's, 209-271  
 of bladder, 372-373  
 traction, of small gut, 111  
 DOCA, in adrenalectomy, 935  
 Dolichocolon, diagnosis from Hirschsprung's  
 disease, 273  
 DOPA reaction, 833  
 Drusen, simulating papilloedema, 672  
 Ductus arteriosus, patent, 626-629  
 operative technique for, 627-629  
 Dugas' sign, 1101  
 Dukes and Maslans' classification of bladder  
 tumours, 378  
 Dumb-bell ganglioneuroma, 802  
 tumours, 789  
 Duodenal ulcer, diagnosis from cholecystitis,  
 50  
 simulating gastric carcinoma, 18. *See*  
*also* Peptic ulcer.  
 Duodenum, atresia of, 258-259  
 diverticulum of, 110-111  
 obstruction of, extrinsic, 259  
 intrinsic, 258-259  
 stenosis of, 258-259  
 Dupuytren's contracture, 1107-1108  
 fracture, 1266-1268, 1271  
 Durham's tracheotomy outfit, 879  
 Duval forceps, 552  
 Dwarfism, pituitary, 725, 729  
 Dysentery, amoebic, 183  
 Dyskinesia, biliary, 55  
 Dysphasia, in head injuries, 654  
 in temporal lobe syndrome, 711  
 Dysplasia of bone, fibrous, 1065-1066
- 
- Ectopia vesicae, 309  
 and epispadias, 238-240  
 EEG. *See* Electroencephalography.  
 Egger's plates, 1107-1108  
 Eisenmenger's complex, 610  
 Elastoplast treatment, of varicose ulcer,  
 1013  
 Elbow-joint, dislocations of, 1165-1166  
 fractures around, 1222-1225  
 osteoarthritis of, 1100  
 Electrocardiography, 762  
 Electroencephalography, in brain abscess,  
 661  
 in epilepsy, 759, 761  
 in grand mal and small grand mal, 757,  
 759  
 in intracranial tumours, 704-705, 758  
 in Jacksonian seizures, 756, 758  
 in juvenile petit mal, 756, 758  
 Electromyography, in amyotrophic lateral  
 sclerosis, 810  
 in peripheral nerve injury, 806, 808  
 Electrotome, McCarthy's "resectoscope,"  
 397, 399  
 Elephantiasis, 1029-1031  
 Embolctomy, technique of, 953-994  
 Embolism, 961-962, 931  
 air, 1014-1015  
 cerebral, 1015  
 diagnosis from thrombosis, 982  
 fat, 1015, 1201, 1307  
 from thyrotoxic auricular fibrillation, 909  
 mesenteric arterial, intestinal obstruction  
 and, 121  
 paradoxical, 962  
 pulmonary, 962, 996, 993, 1015  
 complicating appendicectomy, 10  
 cholecystectomy, 59  
 varicose veins, 1006, 1003, 1009  
 Embolus, cardiac, causing mesenteric  
 arterial occlusion, 107, 103  
 mesenteric, causing arterial occlusion, 107  
 Embryological maldevelopments, 1035-1037  
 Embryoma, renal, 304-306  
 Emetine in amoebiasis, 184, 188, 189  
 Emphysema, bronchiectasis and, 492-493  
 obstructive, 452, 455  
 papilloedema and, 672  
 Empyema, chronic, 488-488  
 decortication in, 488, 546-548  
 resection and unroofing operations, 488  
 diagnosis from mediastinal tumour, 518  
 in amoebiasis, 186  
 localised, 472  
 necessitatis, 485  
 non-tuberculous, 476-489  
 of appendix, 3

- Empyema, of ethmoid and sphenoid sinuses, 668-669  
     of gall-bladder, diagnosis from appendicitis, 6  
     post-operative, 530  
     staphylococcal, 471  
     subdural, 656-657  
     tuberculous, 545, 546-548  
 Encephalography, air, for epilepsy, 759  
     for pituitary tumours, 729  
     for porencephaly, 761  
 Encephalopathy, hypertensive, 707  
 Enchondroma, 1070-1071, 1077  
 Endarterectomy, 974  
 Endo-aneurysmorrhaphy, technique of, 989-990  
 Endocarditis, bacterial, 627  
 Endometriosis, appendix in, 12  
 Endothelioma, dural, 708  
     *See also* Meningioma.  
 Enteritis, ulcerative, in Crohn's disease, 114  
 Enterocolitis, pseudomembranous, 116  
 Enuresis, 316-318, 320  
 Enzyme therapy, of empyema, 481-482  
 Ependymoma, of posterior fossa, 721  
 Epicondyles, fractures of, 1223  
 Epidermoid cysts, 700  
     tumours, 710, 723  
 Epidermoids, 728-729  
 Epididymectomy, 426, 435, 444  
 Epididymis, cysts of, 429-430  
 Epididymitis, 424-426, 444  
     acute, 424, 425  
     chronic, 425  
     non-specific, 424  
     subacute, 425  
     tuberculous, 424  
 Epididymo-orchitis, 424, 432-433  
 Epilepsy, 755-757  
     gyratory, 766  
     idiopathic, 757  
     in brain abscess, 660  
     in head injuries, 653  
     investigations, 757-759  
     Jacksonian, 755-756  
     major, 755  
     minor, 756  
     nature of, 755  
     psychomotor, 756  
     temporal lobe, 690, 756, 760  
     traumatic, 761  
     treatment of, dosage of anticonvulsants, 759-760  
         surgical, diseases amenable to, 760-762  
     uncinate, 756  
 Epiphysis, injuries of, of humerus, 1224-1225  
     of medial epicondyle, 1224  
     of tibia, 1278  
     radial, displacement of, 1214  
     slipped femoral, 1116-1117, 1118  
 Epiphysitis, syphilitic, 1037  
 Epispadias, ectopic vesicae and, 236-240  
 Epulis, varieties and treatment of, 855-856  
 Erb-Duchenne palsy, 812  
 Erysipeloid of Rosenbach, 1023-1024  
 Erythema induratum, 980  
 Erythrocyanosis frigida, 980, 1031  
 Erythromelalgia, 979  
 Erythromycin, 1317, 1318  
 Ethmoid, sinus, mucocoele and empyema of, 668-669  
     sinusitis, papilloedema and, 671, 672  
 Eucortone, in head injuries, 638  
 Ewing's tumours, 80, 1074  
 Exomphalos, 236-237  
 Exophthalmos, malignant, 891-892  
 Exostosis, subungual, 1154  
 Extradural hæmatoma, 639-643  
 Extramedullary tumours, 773, 776  
 Eyelids, burns of, 1297  
 Eyes, burns involving, 1297  
     wounds of, fistulous, papilloedema in, 672
- F
- Face, burns of, 1297  
     carcinoma of, 831-832  
     congenital cysts and sinuses of, 825-826  
     epithelioma of, 830, 831  
     infections of, 827-828  
     inflammatory lesions of, 827-828  
     lipomas of, 830  
     non-inflammatory lesions of, 828  
     tumours of, 829-833  
 Facial nerve lesions, 683  
 Fæcal impaction, diagnosis from intestinal obstruction, 126  
     following Thiersch's operation, 167, 168  
 Fæcoliths, in appendix, 2, 11  
 Fallot's tetralogy, 610, 611-624, 660  
 Fat embolism, 1015, 1201, 1307  
 Femoral canal, 214  
 Femur, neck of, fractures of, 1230-1240, 1242-1243  
     shaft of, fractures of, 1248-1253  
         double, 252  
     supracondylar fractures of, 1254  
     upper end of, fractures of, 1230-1247  
 Ferrous sulphate poisoning, pyloric obstruction in, 258  
 Fibro-adenoma, 946-947  
 Fibroblastoma, acoustic, 721  
     meningeal. *See* Meningiomas.  
     perineural. *See* Neurofibroma.  
 Fibroma, 1071  
     *See also* under organs.  
 Fibrosarcoma, 1074  
     neural, 824  
 Fibrositis, 1061-1062  
 Fibula, diastasis of, 1267-1268  
     dislocations of, 1171  
     fractures of, 1265-1267  
     lower end of, fractures of, 1267  
     shaft of, fractures of, 1267  
     upper end of, fractures of, 1267  
 Fingers, burns of, 1297  
     clubbing of, 491, 610, 611  
 Fistula, anal, in Crohn's disease, 114  
     ano-urethral, congenital, 312  
     arteriovenous, 990-991, 1030  
     congenital, 990, 991  
     traumatic, 990, 991

- Fistula, between internal carotid artery and cavernous sinus, 746-747, 750**  
 biliary, 50, 59  
 bladder, 370-372  
 bronchial, 468  
 bronchiopleural, 477, 545  
   post-operative, 545, 565-566  
 cerebrospinal fluid, 739  
 diverticulosis and, 135, 137  
 fecal, 9  
 gastro-jejuno-colic, 31  
 in-ano, 165, 166  
 in Crohn's disease, 114  
 intestinal, 117-120  
 lymphatic, 1020, 1032  
 recto-urethral, congenital, 312  
 salivary, 840  
 sign, in chronic otitis media, 649  
 umbilical, 109, 371  
 urethral, 416  
 vesicocolic, 371  
 vesicovaginal, 371
- Flat-foot, everted, 1143-1144**  
 spasmodic, 1144
- Flocculation tests of liver function, 78**
- Fluid balance chart, in intestinal obstruction, 128, 130**  
 requirements, for infants and children undergoing surgical treatment, 297-301  
 therapy, 1305  
   according to weight and age, 300  
   in burns, 1298-1300  
   regime for patients, 1306-1307  
   in infancy and childhood, 298-301
- Foley catheter, 392**  
 "Y-V," pyeloplasty for hydronephrosis, 341
- Fontana's silver method, 835**
- Foot, bony swellings in, 1144-1145**  
 deformities of, severe, 1155-1156  
 dislocations of, 1171  
 fore-pain in, 1149  
 hind-pain in, 1146  
 lobster claw deformity, 1141  
 pain in mid-tarsal region, 1148  
 postural defects of, 1142-1143  
 pressure effects, 1144-1145
- Foramen magnum syndrome, 773-774**  
 tumours at, 680-681
- Forearm, fractures of, 1219-1221**
- Foreign bodies, empyema and, 486**  
 in alimentary tract in infancy, 295-296  
   in appendix, 11  
   in bladder, 370-372  
   in bronchus, 450-454, 476  
   in intestinal obstruction, 121  
   in Meckel's diverticulum, 271  
   in oesophagus, 295-296  
   in urethra, 417
- Foster-Kennedy syndrome, 671, 709**
- Fractional test meal, in gastric carcinoma, 16-17**
- Fractures, "beak," 1281**  
 callus formation in, 1204, 1207, 1208  
 Colles', 1211-1214  
 comminuted, 1177  
 compound, 1189-1190
- Fractures, compound, infection of, established, 1189**  
 prevention of, 1188-1189  
 infections of, 1192-1195  
 treatment of, 1188-1195  
 types of, 1189  
 with skin loss, 1191-1192  
 compression in, 1181  
 deformities caused by, 1183  
 dislocations complicated by, 1160  
 Dupuytren's, 1266-1267, 1268, 1271  
 fatigue, 1177, 1282  
 greenstick, 1172  
 infection of, 1180, 1200  
   *See also Fractures, compound.*  
 infraction, 1172  
 internal fixation of, 1196-1198  
 into joints, 1209  
 lymphoedema following, 1031  
 Maisonneuve's, 1265  
 malunion of, 1209-1210  
 march, 1282  
 Monteggia, 1220-1221  
 movement and union, 1181  
 natural history and repair of, 1177, 1178  
 non-union of, 1206, 1207  
   established, 1207-1209  
   hypertrophic, 1207-1208  
 pathological, 1177  
 pseudo-, 1067  
 radiology of, 1174-1175  
 reduction of, 1183  
 re-education after, 1186-1187  
 repair of, 1179-1180  
   natural history and, 1177-1178  
 separations, epiphyseal, 1172-1173  
 Smith's, 1214  
 spiral, 1176  
 traction in, 1185-1186  
 treatment of, 1172-1187  
 union of, 1180-1182  
   delayed, 1205-1206  
 varieties of, 1172  
   *See also under names of bones.*
- Fragilitas ossium, 1034-1035**  
 diagnosis from fibrous dysplasia, 1066
- Freiberg's disease of second metatarsal head, 1153**
- Frei's test, 1025**
- Freyer's prostatectomy, 396**
- Frey's syndrome, 843**
- Frohlich's syndrome, 729**
- Frontal lobe, oligodendroglioma of, 708**  
 operations, 763  
 syndrome, 706-707, 710  
 tumours of, diagnosis from tumours of posterior fossa, 719
- Frost-bite, 981**  
 operative treatment of, 796-797, 798
- FSH, 724, 725**
- Furuncle of neck, 861**

## G

- Galactoceles, 430, 945**
- Gall bladder, abnormalities of, 48, 263**



- Empyema, of ethmoid and sphenoid sinuses, 668-669  
     of gall-bladder, diagnosis from appendicitis, 6  
     post-operative, 530  
     staphylococcal, 471  
     subdural, 636-657  
     tuberculous, 545, 546-548  
 Encephalography, air, for epilepsy, 759  
     for pituitary tumours, 729  
     for porencephaly, 761  
 Encephalopathy, hypertensive, 707  
 Enchondroma, 1070-1071, 1077  
 Endarterectomy, 974  
 Endo-aneurysmorrhaphy, technique of, 989-990  
 Endocarditis, bacterial, 627  
 Endometriosis, appendix in, 12  
 Endothelioma, dural, 708  
     *See also* Meningioma.  
 Enteritis, ulcerative, in Crohn's disease, 114  
 Enterocolitis, pseudomembranous, 116  
 Enuresis, 316-318, 320  
 Enzyme therapy, of empyema, 481-482  
 Ependymoma, of posterior fossa, 721  
 Epicondyles, fractures of, 1223  
 Epidermoid cysts, 700  
     tumours, 710, 723  
 Epidermoids, 728-729  
 Epididymectomy, 426, 435, 444  
 Epididymis, cysts of, 429-430  
 Epididymitis, 424-426, 444  
     acute, 424, 425  
     chronic, 425  
     non-specific, 424  
     subacute, 425  
     tuberculous, 424  
 Epididymo-orchitis, 424, 432-433  
 Epilepsy, 755-757  
     gyratory, 750  
     idiopathic, 757  
     in brain abscess, 600  
     in head injuries, 653  
     investigations, 757-759  
     Jacksonian, 755-756  
     major, 755  
     minor, 756  
     nature of, 755  
     psychomotor, 756  
     temporal lobe, 690, 756, 760  
     traumatic, 761  
     treatment of, dosage of anticonvulsants, 759-760  
         surgical, diseases amenable to, 760-762  
         uncinate, 756  
 Epiphysis, injuries cf. of humerus, 1224-1225  
     of medial epicondyle, 1224  
     of tibia, 1228  
     radial, displacement of, 1214  
     slipped femoral, 1116-1117, 1118  
 Epiphysitis, syphilitic, 1037  
 Epispadias, ectopiae varicæ and, 238-240  
 Epulis, varieties and treatment of, 853-856  
 Erb-Duchenne palsy, 812  
 Erysipeloid of Rosenbach, 1023-1024  
 Erythema induratum, 980  
 Erythrocyanosis frigida, 980, 1031  
 Erythromelalgia, 979  
 Erythromycin, 1317, 1318  
 Ethmoid, sinus, mucocele and empyema of, 668-669  
     sinusitis, papilloedema and, 671, 672  
 Eucortone, in head injuries, 638  
 Ewing's tumours, 80, 1074  
 Exomphalos, 236-237  
 Exophthalmos, malignant, 891-892  
 Exostosis, subungual, 1154  
 Extradural hæmatoma, 639-643  
 Extramedullary tumours, 773, 776  
 Eyelids, burns of, 1297  
 Eyes, burns involving, 1297  
     wounds of, fistulous, papilloedema in, 672
- ## F
- Face, burns of, 1297  
     carcinoma of, 831-832  
     congenital cysts and sinuses of, 825-826  
     epithelioma of, 830, 831  
     infections of, 827-828  
     inflammatory lesions of, 827-828  
     lipomas of, 830  
     non-inflammatory lesions of, 828  
     tumours of, 829-833  
 Facial nerve lesions, 683  
 Facial impaction, diagnosis from intestinal obstruction, 126  
     following Thiersch's operation, 167, 168  
 Fæcoliths, in appendix, 2, 11  
 Fallot's tetralogy, 610, 611-624, 660  
 Fat embolism, 1015, 1201, 1307  
 Femoral canal, 214  
 Femur, neck of, fractures of, 1230-1240, 1242-1245  
     shaft of, fractures of, 1248-1253  
         double, 252  
         supracondylar fractures of, 1254  
         upper end of, fractures of, 1230-1247  
 Ferrous sulphate poisoning, pyloric obstruction in, 258  
 Fibro-adenosis, 940-947  
 Fibroblastoma, acoustic, 721  
     meningeal. *See* Meningiomas.  
     perineural. *See* Neurofibroma.  
 Fibroma, 1071  
     *See also* under organs.  
 Fibrosarcoma, 1074  
     neural, 824  
 Fibrositis, 1061-1062  
 Fibula, diastasis of, 1267-1268  
     dislocations of, 1171  
     fractures of, 1265-1267  
     lower end of, fractures of, 1267  
     shaft of, fractures of, 1267  
     upper end of, fractures of, 1267  
 Fingers, burns of, 1297  
     clubbing of, 491, 610, 611  
 Fistula, anal, in Crohn's disease, 114  
     ano-urethral, congenital, 312  
     arteriovenous, 990-991, 1030  
         congenital, 990, 991  
         traumatic, 990, 991

- Hemangioma, cavernous**, 1019  
 classification of, 1018  
 cutaneous, 1018, 1022  
   *malignant change in*, 1018  
 of liver, 85  
 of skull, 765, 769  
 strawberry, radiotherapy of, 1323-1324
- Hemarthrosis**, 1200
- Hematemesis, in gastric carcinoma**, 16  
 in peptic ulcer, 25, 36
- Hematocoele**, of testis, 430
- Hematuria, congenital splenomegaly**, 97
- Hematoma, complicating pelvic fractures**, 1237  
 extralural, 639-643, 651  
   posterior fossa, 640-641  
   ossifying, 1200-1201  
   subdural, 643-644  
   subperiosteal, 647
- Hematuria**, 321-323, 326  
 in bladder tumours, 326  
 in renal tumours, 330, 331  
 in urinary tuberculosis, 440
- Hemorrhage, cerebellar**, 753  
 cerebral, 745, 753  
 extralural, 639-643  
 in arterial trauma, 692, 693  
 in corpus luteum, diagnosis from appendicitis, 6  
 in fractures, 1179  
 in injury, 1302, 1303  
 intracerebral, traumatic, 646  
 intracranial, post-operative, 763  
 in varicose veins, 1008-1009  
 meningeal, middle, 639-643  
 reactionary, after intracranial operations, 736-737  
 renal, 330  
 subarachnoid, 645, 671  
 subdural, 642
- Hemorrhoidectomy, post-operative stricture**, 168
- Hemorrhoids, external**, 161-162  
 internal, 157
- Hallux rigidus**, 1146, 1151  
 valgus, 1151-1153
- Halsted's operation for inguinal hernia**, 212
- Halsted-Tanner "slide"**, 212
- Hamatoma**, 745, 752-753, 1018
- Hamilton's ruler test**, 1161
- Hammer-toe**, 1142, 1145-1146, 1154
- Hammock method, in spinal fractures**, 1232
- Hand, benign synovium of**, 1103-1104  
 burns of, treatment of, 1297  
 epidermoid cysts of, 1103  
 ganglia of, 1102  
 infections of, 1289-1292  
   of joints, 1292  
   of palmar space, 1291-1292  
   of pulp space, 1291  
   of tendon-sheath, 1291  
   of web space, 1291  
   subcutaneous and subcuticular, 1290  
   subungual, 1291  
 injuries of, 1284-1289
- Hand, muscles of, nerve supply variations**, 801  
 wasting, differential diagnosis, 818-820
- Tendon of, repair of**, 1288-1289
- Rupture of**, 1105-1106
- Tenosynovitis in**, 1101-1105
- Tumours of**, 1102
- Hand-Schüller-Christian disease**, 766, 769
- Hare lip, See Cleft lip and palate**
- Harris's needle**, 676  
 prostatectomy, 396
- Hartmann's operation**, 178-179
- Hashimoto's thyroiditis**, 804
- Head, circumference of**, 740
- Head injuries**, 635-651  
 anaemia in, 666  
 clinical examination of, 636  
 complications of, 650-654  
 cranial nerve injuries in, 679-680  
 epilepsy after, 761  
 open, 647, 649-654  
 optic atrophy in, 667-668  
 small penetrating, 650  
 tangential, 648  
 tinnitus following, 688
- Heart, congenital disease of**, 610-625  
 failure, thyroidectomy in, 911
- Heberden's nodes**, 1033
- Heel, pain in**, 1147
- Heller's operation**, 581-593
- Hemianopia**, 747  
 causes of, 710, 726-727, 729
- Hemicolectomy, right**, 154  
 in tuberculous, 117
- Hemiplegia, in head injuries**, 654  
 infantile, 760-761
- Henoch's purpura**, diagnosis from  
 intussusception, 126
- Heparin**, 1015-1016
- Hepatoduodenostomy**, 64
- Hepatoma**, 85
- Hereditary diseases, classification of**, 1033
- Hernia, abdominal, aetiology of**, 197-193  
 clinical features, 199-200  
 complications of, 200-201  
 coverings of, 199  
 external, 197-200  
   rarer forms of, 220  
   varieties of, 197  
 in adults, 197-222  
 internal, 221-222  
 massive, 221-222  
 treatment of complications, 202-203
- Diaphragmatic, congenital**, 570  
 diagnosis from mediastinal cysts, 518  
 hiatus, 570-573  
 traumatic, 570
- "en glissade"**, 198
- Epigastric**, 219, 236
- Femoral**, 202, 214-218  
 aetiology of, 214-215  
 complications of, 215  
 diagnosis from varicose veins, 1005  
 differential, 215-216  
 external, 217  
 in infancy and childhood, 227

- Gall bladder, anatomy of, 48  
and bile ducts, 47-64  
carcinoma of, 57-58  
cholesterolosis, 54  
functions of, 47  
tests of, 47-48  
inflammation of, 48-49  
perforated, in cholecystitis, 50  
stasis, 54-55  
strawberry, 54  
stones, 54, 55-57  
chronic cholecystitis and, treatment of, 56-57  
intestinal obstruction caused by, 121
- Ganglia of wrist and hand, 1102
- Ganglionectomy, stellate, 796-797
- Ganglioneuroma, 523-525, 802
- Gangrene, atherosclerotic, 975  
diabetic, 976  
gas, 1192-1193  
ischemic, 962, 968-969  
of extremities and skin, operative treatment of, 796-797, 798  
of tips of fingers, in Raynaud's disease, 978  
perithelial causing, 994  
senile, 963  
surgery of, 976-977  
types of, 962-963
- Garré's chronic non-suppurative osteomyelitis, 1045
- Gastrectomy, partial, for anastomotic ulcer, 31  
for carcinoma of stomach, 18, 19  
for duodenal ulcer, 28, 29, 30  
for gastric ulcer, 28-29  
for peptic ulcer, 39  
perforated, 35  
technique of, 41-43  
post-gastrectomy, syndrome, 45-46  
total, for carcinoma of stomach, 18, 19  
technique of, 43-44
- Gastric secretion, 22, 23
- Gastritis, acute, diagnosis from perforated peptic ulcer, 33  
chronic, diagnosis from peptic ulcer, 27
- Gastro-enteritis, diagnosis from enteritis, 288  
intestinal obstruction, 126  
intussusception, 293  
in infants, diagnosis from appendicitis 5
- Gastro-enterostomy, anastomotic ulcer following, 31  
for duodenal ulcer, 28, 29, 40  
for peptic ulcer, 39  
technique of, 40-41
- Gastro-intestinal suction, in intestinal obstruction, 127, 130
- Gastrosocopy, in gastric carcinoma, 16  
in peptic ulcer, 25
- Gastrostomy, technique of, 44-45
- Gaucher's disease, 98
- Genetics and radiation, 1322
- Genito-urinary case, investigation of, 319-327
- Genu valgum, 1136  
varum, 1136, 1137
- Giant-cell tumour, 765, 1074-1075, 1078  
of tendon sheaths, 1103-1104  
*See also Osteoclastoma.*
- Gigantism, 727, 1068  
hemangiomatous diffuse, 1021
- Gillies-Fry operation, for cleft lip, 249
- Glaucoma, chronic, 867
- Gleet, 410
- Glioblastoma multiforme, angiogram of, 701  
parieto-occipital, 717  
pathology of, 708
- Glioma, frontal, 666  
of temporal region, 713  
pathology of, 707-708
- Gliomatous cysts, diagnosis from porencephaly, 646
- Glossitis, acute, 849  
atrophic, 849  
chronic superficial, 849  
mediana rhomboides, 852
- Gout, aberrant, 887  
adenomatous, 887-888  
endemic, 887  
intrathoracic, 890, 919  
lymphadenoid, 894, 902  
non-toxic, 887-890  
pubertal, 887  
recurrent, 910  
retrosternal, 888-890, 902, 910  
sporadic, 887  
substernal, 890  
toxic diffuse, primary, 890-893, 900, 905  
nodular, secondary, 893, 900, 907
- Gold therapy of rheumatoid arthritis, 1061
- "Golf-hole" ureter, 441
- Gonadotropins, 725
- Gonorrhoea, 169, 424
- Gout, 1156
- Grafting, arterial, 974-976  
bone, 1198, 1209  
skin, for burns, 1206-1207
- Grand mal, 755
- Grasp reflex, frontal tumours causing, 706-707
- Grawitz tumour, 348
- Gridiron incision, 7
- Groin, block dissection of, for anal carcinoma, 176
- Gross's technique, in reattachment of aorta, 634
- Gynæcomastia, 959-960
- Gumma, of bone, 1078  
of testis, 426  
of tongue, 852
- Gums, hypertrophy of, 856  
swellings of, 853
- Guyon's disease, 398-399

## II

- Hemangioblastoma of posterior fossa, 721
- Hemangioma, arterial, 1021  
capillary, superficial, 1019

- Hemangioma, cavernous**, 1019  
 classification of, 1018  
 cutaneous, 1018, 1022  
   malignant change in, 1018  
 of liver, 83  
 of skull, 765, 769  
 strawberry, radiotherapy of, 1323-1324
- Hemarthrosis**, 1200
- Hematemesis**, in gastric carcinoma, 16  
 in peptic ulcer, 25, 36
- Hematocele**, of testis, 430
- Hematocytopenia**, congestive splenomegaly, 97
- Hematomy**, complicating pelvic fractures, 1237  
 extradural, 639-643, 671  
   posterior fossa, 640-641  
 ossifying, 1200-1201  
 subdural, 643-644  
 subperiosteal, 647
- Hematuria**, 321-323, 326  
 in bladder tumours, 376  
 in renal tumours, 349, 351  
 in urinary tuberculosis, 440
- Hemorrhage, cerebellar**, 753  
*cerebral*, 745, 753  
 extradural, 639-643  
 in arterial trauma, 992, 993  
 in corpus luteum, diagnosis from appendicitis, 5  
 in fractures, 1179  
 in injury, 1302, 1303  
 intracerebral, traumatic, 646  
 intracranial, post-operative, 763  
 in varicose veins, 1008-1009  
 meningeal, middle, 639-643  
 reactionary, after intracranial operations, 756-757  
 renal, 330  
 subarachnoid, 645, 671  
 subdural, 642
- Hemorrhoidectomy**, post-operative stricture, 168
- Hemorrhoids**, external, 161-162  
 internal, 157
- Hallux rigidus**, 1146, 1151  
 valgus, 1151-1153
- Halsted's operation** for inguinal hernia, 212
- Halsted-Tanner "slide"**, 212
- Hamartoma**, 745, 752-753, 1018
- Hamilton's ruler test**, 1161
- Hammer-toe**, 1142, 1145-1146, 1154
- Hammock method**, in spinal fractures, 1232
- Hand**, benign synovium of, 1103-1104  
 burns of, treatment of, 1297  
 epidermoid cysts of, 1103  
 ganglia of, 1102  
 infections of, 1280-1292  
   of joints, 1292  
   of palmar space, 1291-1292  
   of pulp space, 1291  
   of tendon-sheath, 1291  
   of web space, 1291  
   subcutaneous and subcuticular, 1290  
   subungual, 1291  
 injuries of, 1284-1289
- Hand**, muscles of, nerve supply variations, 804  
   wasting, differential diagnosis, 818-820
- Tendons of**, repair of, 1288-1289
- Rupture of**, 1105-1106
- Tenosynovitis in**, 1104-1105
- Tumours of**, 1102
- Hand-Schüller-Christian disease**, 766, 769
- Hare lip**. *See* Cleft lip and palate.
- Harris's needle**, 676
- Prostatectomy**, 396
- Hartmann's operation**, 178-179
- Hashimoto's thyroiditis**, 894
- Head**, circumference of, 740
- Head injuries**, 635-654  
   anoxia in, 666  
   clinical examination of, 636  
   complications of, 650-654  
   cranial nerve injuries in, 679-680  
   epilepsy after, 761  
   open, 647, 649-654  
   optic atrophy in, 667-668  
   small penetrating, 650  
   tangential, 648  
   tinnitus following, 688
- Heart**, congenital disease of, 610-625  
 failure, thyroidectomy in, 911
- Heidenhain's nodes**, 1053
- Heel**, pain in, 1147
- Heller's operation**, 541-543
- Hemianopia**, 747  
 causes of, 710, 726-727, 729
- Hemicolecotomy**, right, 154  
 in tuberculosis, 117
- Hemiplegia**, in head injuries, 654  
 infantile, 760-761
- Henoch's purpura**, diagnosis from  
 intussusception, 126
- Heparin**, 1015-1016
- Hepatoduodenostomy**, 64
- Hepatoma**, 85
- Hereditary diseases**, classification of, 1033
- Hernia**, abdominal, aetiology of, 197-198  
 clinical features, 199-200  
 complications of, 200-201  
 coverings of, 199  
 external, 197-200  
   rarer forms of, 220  
   varieties of, 197  
 in adults, 197-222  
 internal, 221-222  
*massive*, 221-222  
 treatment of complications, 202-203
- Diaphragmatic, congenital**, 570  
 diagnosis from mediastinal cysts, 518  
 hiatus, 570-573  
 traumatic, 570  
 "en glissade," 198
- Epigastric**, 219, 236
- Femoral**, 202, 214-218  
 aetiology of, 214-215  
 complications of, 215  
 diagnosis from varicose veins, 1005  
   differential, 215-216  
 external, 217  
 in infancy and childhood, 227

- Hernia, femoral, irreducible, diagnosis from**  
 enlarged lymph gland, 216-217  
 strangulated, 215  
 treatment of, 218  
 treatment of, 217-218  
**gfuteal, 220**  
**incisional, 220-221**  
 after appendicectomy, 10  
**inguinal, 202-208, 221**  
 constriction in, 199  
 diagnosis from femoral hernia, 215-216  
 differential, 208  
 direct, 204, 205-206  
 differentiation from indirect, 206-207  
 indirect, 204, 205  
 in infancy and childhood, 223-227  
 inguinofemoral, 217  
 interstitial, 198  
 pantaloon, 205  
 saddle, 204  
 scrotal, hydrocele and, 427  
 intestinal obstruction and, 121, 124  
 irreducible, 200, 202, 205  
 Littre's, 199  
 lumbar, 220  
 Maydl's, 201  
 obstructed, 200, 203  
 obturator, 220  
 perineal, 220  
 post-operative, 222  
 properitoneal, 198  
 reducible, 199  
 Richter's, 198-199, 224  
 scrotic, 220  
 semiumbilical, in infancy, 234-235  
 Spigelian, 220  
 strangulated, 200-201, 202, 203, 224  
 taxis in, 202-203  
 umbilical, 218-219, 221, 234-236  
**Hernioplasty, for inguinal hernia, 209-210**  
**Herniorrhaphy, inguinal, 209, 210**  
**Herniotomy, inguinal, 209, 210**  
**Herpes, penile, 419**  
 zoster, 678-679, 684, 689  
**Hesselbach's triangle, 204**  
**Hey's prostatectomy, 399**  
**Hip-joint, arthritis of, acute infective, 1114-1115**  
 tuberculous, 1118-1119  
 clinical examination of, 1100-1112  
 diseases of, 1112, 1119-1120  
 dislocation of, 1108-1170  
 congenital dislocation of, 1112-1114  
 malposition in utero and, 1037  
 transient synovitis of, 1115  
**Hirschsprung's disease, 271-274**  
**Histamine phosphate test for phaeochromocytoma, 336**  
**Hodgkin's disease, 827, 788, 875, 1027, 1076**  
 sarcoma of small gut and, 113  
 spleen in, 98  
**Hoffmann's reflex, 695**  
**Homans' sign, 1000**  
**Hormones, growth, 724-725**  
 in injury, 1301-1302  
**Hormones, in peptic ulcer, 22**  
 pituitary, 724-726  
 deficiency symptoms, 725-726  
 sex, in metabolic bone disturbances, 1068  
**Horner's syndrome, 774, 775, 812**  
 in bronchial carcinoma, 500  
 post-operative, 797  
**Horseshoe kidney, 302, 329**  
**Hot cross bun head, 1037**  
**Housemaid's knee, 1123-1134**  
**Howard's extractor, 361**  
**Hudson's brace, 734**  
**Hufnagel's operation, for aortic incompetence, 625**  
**Humerus, fractures of, head of, 1225-1226**  
 lower end of, 1223-1225  
 neck of, 1226  
 shaft of, 1226  
 upper end of, 1225  
 radial nerve paralysis following, 1204  
 ruptures of short rotator cuff, 1225-1226  
**Hunger pain, 24-25**  
**Hunner's ulcer, 367**  
**Hunt (Ramsay) syndrome, 684**  
**Hunterian chancre of penis, 410**  
**Hunter's ligature, 989**  
**Hutchinson's pupil, 639**  
 syndrome, 801  
 teeth, 1037  
**Hydatids of liver, 82-85**  
 of lung, 474-475, 517  
 of pancreas, 71  
**Hydrocalicosis, 337**  
**Hydrocele, 426-428, 435**  
 complications of, 427  
 congenital, 428  
 diagnosis from inguinal hernia, 208  
 infantile, 227-229, 428  
 of canal of Nuck, 208, 228  
 of cord, 208, 428-429  
 of testicle, complicating hernia operations, 214  
 primary or idiopathic, 426-427  
 secondary or symptomatic, 428  
 treatment of, 427-428, 435  
 in infancy and childhood, 228-229  
**Hydrocephalus, 671, 774**  
 infantile, 739-744  
 causes of, 741-742  
 clinical signs of, 739-740  
 diagnosis of, 740-741  
 treatment of, 742-744  
 short-circuiting operations, 743-744  
**Hydrocortisone, 930**  
 in adrenalectomy, 935  
 in coccygeal injuries, 1238  
 in hypopituitarism, 731  
 in osteo-arthritis, 1056  
 in pancreatitis, 69  
 in subacromial bursitis, 1097  
**Hydroma, subdural, 644-645. See also Hygroma.**  
**Hydronephrosis, 307-308, 309, 329, 337-342, 390**  
 aetiology of, 337-339  
 diagnosis of, 340  
 from appendicitis, 288

- Hydronephrosis, diagnosis of, from Wilms' tumour, 306  
   symptoms of, 340  
   treatment of, 341-342  
 Hydrops of membranous labyrinth, 649  
 Hydro-ureter, 363  
 Hygroma, cystic, 1020-1027  
   subdural, 644-645  
 Hyperæmia reflex, as test for ischaemia, 971  
 Hypercalcuria, 342  
 Hyperchloræmia and hypochloræmia, after head injuries, 637-638  
 Hyperhidrosis, 709  
   operative treatment of, 706-707, 709  
 Hyperneplasmia, 347-348  
 Hyperostosis, meningioma causing, 709  
 Hyperparathyroidism, 923-924, 1066  
   calculi in, 342, 345-346  
   diagnosis of, 923-926  
   differential, 926  
   renal changes in, 925  
   surgical treatment of, 926-929  
   symptoms of, 923, 925  
 Hyperpituitarism, 1069  
 Hypersplenism, 94  
 Hypertension, malignant, 671, 753  
   portal. *See* Portal hypertension.  
   renal diseases and, 354-355  
 Hyperthermia, post-operative, 730  
 Hyperthyroidism, bone in, 1068  
   BMR in, 886  
   investigations in, 886-887  
 Hypomastia, 942  
 Hypoparathyroidism, 928-929  
 Hypophysectomy, for breast cancer, 928  
 Hypophyseal duct, tumours of, 728  
 Hypopituitarism, 727, 730-731  
 Hypospadias, 311-312  
 Hypothalamic disorders, in head injuries, 634  
 Hypothermia, in cardiac surgery, 624, 625, 631  
   in neurosurgery, 751  
   open cardiac exploration under, 622-623  
 Hypothyroidism, investigations, 886-887
- I
- ICSH, 724  
 Idiocy, amaurotic family, 667  
 Idioglossia, in congenital deafness, 688  
 Ileitis, regional, 113  
   *See also* Crohn's disease.  
   terminal, acute, 115-116  
 Ileocolostomy for obstruction, 129  
 Ileostomy, colectomy and, 155  
   for ulcerative colitis, 141-142  
   terminal, for familial polyposis, 144  
 Ileum, angioma of, intussusception due to, 126  
   atresia and stenosis of, 265-267  
   diverticulum of, 111  
 Ileus, mechanical, 9-10  
   meconium, 267  
   paralytic, 9, 129-130  
   complicating cholecystectomy, 59  
   fracture, 1233  
 Ileus, paralytic, hernia and, 201  
   in peptic ulcer, 35, 38  
   in peritonitis, 100  
   intestinal obstruction and, 121  
   post-operative, 174, 589  
   spastic, 130-131  
 Indigo-carminic, in tests of renal function, 327  
 Infarction, myocardial, diagnosis from pancreatitis, 68  
 Infusions, composition of solutions, 298-299  
 Inguinal canal, 203  
   lipoma within, diagnosis from hernia, 208  
 Injection treatment, of hæmorrhoids, 159-160  
   of varicose veins, 1006  
 Injury, fat embolism after, 1307  
   reactions to, 639, 1301-1308  
   immediate, 1302  
   metabolic response to, 1304-1305  
   treatment of, 1305-1307  
 Insulin tolerance test, 726  
 Intervertebral disc protrusions, 780-781  
 Intestinal obstruction, 121-131  
   after appendicectomy, 10  
   appendicitis simulating, 5  
   causes of, 121-122, 124-125  
   clinical features of, 123-124  
   diagnosis of, differential, 125-127  
   from cholecystitis, 50  
   from pancreatitis, 68  
   due to bands and adhesions, 124-125  
   carcinoma, 147  
   hernias, 122, 124, 569  
   intussusception, 123  
   stricture, 125  
   volvulus, 125  
   effects of, 122-123  
   functional, 129-131  
   in diverticulosis, 137  
   in Hirschsprung's disease, 272  
   in infancy, 224  
   in perforated peptic ulcer, 36  
   Meckel's diverticulum and, 270-271  
   treatment of, 127-129  
   vascular, 131  
 Intestines, carcinoma of, 145-156  
   cysts of, 525-526  
   duplication of, enterogenous cysts and, 275  
   injuries of, 201  
   obstruction of. *See* Intestinal obstruction.  
   small, 109-120  
   adenocarcinoma of, 112  
   carcinoid tumours of, 111-112  
   inflammation of, 113-116  
   neoplasm of, 111-116  
   sarcoma of, 112-113  
   tuberculosis of, 116-117  
   diagnosis from Crohn's disease, 113, 114  
   tumours of, malignant, 111-113  
   diagnosis from Crohn's disease, 115  
   simple, 111

Intestines, at strangulation of, 121-122  
 volvulus of, malrotation and, 264-265

Intra-alveolar epidermoid carcinomas, 857

Intracranial operations, complications of, 738-738  
 suppuration, 655-665  
 surgery, operative technique and exposures of, 732-738  
 post-operative measures, 732

tumours, 695-731  
 angiography of, 697-701  
 burr-hole biopsy of, 705  
 causing optic atrophy, 668  
 clinical presentation of, 705-706  
 diagnosis of, 695-705  
 dizziness caused by, 690  
 electroencephalography for, 704-705, 758  
 lumbar puncture in, 703-704  
 metastatic, 697, 710, 791  
 papilloedema in, 670-671  
 pneumoencephalography in, 704  
 radiography of, 696-697  
 ventriculography in, 701-703

vascular diseases, surgery of, 745-754

Intradural extramedullary lesions, 788-791

Intraluminal intestinal obstruction, 125

Intramedullary nails, in fractures, 1198

tumours, 776, 791

Intravenous fluids, in infancy and childhood, 298-301  
 in intestinal obstruction, 127-128, 130  
 regime for patients, 1306-1307

Intraventricular tumours, diagnosis from frontal tumours, 707

Intussusception, acute, differential diagnosis of, 293  
 chronic, 295  
 in childhood, 290-295  
 intestinal obstruction and, 125  
 Meckel's diverticulum and, 109  
 of appendix, 12

Iodine, in thyrotoxicosis, 901  
 radioactive, 899-900, 1321  
 in diagnosis, 886-887, 893, 899-900  
 serum protein-bound, in diagnosis, 887

Iodoxy, 697

Irradiation, rectal stricture caused by, 188, 189

Ischaemia, after arterial injury, 992  
 arterial, chronic peripheral, clinical examination, 969-970  
 manifestations of, 967-969  
 tests for, 971  
 treatment of, 972-974

Isoniazid, in pulmonary excision, 843, 875  
 in tuberculosis, peritonitis in, 103  
 ulcerative, 117  
 urinary, 442  
*See also* Isonicotinic acid hydrazide.

Isonicotinic acid hydrazide, streptomycin and, in tuberculosis, 1049

Isotopes, 1320  
 definition of, 886  
 radioactive, 1320, 1321  
 in occlusive disease, 973

## J

Jacksonian epilepsy, 752, 753, 755-756

Jaundice, aetiology, 50, 56, 57  
 antibiotic therapy of, 63  
 in carcinoma of bile ducts, 58  
 in congenital biliary conditions, 261, 263  
 in pancreatitis, 68, 70  
 vitamin K in, 77

Jaws, actinomycosis of, 854-850  
 adamantinoma of, 856  
 carcinoma of, 858, 859-860  
 chondroma of, 858  
 epithelial-lined cysts of, 857-858  
 fibrous dysplasia of, 858  
 fractures of, 854  
 infection of, 854-855  
 osteitis of, 854  
 osteoclastoma of, 858  
 osteoma of, 858  
 radionecrosis of, 858  
 swellings of, 850-850  
 "Jeep disease," 179

Jejunum, atresia and stenosis of, 265-267  
 diverticulum, 111

Joints, false, formation of, 1207  
 injuries to, 1157-1171, 1200  
 loose body in, 1200  
 tumours of, 1078-1079

Joly (Swift), urethroscope, 409

Jones (Robert), compression bandage, 1256

Judet's arthroplasty, 1122

## K

Kartagena's syndrome, 612

Keller's arthroplasty, 1152

Keloid scars, treatment of, 909, 1323

Kenwood mixing machine, 638

Kerato-acanthoma, 830-831

Keratosis, 831

Kernohan classification of gliomas, 708  
 -Woltman phenomenon, 711-712, 710

17-Ketosteroids, in urine, 726, 930, 933

Kidneys, aberrant renal blood vessel, 309  
 adenocarcinoma of, 347  
 angioma of, 3022  
 calculi in, 342-347  
 carbuncle of, 334-335  
 carcinoma of, 349, 351  
 calculi and, 344, 347  
 cysts of, 303-304  
 congenital, 304  
 diagnosis from Wilms' tumour, 306  
 multicystic, 304  
 solitary, 304, 353-354  
 developmental anomalies of, 328-329  
 discoid, 302  
 diseases of, 328-356  
 ectopia of, 303  
 failure of, after injury, 1207  
 development, 302  
 function of, assessment of, 326

- Kidneys, function of, tests of, 326-327  
 fusion of, 302-303  
 horse-shoe, 302, 329  
 hypertension in disease of, 354-355  
 hypoplasia of, 302  
 infections of, 330-337  
 operations on, 335-356  
 pelvis, double, 328  
 polycystic, 304, 352-353  
 trauma of, 330  
 tuberculosis of, 437  
 tumours of, 347,  
 Kienböck's disease, 1107  
 King's operation, for bilateral vocal cord  
 paralysis, 909  
 Kirschner's wire, 1185  
 Kite's plaster for club-foot, 1141  
 Klinefelter's syndrome, 960  
 Klumpke's palsy, 812-813  
 Knee-joint, clinical examination of, 1123-  
 1124  
   congenital discoid meniscus, 1127, 1129-  
   1130  
   cysts of menisci, 1129  
   dislocation of, 1170  
     recurrent, of patella, 1132  
   effusions into, 1131-1132  
   extensor expansion, rupture of, 1132  
   fractures around, 1254-1259  
   injuries of cartilage, 1128-1129  
   internal derangements of, 1124-1125  
   lax cruciate ligaments, 1127  
   loose bodies in, 1124-1125, 1126  
   osteo-arthritis of, 1131, 1135  
   rheumatoid arthritis of, 1135  
   tuberculosis of, 1134  
 Knock-knee, 1136  
 "Knuckle-cluster" splint, for ulnar nerve  
 palsy, 817  
 Kocher's incision, 194  
   manoeuvre, 1161  
 Köhler's disease, 1148  
 Kondoleon's operation for lymphadema,  
 1032  
 Kraurosis, 420  
 Kultschitzky cell carcinoma, of small gut,  
 111  
 Kummel's syndrome, 780  
 Küntscher nails, 1108  
 Kyphosis, 1081  
   adolescent, 1086-1087

## L

- Labyrinthitis, 689  
 Labyrinthotomy, 689  
 Laminectomy, for epidural abscess, 787  
   for Pott's disease, 787  
   technique of, 778-779  
 Laryngectomy, total, indications for, 883-  
 884  
 Laryngocele, 868-869  
 Laryngofissure, 883  
 Laryngopharynx, growths of, 883

- Laryngotomy, complications of, 880  
   technique of, 880-881  
 Larynx, cancer of, 883-884  
   injuries of, compression, 881  
     penetrating, 881-882  
 Leadbetter's operation, 364  
 Lead poisoning, gut in, 126, 130  
 Leiomyoma. *See under organs.*  
 Leontiasis ossea, 897, 858-859  
   optic nerve compression in, 668  
 Leptomenigitia, 857  
 Leukæmia, 876, 1076  
   acute, spontaneous brain hæmorrhage in,  
   753  
   arterial thrombosis in, 108  
   lymphatic, small gut and, 113  
   priapism in, 421  
   skull in, 769  
 LI, 724, 725  
 Ligaments, injuries of, 1158  
 Lindau's syndrome, 721  
 Linear accelerator, 1319  
 Linitis plastica, 14  
 Lip, carcinoma of, 846, 876  
   cleft, 844  
   tumours of, 845-847  
     upper, congenital frænum of, 844  
 Lipadema, 1030-1031  
 Lipoid deposits, in cholesterolemia, 54, 57  
 Lister's curved steel sounds, 409  
 Litholapaxy, for vesical calculus, 360  
 Lithotomy position, 370  
 Littre's hernia, 199  
 Liver, 77-93  
   abscess, 82  
     diagnosis from hydatid cyst, 84  
   actinomycosis of, 82  
   biopsy, 78  
   carcinoma of, 85-87  
     primary, 85  
     secondary, 86-87  
   cirrhosis of, 87-93  
     carcinoma and, 85  
     venous occlusion in, 108  
   cysts of, 82-85  
   function tests, 77-78  
    hæmangioma of, 85, 1022  
   "hornail," 85  
   hydatid disease of, 82-85  
   polycystic disease of, 85  
   pyogenic infection of, 80  
   rupture of, 78-79  
   tumours of, benign, 82-85  
     malignant, 85-87  
 Lividity, in intracranial compression,  
 706  
 Lobectomy, 558-563  
   for bronchiectasis, 492-494  
   for chronic empyema, 488  
   for hydatids of lung, 475  
   for lung cysts, 513  
   lower left, 559  
     right, 561  
     middle, 560-561  
   post-operative care, 563  
   upper left, 558-559  
     right, 559-560



Logan's bow, 245-246, 248

Lordosis, 1081

Lotheissen's operation, 217

Ludwig's angina, 862, 870

Lumbago, 781

Lumbar disc protrusion, 781-783

treatment of, 784-785

Lumbar puncture, in brain abscess, 61

in cauda equina ependymoma, 791

in epidural abscess, 787

in extradural hæmatoma, 641

in head injuries, 645

in raised intracranial pressure, 703

704

in spinal cord compression, 772

in subarachnoid hæmorrhage, 645,

745-746

Lumbarisation, 1086

Lunate, fractures of, 1217

Lung abscess, 457-475

actinomyces causing, 474

etiology of, 460-461, 466

aspiration or acute putrid, 459-464

chronic, 468-470

external drainage of, 467-468

Friedlander's bacillus causing, 773

hydatid, 474-475

malignant, 457-459, 501

prophylaxis of, 464-465

putrid, 468

staphylococcal, 470-473

Lungs, adenoma of, 508-509

"cannon ball" secondary due to renal car-

cinoma, 349

carcinoma of, 498

cysts of, 511-517

congenital, 511-513

hamartoma of, cartilaginous, 509

vascular, 509-510

hydatids of, 517

inflammatory diseases of, brain abscess

and, 659

intracranial suppuration and, 665

resection of, 549-565

complications of, 545, 565

for bronchial carcinoma, 504-505,

507

for cysts, 513

for pulmonary tuberculosis, 543-546

pre-operative care, 561-563

segmental, 561, 563

tumours of, innocent, 508-510

Lymphadenitis, 1024-1036

acute, 1024

chronic, 1024-1026

iliac, diagnosis from appendicitis, 6

mesenteric, 5, 107

syphilitic, 1025

tuberculous, 107, 871-875, 1025

Lymphadenoma, 527

See also Hodgkin's disease.

Lymphadenopathy in neck, diagnosis from

parotid tumours, 842

Lymphangiography, technique of, 1023

Lymphangioma, capillary, 1026

cavernous, 1026

treatment of, 1027

Ly

Lyn

Lyn

Lyn

Lyn

Lyn

McBurney's

point, 4

McCarthy's c

panendosco

McKenzie-Cu

McMurray's o

subtrochanter

test, 1123

Macrocheilia, 845,

Macroglossia, 845

congenital, 1030

Macromastia, 942

Macropsia and micro

Main en griffe, 816

Maisonneuve's fracture,

Malgaigne's bulge, 208

Malleolus, medial, fract

third, fractures of, 1272

Mandible, dislocation of, 1

giant-cell, tumour of, 767

Mantoux test, in tuberculo

Marble bones, 1034

March fracture, 1282

Marcoumar, 1016, 1017

Manipulation, in congenital d

hip, 1114

in osteoarthritis, 1056

in torticollis, 1039

Marie's spondylitis ankylopoietica,

in, 786

Marion's disease, 396-399

Marsalid, in pulmonary excision, 643

Marsupialisation, in pseudocyst of pan

71

Martin's syringe, 479

Mastectomy, 956-957

- Mastitis carcinomatosa**, 913  
 chronic (cystic), 916  
 interstitial, 916  
 of male breast, 919  
 periperal, 913
- Mastoiditis**, 663  
 brain abscess and, 659
- Mata's operation**, 947-950
- Mayall's hernia**, 201
- Mayo's operation**, for hallux valgus, 1132-1133  
 for umbilical hernia, 218-219
- Meatitis**, 314-315
- Meiotomy**, 315, 422
- Meckel's cave**, meningiomas in, 641, 643  
 diverticulum, 109-110, 263-271  
 inflammation of, 271  
 diagnosis from appendicitis, 5  
 intussusception and, 291, 293, 295  
 neoplasms of, 109, 111  
 operation for removal of, 271  
 ulceration of, 271  
 volvulus of, 270-271
- Meconium ileus**, 267  
 peritonitis, 267-268
- Median nerve compression**. *See* Carpal tunnel syndrome.
- Mediastinotomy**, 927
- Mediastinum**, chondroma of, 524  
 cysts and tumours of, 518-524  
 fibroma of, 524  
 haemangioma of, 524  
 lipomas of, 523  
 neurogenic tumours of, 523-525  
 plasmocytoma of, 524  
 teratomas and dermoids of, 521-523  
 tumours and cysts of, 518-524
- Medulla oblongata**, intrinsic lesions of, 681
- Medulloblastoma** of posterior fossa, 721
- Megaloureter**, congenital, 304, 363
- Melena**, in gastric carcinoma, 18  
 in peptic ulcer, 25, 36
- Melanoma**, benign, 833  
 malignant, 834-835
- Ménière's syndrome**, 684  
 diagnosis from temporal lobe epilepsy, 110  
 tumours in cerebello-pontine angle, 724
- Meningiomas**, 666, 695, 696, 697  
 angioblastic, 709  
 angiograms of, 701  
 diagnosis from osteoma, 764  
 dumb-bell, 723  
 endotheliomatous, 709  
 en plaque, 715  
 fibroblastic, 709  
 myxomatous, 709  
 of foramen magnum, 773  
 of Meckel's cave, 681, 683  
 of olfactory groove, 709  
 of posterior fossa, 722  
 of temporal region, pathology and symptoms of, 713-715  
 of tuberculum sellæ, 709-710  
 parasagittal, 709, 717-718  
 pathology of, 708-710
- Meningiomas, peritumoral**, 714  
 skull "tumour" secondary to, 767, 769  
 suprasellar, 709-710
- Meningitis**, 657-659  
 anaemia in, 666  
 auditory nerve involvement in, 647  
 diagnosis of, 654  
 labyrinthitis in, 649  
 meningococcal, 654  
 papilloedema and, 671  
 pneumococcal, 654, 659  
 signs of, 645  
 subdural hygroma following, 644-645  
 treatment of, 659  
 tuberculous, 654-659
- Meningocele**, 245, 792, 1035  
 traumatic, 646-647
- Mental disease**, surgery of, 762-763
- Meralgia paraesthetica**, 621
- Mesentery**, 107-109  
 cysts of, 275  
 tumours of, 105-106
- Mestison**, in myasthenia gravis, 914
- Metacarpals**, dislocations of, 1167  
 fractures of, 1217-1218, 1282  
 osteoarthritis of, 1106-1107
- Metatarsalgia**, anterior, 1149-1150  
 Morton's 1150-1151
- Metatarsus primus varus**, 1139, 1141, 1151
- Methyl thiouracil**, 901
- Micturition**, apparatus for study of, 383-384  
 frequency of, 326, 344-345, 440  
 physiology of, 343
- Middle trunk palsy**, 813
- "Milk" leg**, 994
- Millin's retropubic prostatectomy**, 396-397, 398
- Milroy's disease**, 1030
- Mitral stenosis**, aetiology of, 593  
 investigations in, 595-596  
 mesenteric arterial occlusion in, 108  
 post-operative sequelæ, 604  
 symptoms of, 594-595, 596, 597  
 treatment of, surgical, 593-604  
 operative technique, 598-603  
 pre-operative preparation, 597
- Moles**, 833
- Molluscum fibromum**, 823  
 pseudocarcinomatous, 830  
 sebaceous, 830-831
- Mönckeberg's sclerosis**, 963, 970
- Mongolism**, in infantile duodenal occlusion, 259
- Moniliasis**, 845
- Monteggia fracture**, 1220
- Moon's turreted molars**, 1037
- Morgagni, hydatid of**, torsion of, 233
- Morphine**, sedative dosage according to age, 1295
- Morton's metatarsalgia**, 1150-1151
- Mouth**, cancer of, 845-847  
 cystic swellings of, 844  
 inflammation of, 844-845  
 tumours of, 845-847
- Mucocoele**, appendiceal, 3, 12  
 ruptured, 105
- Mule spinners' cancer**, 433

- Logan's bow, 245-246, 248  
 Lordosis, 1081  
 Lotheissen's operation, 217  
 Ludwig's angina, 862, 870  
 Lumbago, 781  
 Lumbar disc protrusion, 781-783  
     *treatment of*, 784-785  
 Lumbar puncture, in brain abscess, 661  
     in cauda equina ependymoma, 790  
     in epidural abscess, 787  
     in extradural hematoma, 641  
     in head injuries, 645  
     in raised intracranial pressure, 703-704  
     in spinal cord compression, 772  
     in subarachnoid hemorrhage, 645, 745-746  
 Lumbarisation, 1086  
 Lunate, fractures of, 1217  
 Lung abscess, 457-475  
     actinomyces causing, 474  
     etiology of, 460-461, 466  
     aspiration or acute putrid, 459-464  
     chronic, 468-470  
     external drainage of, 467-468  
     Friedlander's bacillus causing, 773  
     hydatid, 474-475  
     malignant, 457-459, 501  
     prophylaxis of, 464-465  
     putrid, 468  
     staphylococcal, 470-473  
 Lungs, adenoma of, 508-509  
     " cannon ball " secondary due to renal carcinoma, 349  
     carcinoma of, 498  
     cysts of, 511-517  
         congenital, 511-513  
     hamartoma of, cartilaginous, 509  
         vascular, 509-510  
     hydatids of, 517  
     inflammatory diseases of, brain abscess and, 659  
         intracranial suppuration and, 665  
     resection of, 540-565  
         complications of, 545, 563  
         for bronchial carcinoma, 504-505, 507  
         for cysts, 513  
         for pulmonary tuberculosis, 543-546  
         pre-operative care, 561-563  
         segmental, 561, 563  
     tumours of, innocent, 508-510  
 Lymphadenitis, 1024-1036  
     acute, 1024  
     chronic, 1024-1026  
     iliac, diagnosis from appendicitis, 6  
     mesenteric, 5, 107  
     syphilitic, 1025  
     tuberculous, 107, 871-875, 1025  
 Lymphadenoma, 527  
     *See also* Hodgkin's disease.  
 Lymphadenopathy in neck, diagnosis from parotid tumours, 842  
 Lymphangiography, technique of, 1023  
 Lymphangioma, capillary, 1026  
     cavernous, 1026  
     *treatment of*, 1027  
 Lymphangitis, acute, 1023  
     chronic, 1023-1024  
     recurrent, lymphoedema due to, 1031  
 Lymphatics, 1023  
     of neck, cysts of, 868  
     of stomach, 15  
     tumours of, 1026-1029  
         benign, 1026-1027  
         malignant, 1027-1029  
         secondary, 1028-1029  
 Lymphoblastoma, in children, 527  
 Lymphoedema, 1029-1032  
     acute, 1029  
     artefacta, 1031  
     causes of, 1030-1031  
     chronic, 1029  
     management of, 1031-1032  
     parasitic, 1031  
     post-operative, 1031  
     precox, 1030  
     " spontaneous," 1030  
 Lymphogranuloma inguinale, 1025-1026  
     venereum, 163, 168, 169  
 Lymphoid tissue, tumours of, 526-527  
 Lymphoma, 1027  
 Lymphorrhoea, 1026, 1032  
 Lymphosarcoma, 527, 876, 1027
- M
- McBurney's gridiron incision, 7-8  
     point, 4  
 McCarthy's electrotono, 397, 398  
     panendoscope, 409  
 McKenzie-Cushing clip outfit, 73  
 McMurray's osteotomy, 1037  
     subtrochanteric osteotomy, 1121  
     test, 1123  
 Macrocheilia, 845, 1030  
 Macroglossia, 845  
     congenital, 1030  
 Macromastia, 942  
 Macropsia and micropsia, 758  
 Main en griffe, 816  
 Maisonneuve's fracture, 1263  
 Malgaigne's bulge, 208  
 Malleolus, medial, fractures of, 1271-1272  
     third, fractures of, 1272-1273  
 Mandible, dislocation of, 1185  
     giant-cell, tumour of, 765, 769  
 Mantoux test, in tuberculosis of bone, 1047  
 Marble bones, 1034  
 March fracture, 1282  
 Marcoumar, 1016, 1017  
 Manipulation, in congenital dislocation of hip, 1114  
     in osteoarthritis, 1056  
     in torticollis, 1089  
 Marie's spondylitis ankylopoietica, sciatica in, 786  
 Marion's disease, 396-399  
 Marsalid, in pulmonary excision, 543  
 Marsupialisation, in pseudocyst of pancreas, 71  
 Martin's syringe, 479  
 Mastectomy, 936-957

- Nerve(s), phrenic, lesions of, 811  
 posterior interosseous, injury of, 820, 1165-1166  
 radial, 820  
 sciatic, 821  
 sheaths, neural fibrosarcoma of, 824  
 spinal, accessory, 692-693  
 to serratus anterior, lesions of, 814-815  
 trigeminal, 674, 675-681, 746  
   division of sensory root, 676-678  
 ulnar, 816  
 vagus, 691
- Nervous system, autonomic, 794-795  
   investigations, 795-796
- Neural foramina, occlusion of, 819
- Neuralgia, glossopharyngeal, 690-691  
   post-herpetic, 679  
   post-thrombotic, 1001  
   trigeminal, 675-678
- Neurapraxia, 803, 809
- Neurilemmoma, 721, 788, 823  
   *See also* Neurofibroma.
- Neurinoma, acoustic, 683, 687, 721
- Neuritis, brachial, acute, 813  
   complicating bony injuries, 1204-1205  
   ulnar, traumatic, 817
- Neuroblastoma, 800-802
- Neurofibroma, 788-789  
   of neck, 869  
   of peripheral nerves, 823  
   peripheral, 824
- Neurofibromatosis, 823, 1035
- Neuroma, fusiform, in peripheral nerve injury, 809
- Neuro-mylitis optica, 667. *See also* Devic's disease.
- Neuropathy, carcinoma of bronchus and, 819-820
- Neurotomy, Stöffel's, 822-823
- Nipples, 941-942  
   affections of, 942  
   discharge from, 942  
   Paget's disease of, 942-944  
   supernumerary, 941
- Nissl's degeneration, in peripheral nerve injury, 803
- Noradrenaline, 930

## O

- Odontomes, 857
- Oedema, chylous, 1030  
   complicating body injuries, 1205  
   pulmonary, bronchoscopy for, 455
- Oesophagitis, reflux, in infancy and childhood, 252-254
- Oesophagojejunostomy, for carcinoma of cardia, 19
- Oesophagoscopy, 576-578, 580, 591
- Oesophagus, abnormalities of, congenital 250-254  
   atresia of, 250-252  
   absence of, 250  
   carcinoma of, 583-590  
     empyema and, 477  
     investigations for, 544-585

- Oesophagus, carcinoma of, operative technique, 585-589  
   post-operative management, 589-590  
   symptoms of, 584  
   foreign bodies in, 295-296  
   leiomyoma of, 590-591  
   peptic ulcer of, empyema and, 477  
   rupture of, oesophagoscopy and, 576-578  
   spontaneous, 577-578  
   stricture, simple, 578-580  
   tumours of, innocent, 590-591  
   varices of, 83-89
- Olecranon bursitis, 1101
- Oligodendroglioma, pathology of, 708
- Oliguria, 333
- Oliver's surgical procedure, for occlusion of internal carotid artery, 754
- Ollier's disease, 1070, 1078
- Omentum, great, 106-107
- Onychogryphosis, 1154
- Oophorectomy, adrenalectomy and, for breast cancer, 938
- Ophthalmoplegia, 746  
   causes of, 673-675
- Ophthalmoscopy, 666
- Oppenheim's sign, 696
- Optic atrophy, causes of, 667-670  
   consecutive, 670  
   primary, 666-667  
   secondary, 670  
   chiasm, injuries of, 653-654
- Orbit, tumours of, 669-670, 671
- Orchidectomy, for epididymitis, 425, 426  
   for tuberculosis, 444
- Orchidopexy, for undescended testis, 231-232
- Orethritis, 233, 426
- Oropharynx, malignant growths of, 832
- Osgood-Schlatter disease, 1133
- O'Shaughnessy forceps, 532
- Osler-Rendu syndrome, 1021
- Osteitis deformans, 687, 767, 1064-1065  
   diagnosis from hyperparathyroidism, 926  
   *See also* Paget's disease of bone.
- Osteosarcoma, 858  
   cytic, 769, 1078  
   non-tuberculous, diagnosis from Pott's disease, 1092  
   of maxilla, in infancy, 854  
   syphilitic, diagnosis from sarcoma, 1072  
   tuberculous, 1047-1048
- Osteo-arthritis, 1052-1058  
   acute generalised, 1061  
   cysts in, 1078  
   fractures of femur, complicated by, 1246  
   muscular spasm in, relief of, 1055-1056  
   of elbow, 1100  
   of hip, 1119-1120  
   of knee-joint, 1127, 1131, 1134-1135  
   of metacarpocarpal joint, 1106-1107  
   of spine, 1094-1095  
   pathology of, 1052-1053  
   primary, 1053-1054  
   secondary, 1058  
   treatment of, 1054-1058  
   surgical, 1057-1058

- Mumps, 835-836  
   pancreatitis, complication of, 67  
 Muscles, atrophy of, progressive, 694, 819  
   biopsy of, in peripheral nerve injury, 807  
   dystrophies of, 807, 820  
   M.R.C.S.'s scheme for assessment of  
     power of, 804  
     wasting of, differential diagnosis, 818-820  
 Myasthenia gravis, bulbar type, 913-914  
   characteristics of, 912-913  
   diagnostic tests, 914  
   thymus in relation to, 912-921  
   treatment of, 914-915  
     post-operative, 919  
     pre-operative, 916-917  
 Myelocoele, 792  
 Myelography in disc protrusion, 783  
   in space-occupying lesions of spinal cord,  
     772  
   in spinal meningioma, 788  
 Myelomatosis, multiple, 1075  
   diagnosis from hyperparathyroidism,  
     926  
   of jaw, 858  
   of skull, 760  
 Myelomeningocele, 792, 1035  
 Myer's type of stripper, 1007  
 Myodil, 762  
 Myositis fibrosa, 1201-1203  
   oesificans, 1200-1201  
 Myringotomy, 665  
 Myxœdema, following thyroid operation,  
   909
- N
- Nævus, 1018  
   pigmented, 833-834  
   spider, 1019  
 Nail, ingrowing, of toe, 1154  
   of finger, infections around, 1290-1291  
 Nasopharynx, carcinoma of, 882  
   tumours of, cranial nerve palsies in, 683  
   optic atrophy and, 668  
 Navicular, fractures of, 1215-1217  
 Neck, actinomycosis of, 870  
   œrocele of, 868-869  
   block dissection of, for carcinoma of  
     thyroid, 899  
   technique of, 876-877  
   boil or furuncle of, 861  
   carbuncle of, 861-862  
   carcinomatous lymph glands of, 876  
   cellulitis of, 862-863  
   cysts of, 864-865, 868  
   lipomas of, 869  
   lymphosarcoma of, 875  
   neurofibromas of, 865  
   pneumatocoele of, 869  
   stiffness of, causes of, 645  
   in intracranial pressure, 706  
   swellings of, 861-870  
   chronic, 864-870  
   tuberculosis of, 871-877  
 Necrosis, avascular, in fractures, 1180-  
   1181, 1204, 1216, 1245  
   surgery of, 976-977
- Negus œsophagoscope, 578  
 Neomercazole, 901  
 Neomycin, in bowel sterilization, 150-151  
 Neonatal staining, 1019  
 Nephrectomy, 356  
   for calculi, 347  
   for carcinoma, 351  
 Nephritis, chronic, fundi in, 671  
 Nephroblastoma, 304-306, 347  
 Nephrocalcinosis, 343  
 Nephrolithotomy, 346-347  
 Nephrostomy, 334, 356  
 Nephroureterectomy, for tumours of renal  
   pelvis, 351  
   of ureter, 361  
 Nerve(s), acoustic, tumours of, 721-722, 823  
   auditory, 685-686  
   deafness, traumatic, 653  
   division of vestibular part, for Ménière's  
     syndrome, 689  
   tests of function, 686  
   bulbar accessory, 691  
   cervical, palsies, 774  
   cranial, 666-694  
     abducens, 673, 674, 675  
     fifth, 674, 675-681  
     fourth, 673  
     palsies, 774  
     sixth, 673, 674, 675  
     third, 673  
   digital, injuries of, 821  
   facial, 681-682  
   femoral, lesions of, 821  
   glossopharyngeal, 690  
   grafting, 810-811  
   hypoglossal, 693-694  
   laryngeal, 691  
     recurrent, 885-886  
     injury of, 908-909  
   lateral, cutaneous, of thigh, lesions of, 821  
   median, 815  
   olfactory, 666  
   optic, 666-673  
     glioma of, 667  
     injuries of, 653-654  
     opaque fibres of, diagnosis from papill-  
       œdema, 672  
   peripheral, block, 795-796  
   injuries of, 803-818  
     anatomical anomalies and, 804-805  
     biopsy of, 807  
     causalgia following, 806  
     electromyography of, 806-808  
     modes of, 803  
     muscle biopsy in, 807  
     physiotherapy and rehabilitation in,  
       811  
   reaction of degeneration in, 806, 803  
   sequelæ of, 803-804  
   signs of, 804  
   splinting of, 808  
   sweat-test in, 806-807  
   technique of repair, 810-811  
   treatment of, 807-808  
   vasomotor and trophic changes in, 805  
   operations on, 809-810  
   tumours of, 823

- Nerve(s), phrenic, lesions of, 811  
 posterior interosseous, injury of, 820,  
 1165-1166  
 radial, 820  
 sciatic, 821  
 sheaths, neural fibrosarcoma of, 824  
 spinal, accessory, 692-693  
 to serratus anterior, lesions of, 814-815  
 trigeminal, 674, 675-681, 746  
   division of sensory root, 675-678  
 ulnar, 816  
 vagus, 691
- Nervous system, autonomic, 794-795  
   investigations, 795-796
- Neural foramina, occlusion of, 819
- Neuralgia, glossopharyngeal, 690-691  
   post-herpetic, 679  
   post-thrombotic, 1001  
   trigeminal, 675-678
- Neuroapraxia, 803, 809
- Neurilemmoma, 721, 789, 823  
   *See also* Neurofibroma.
- Neurinoma, acoustic, 683, 687, 721
- Neuritis, brachial, acute, 813  
   complicating bony injuries, 1204-1205  
   ulnar, traumatic, 817
- Neuroblastoma, 800-802
- Neurofibroma, 789-789  
   of neck, 869  
   of peripheral nerves, 823  
   peripheral, 824
- Neurofibromatosis, 823, 1035
- Neuroma, fusiform, in peripheral nerve  
 injury, 809
- Neuro-myelitis optica, 667. *See also* Devic's  
 disease.
- Neuropathy, carcinoma of bronchus and,  
 819-820
- Neurotomy, Stöffel's, 822-823
- Nipples, 941-942  
   affections of, 942  
   discharge from, 942
- Paget's disease of, 942-944  
   supernumerary, 941
- Nissl's degeneration, in peripheral nerve  
 injury, 803
- Noradrenaline, 930

## O

- Odontomes, 857
- Oedema, chylous, 1030  
   complicating body injuries, 1205  
   pulmonary, bronchoscopy for, 455
- Oesophagitis, reflux, in infancy and child-  
 hood, 252-254
- Oesophagojejunostomy, for carcinoma of  
 cardia, 19
- Oesophagoscopy, 576-578, 580, 591
- Oesophagus, abnormalities of, congenital  
 250-254  
   atresia of, 250-252  
   absence of, 250  
   carcinoma of, 583-590  
     empyema and, 477  
     investigations for, 544-585

- Oesophagus, carcinoma of, operative tech-  
 nique, 585-589  
   post-operative management, 589-590  
   symptoms of, 584  
   foreign bodies in, 255-256  
   leiomyoma of, 590-591  
   peptic ulcer of, empyema and, 477  
   rupture of, oesophagoscopy and, 576-578  
   spontaneous, 577-578  
   stricture, simple, 578-580  
   tumours of, innocent, 590-591  
   varices of, 89-89
- Olecranon bursitis, 1101
- Oligodendroglioma, pathology of, 709
- Oliguria, 333
- Oliver's surgical procedure, for occlusion of  
 internal carotid artery, 754
- Ollier's disease, 1070, 1078
- Omentum, great, 106-107
- Onychogryphosis, 1154
- Oophorectomy, adrenalectomy and, for  
 breast cancer, 938
- Ophthalmoplegia, 746  
   causes of, 673-675
- Ophthalmoscopy, 666
- Oppenheim's sign, 696
- Optic atrophy, causes of, 667-670  
   consecutive, 670  
   primary, 666-667  
   secondary, 670  
   chiasm, injuries of, 653-654
- Orbit, tumours of, 669-670, 671
- Orchidectomy, for epididymitis, 425, 426  
   for tuberculosis, 444
- Orchidopexy, for undescended testis, 231-  
 232
- Orchitis, 233, 426
- Oropharynx, malignant growths of, 882
- Osgood-Schlatter disease, 1133
- O'Shaughnessy forceps, 532
- Oster-Rendu syndrome, 1021
- Osteitis deformans, 687, 767, 1064-1065  
   diagnosis from hyperparathyroidism,  
   926  
   *See also* Paget's disease of bone.
- fibrosa, 858  
   cystica, 769, 1078
- non-tuberculous, diagnosis from Pott's  
 disease, 1092
- of maxilla, in infancy, 854
- syphilitic, diagnosis from sarcoma, 1072
- tuberculous, 1047-1048
- Osteo-arthritis, 1052-1058  
   acute generalised, 1061  
   cysts in, 1078  
   fractures of femur, complicated by, 1246  
   muscular spasm in, relief of, 1055-1056  
   of elbow, 1100  
   of hip, 1119-1120  
   of knee-joint, 1127, 1131, 1134-1135  
   of metacarpocarpal joint, 1106-1107  
   of spine, 1094-1095  
   pathology of, 1052-1053  
   primary, 1053-1054  
   secondary, 1058  
   treatment of, 1054-1058  
   surgical, 1057-1058

- Osteoarthropathy, pulmonary, hypertrophic,** 500, 502
- Osteochondritis dissecans,** 1130-1131  
of elbow, 1100-1101  
juvenilis, 1115  
diagnosis from Pott's disease, 1092  
of tarsal navicular, 1145  
of tibial tubercle, 1132, 1133
- Osteochondroma,** 1069-1070
- Osteoclastoma,** 1074-1075  
of skull, 765. *See also* Giant-cell tumour.
- Osteoclasts,** 1180
- Osteogenesis imperfecta,** 1034-1035, 1066  
diagnosis from hyperparathyroidism, 926
- Osteoma,** 1069-1070  
cancellous or spongy, 764  
ivory, 764  
of skull, 764-765, 769
- Osteomalacia,** 1066, 1067  
diagnosis from hyperparathyroidism, 926
- Osteomyelitis, acute,** 1039, 1040  
chronic, 769, 1043-1045  
fulminating, 1039  
haematogenous, 1038-1045  
complications of, 1042-1043  
differential diagnosis of, 1040-1041  
pathology of, 1038-1039  
radiological appearances of, 1039  
treatment of, 1041-1042  
of pelvis, diagnosis from appendicitis, 8  
of skull, 655, 658, 660  
subacute, 1040
- Osteopetrosis,** 1034
- Osteoporosis,** 1039  
in Cushing's syndrome, 931  
in hyperparathyroidism, 925  
senile, 1087, 1093
- Osteopsathyrosis.** *See* Fragilitas ossium.
- Osteotomy for osteo-arthritis,** 1057
- Otitis externa, primary,** 687  
media, 665  
brain abscess and, 659  
chronic, fistula sign in, 689
- Otorrhoea, cerebrospinal fluid,** 651
- Otosclerosis,** 687
- Ovary, cyst of, twisted, diagnosis from**  
appendicitis, 8  
from cholecystitis, 51
- Oxytetracycline,** 1316  
in biliary disease, 63  
in pancreatitis, 69  
in peritonitis, 35, 101  
*See also* Terramycin.
- Oxyuris vermicularis infestation, of appendix,** 11
- P**
- Pachymeningitis,** 653, 656  
spinal, acute, 787
- Paget's disease of bone,** 687, 767, 1064-1065  
bony repair and, 1178  
diagnosis from carcinoma of prostate, 401  
from Pott's disease, 1093  
of jaw, 859
- Paget's disease of bone,**  
optic nerve compression in, 668  
*See also* Osteitis deformans.
- Paget's disease of nipple,** 942-944
- Pain, dental, neurosurgery and,** 678  
intractable, leucotomy for, 763  
investigation of, in neurological disease, 319-320  
renal, 319, 320  
visceral, 319-320
- Palate, cancer of,** 847, 856  
congenital clefts of, 856  
cylindroma of, 856  
perforation of, 856  
tumours of, 856
- Palsy, bulbar,** 692, 694  
external popliteal, 1265  
facial, operations for, 684-685  
physiotherapy for, 695  
rectus, external, 706
- Pancoast's syndrome,** 500, 503, 819
- Pancreas, aberrant tissue of,** 85-86  
adenocarcinoma of, 73  
anatomy of, 65  
annular, 66, 259-260, 261  
carcinoma of, 73-76  
chronic pancreatitis simulating, 70  
cysts of, 71  
head of, resection of, for carcinoma, 75-76  
injuries of, 66  
polycystic disease of, 71  
sympathectomy in diseases of, 800  
tumours of, 71-76  
benign, 72  
islet-cell, 72-73
- Pancreatectomy, total, in carcinoma of**  
pancreas, 76
- Pancreatitis, acute,** 66-69  
diagnosis from perforated peptic ulcer, 33  
from small gut obstruction, 126  
treatment of, 68-69  
chronic, 69-71  
relapsing, 70  
simulating carcinoma, 70-71  
treatment of, 69, 70-71  
in cholecystitis, 50, 54
- Pansinusitis,** 664
- Papilloedema,** 670-672  
in brain abscess, 660  
in head injuries, 639, 643  
in raised intracranial pressure, 706  
unilateral, causes of, 672
- Para-aminosalicylic acid, in tuberculosis,**  
ulcerative, 117. *See also* PAS.
- Paracentesis abdominis, in constrictive pericarditis,** 607
- Parasthesia, acoustic nerve tumours caus-**  
ing, 722
- Paralysis, abductor and adductor of the**  
larynx, 692  
Erb-Duchenne, 812  
facial, 653, 694  
Klumpke's, 812  
laryngeal, unilateral total, 691
- Paraphimosis,** 313-314, 420-421

- Parathyroid, adenoma, 528**  
glands, 922-923  
tetany, post-operative, 909  
*See also* Hypoparathyroidism.  
tumours of, 923
- Parham's band, 1197**
- Parieto-occipital syndrome, 715-717**  
tumours, 717
- Paronychia, 1290-1291**
- Parotid adenolymphoma, 841**  
adenoma, 684, 840  
pleomorphic, 840, 841, 842  
carcinoma, 694, 841  
cylindroma, 841  
fistula, 840
- Parotidectomy, 843**
- Parotitis, acute and recurrent, 836-838**  
acute epidemic, 835-836
- Parrot's nodes, 1037**
- PAS, 875**  
in pericarditis, tuberculous, 607  
in peritonitis, tuberculous, 103  
in pulmonary tuberculosis, 543  
in tuberculosis, 1049  
in urinary tuberculosis, 442  
*See also* Para-aminosalicylic acid.
- Past-pointing, 688**
- Patella, dislocations of, 1170-1171**  
recurrent, 1132  
excision of, 1258  
fractures of, 1257-1259
- Patent ductus arteriosus, 626-629**  
technique of operation for, 627-629
- Paterson-Brown Kelly syndrome, 883**  
*See also* Plummer-Vinson syndrome.
- Paul-Mikulicz operation, 151, 152**  
technique of, 155
- Pel-Ebstein curve, 1028**
- Pellegrini-Stella's disease, 1127**
- Pelvis, fractures of, 1234-1238**  
complications of, 1237  
ring of, fractures, 1234-1237
- Penicillin, 1314-1315**  
in actinomycosis, 82  
in appendicitis, 9  
in cholecystectomy, 51  
in peritonitis, 101
- Penis, burns of, treatment of, 1297**  
carcinoma of, 417-419  
diseases of, 417-422  
operations on, 421-422
- Pentothal, gangrene from, 994**
- Pepper's syndrome, 800-801**
- Peptic ulcer, 20-45**  
acute, 23  
aetiology of, 20-22  
bleeding from, 36-38  
chronic, 21, 23-24  
clinical features of, 24-25  
complications of, 31-45  
diagnosis of, 25  
differential, 25-27  
from carcinoma, 15, 18, 26, 27  
from cholecystitis, 50, 54  
obstructive, 38-39  
of oesophagus, empyema and, 477  
pathology of, 22-24
- Peptic ulcer, perforated, 31-36, 39-40**  
clinical features of, 31-33  
complications of, 35  
diagnosis, differential, 33  
from appendicitis, 5  
from pancreatitis, 69  
treatment of, 33-35  
psychoneurosis and, 27  
sites of, 22  
treatment of, 27-31, 33-35
- Pericardectomy, 607-609**
- Pericarditis, constrictive, 605-609**  
diagnosis of, 605-606  
pericardectomy for, 607-609  
portal hypertension in, 87  
symptoms of, 605  
tuberculous, 605-607
- Pericardium, cysts of, 523**
- Perineum, burns of, treatment of, 1297**
- Periorbitis, syphilitic, 1037**
- Peritoneum, 99-106**  
adhesions, 106  
loose bodies in, 107  
sutures for, 104  
tumours of, 105
- Peritonitis, biliary, 262**  
causes of, 99  
clinical features of, 100  
diagnosis from appendicitis, 288  
from intestinal obstruction, 126  
gonococcal, 101  
hernia and, 201  
in appendicitis, 7, 31  
in infancy, 268-269  
meconium, 267-268  
paralytic ileus and, 129  
pathology of, 99-100  
pneumococcal, 101, 288  
post-operative, in rectal carcinoma, 175  
special types of, 101-103  
streptococcal, 101  
treatment of, 100-101  
tuberculous, 101-103, 117  
chronic, adhesive and ascitic, 102
- Pernio, 979-980**
- Perniosis, chronic, 1012**
- Peroneal spasm, 1144**
- Perth's disease, 1115-1116**  
diagnosis from tuberculous arthritis, 1118
- Petrochanteric fractures, 1246-1247**
- Pes planus, 1143-1144**
- Petit mal, 756-757**
- Peyronie's disease, 421**
- Phaeochromocytoma, 936-937**
- Phalanges, dislocations of, 1167-1168**  
fractures of, 1219
- Pharyngolaryngectomy, 883**
- Pharynx, cancer of, 882-883, 884**
- Phenol-sulphone-phthalein excretion, as test of renal function, 327**
- Phentolamine test, for phaeochromocytoma, 936**
- Phimosi, 420**  
acquired, 313  
congenital, 312-313
- Phlebitis, iliac, diagnosis from appendicitis, 6**



- Osteoarthropathy, pulmonary, hypertrophic, 500, 502
- Osteochondritis dissecans, 1130-1131  
of elbow, 1100-1101  
juvenile, 1115  
diagnosis from Pott's disease, 1092  
of tarsal navicular, 1145  
of tibial tubercle, 1132, 1133
- Osteochondroma, 1069-1070
- Osteoclastoma, 1074-1075  
of skull, 765. *See also* Giant-cell tumour.
- Osteoclasts, 1180
- Osteogenesis imperfecta, 1034-1035, 1066  
diagnosis from hyperparathyroidism, 928
- Osteoma, 1069-1070  
cancellous or spongy, 764  
ivory, 764  
of skull, 764-765, 769
- Osteomalacia, 1068, 1067  
diagnosis from hyperparathyroidism, 926
- Osteomyelitis, acute, 1039, 1040  
chronic, 769, 1043-1045  
fulminating, 1039  
haematogenous, 1038-1045  
complications of, 1042-1043  
differential diagnosis of, 1040-1041  
pathology of, 1038-1039  
radiological appearances of, 1039  
treatment of, 1041-1043  
of pelvis, diagnosis from appendicitis, 8  
of skull, 833, 838, 889  
subacute, 1040
- Osteopetrosis, 1034
- Osteoporosis, 1039  
in Cushing's syndrome, 931  
in hyperparathyroidism, 925  
senile, 1087, 1093
- Osteosarcoma. *See* Fragilitas ossium.
- Osteotomy for osteo-arthritis, 1057
- Otitis externa, primary, 687  
media, 665  
brain abscess and, 659  
chronic, fistula sign in, 689
- Otorrhoea, cerebrospinal fluid, 651
- Otosclerosis, 687
- Ovary, cyst of, twisted, diagnosis from  
appendicitis, 5  
from cholecystitis, 51
- Oxytetracycline, 1316  
in biliary disease, 63  
in pancreatitis, 69  
in peritonitis, 35, 101  
*See also* Terramycin.
- Oxyuris vermicularis infestation, of appendix, 11
- P**
- Pachymeningitis, 655, 656  
spinal, acute, 787
- Paget's disease of bone, 687, 767, 1064-1065  
bony repair and, 1178  
diagnosis from carcinoma of prostate, 401  
from Pott's disease, 1093  
of jaw, 859
- Paget's disease of bone,  
optic nerve compression in, 668  
*See also* Osteitis deformans.
- Paget's disease of nipple, 942-944
- Pain, dental, neurosurgery and, 678  
intractable, leucotomy for, 763  
investigation of, in neurological disease, 319-320  
renal, 319, 320  
visceral, 319-320
- Palate, cancer of, 847, 856  
congenital cleft of, 856  
cylindroma of, 858  
perforation of, 858  
tumours of, 856
- Palsy, bulbar, 692, 694  
external popliteal, 1265  
facial, operations for, 684-693  
physiotherapy for, 685  
rectus, external, 706
- Pancoast's syndrome, 500, 503, 819
- Pancreas, aberrant tissue of, 65-66  
adenocarcinoma of, 73  
anatomy of, 65  
annular, 66, 259-260, 261  
carcinoma of, 73-76  
chronic pancreatitis simulating, 70  
cysts of, 71  
head of, resection of, for carcinoma, 75-76  
injuries of, 66  
polycystic disease of, 71  
sympathectomy in diseases of, 800  
tumours of, 71-76  
benign, 72  
islet-cell, 72-73
- Pancreatotomy, total, in carcinoma of pancreas, 76
- Pancreatitis, acute, 66-69  
diagnosis from perforated peptic ulcer, 33  
from small gut obstruction, 126  
treatment of, 68-69  
chronic, 69-71  
relapsing, 70  
simulating carcinoma, 70-71  
treatment of, 69, 70-71  
in cholecystitis, 50, 54
- Pansinusitis, 664
- Papilloedema, 670-672  
in brain abscess, 660  
in head injuries, 639, 643  
in raised intracranial pressure, 706  
unilateral, causes of, 672
- Para-aminosalicylic acid, in tuberculosis, ulcerative, 117. *See also* PAS.
- Paracentesis abdominis, in constrictive pericarditis, 607
- Parasthesia, acoustic nerve tumours causing, 722
- Paralysis, abductor and adductor of the larynx, 692  
Erb-Duchenne, 812  
facial, 653, 694  
Khumpke's, 812  
laryngeal, unilateral total, 891
- Paraphimosis, 313-314, 420-421

Prostate, calculi of, 405  
 carcinoma of, 401-403  
   diagnosis from tuberculosis, 443  
   hematuria in, 322  
   treatment of, 402-403  
 enlargement of, senile, 393-398  
   investigations, 395  
   physical signs of, 395  
   treatment of, 393-398  
 obstruction of, 399  
 tuberculosis of, 443  
 Prostatectomy, complications of, 400-401  
 epididymitis and, 425-426  
 for carcinoma of prostate, 402  
 "immediate", in acute retention of urine, 399-400  
 Millin's, 396-397, 398  
 perineal, 397-398  
 perurethral, 397-398  
 post-operative management of, 400-401  
 pre-operative management of, 400  
 retropubic, Millin's, 396-397, 398  
 suprapubic, 396  
 transvesical, 396, 398  
   Freyer's, 396  
   Harris's and Wilson Hey's methods, 396  
   two-stage, 399  
 Prostatitis, 403-405  
   non-specific, diagnosis from tuberculosis, 443  
   non-suppurative, subacute, 404-405  
   suppurative, acute, 403  
   chronic, 403-404, 405  
 Prostaglandin, spastic ileus following administration of, 130  
 Proteins, serum, test of liver function, 78  
 Prothrombin, plasma, test of liver function, 77  
 Pruritus ani, 170  
   of scrotum, 433  
 Psammoma, spinal, 788  
 Pseudo-fractures, 1067  
 Pseudo-haematoma syndrome, 646  
 Pseudomyxoma peritonei, 12, 105  
 Pseudo-papilloedema, 672  
 Pseudo-polyposis, ulcerative colitis and, 138, 139  
 Psychosis, after head injuries, 654  
   manic-depressive, surgery of, 763  
 Pulmonary atresia, 614  
   resections, 549-565  
   for tuberculosis, 544  
   post-operative care, 563-566  
   pre-operative care, 561-563  
   stenosis, valvotomy and, 619-622  
 Purpura, 95-96  
 Pyæmia, portal, 80-81  
   appendicectomy complicated by, 10  
   piles and, 158  
 Pyelitis, 331-332  
   diagnosis from appendicitis, 5, 288  
   from cholecystitis, 50  
 Pyelogram, 323-324  
 Pyelography, intravenous, 305, 308  
 Pyelolithotomy, 346  
 Pyelon duplex, 328

Pyelonephritis, 332-333  
   acute, 332-333  
   chronic, 333, 335  
   post-operative, 400  
 Pyeloplasty, 341-342  
 Pyknolepsy, 757  
 Pylephlebitis, in appendicitis, 5  
 Pyloric obstruction, in children, after ferrous sulphate poisoning, 258  
   in peptic ulcer, 38-39  
   stenosis, congenital. *See* Pyloric stenosis, hypertrophic infantile.  
   hypertrophic infantile, 255-258  
   complications of, 258  
   diagnosis of, 256  
   from reflux œsophagitis, 253  
   treatment of, 256-258  
 Pyonephrosis, 333-334  
 Pyrexia of unknown origin, 658  
 Pyuria, abacterial, 367

Q

Queckenstedt's test, 703  
 Queen Elizabeth Hospital for Children, graduated flask for intravenous therapy, 301  
 Quinizarin sweat test, 799, 806-807

R

Rad, definition of, 1321  
 Radiation, biological effects of, 1322  
   protection, 1322-1323  
   reactions, 1326-1327  
   rectal stricture caused by, 168, 169  
 Radioactive iodine, 886, 899-900. *See also* Iodine, radioactive.  
   substances, 1319-1320  
 Radiotherapy, dosage, 1321-1322  
   in surgery, 1323-1326  
   in tumours, 1323-1326  
   physical principles of, 1319  
 Radio-ulnar joint, lower, dislocation of, 1166  
 Radium, 1319, 1320  
   therapy, methods of, 1320-1321  
 Radius, fractures of, 1220-1221  
   of head, 1222-1223  
   of lower end, 1214-1215  
 Radon, 1321  
 Rammstedt's operation, 256  
 Ramsay Hunt's syndrome, 684, 689  
 Ranula, 844  
 Rathke's pouch, tumours of, 728  
 Raynaud phenomenon, 977  
   primary, 977-978  
   secondary, 978, 979  
 Raynaud's disease, operative treatment, 796, 798. *See also* Raynaud phenomenon, primary.  
 Reaction of degeneration, 806, 808  
 von Recklinghausen's disease, 523, 721, 788-789, 823, 824, 869  
   generalised osteitis fibrosa cystica, 923

- Phlebolith, 997, 1018  
 diagnosis from ureteric calculus, 359
- Phlebothrombosis, 996, 999  
 post-operative, 999-1001
- Phlegmasia alba dolens, 998
- Phrenic crush, in pulmonary tuberculosis, 532-533, 811
- Pick's presenile cerebral atrophy, diagnosis from frontal lobe tumours, 707
- Pigment gall-stones, 55
- Piles, 157-161
- Pilonidal sinus, 179-181, 286
- Pineal tumour, 687
- Pitressin, 731
- Pituitary adenoma, 931  
 acidophil, 727-728, 729  
 chromophobe, 710  
 lobe, anterior, adenocarcinoma of, 728  
 tumours, 724-731  
 differential diagnosis of, 728  
 invading skull, 768  
 operations for, 729-730  
 post-operative complications, 730  
 radiotherapy for, 731
- Plantar fasciitis, 1147
- Plasma solutions, in infancy and childhood, 301
- Plasmocytoma, solitary, 1075, 1078
- Plastic grafts, 995
- Plaster casts, arterial injuries from, 994
- Plethysmography, in ischaemia, 971
- Pleurisy, diagnosis from appendicitis, 6
- Plombage, plastic, extra-periosteal, in pulmonary tuberculosis, 541
- Pneumaturia, 371
- Pneumoencephalography, in intracranial tumours, 704
- Pneumectomy, for chronic empyema, 488  
 for lung cysts, 513  
 intrapericardial, technique of, 558  
 left, technique of, 551-558  
 post-operative care, 563, 564  
 right, technique of, 556-557
- Pneumonia, diagnosis from appendicitis, 6, 288  
 from bronchial carcinoma, 499  
 from intestinal obstruction, 126-127  
 from peptic ulcer, perforated, 33  
 post-operative, 455  
 staphylococcal, 471, 516
- Pneumonitis, appendicectomy followed by, 10  
 chronic suppurative, 463
- Pneumothorax, artificial, for pulmonary tuberculosis, empyema after, 548  
 extra-pleural, for pulmonary tuberculosis, technique of, 540-541  
 persistent, 517  
 spontaneous, 516  
 cysts causing, 513-517
- Podagra, 1156
- Poisons, causing optic atrophy, 667  
 papilloedema from, 672
- Polio-myelitis, diagnosis from appendicitis, 6  
 from haematogenous osteomyelitis, 1040-1041  
 lumbar sympathectomy for, 798
- Polio-myelitis, spine in, 1083
- Polya operation, 42
- Polyarteritis nodosa, presenting as aneurysm, 985
- Polycythaemia rubra vera, papilloedema in, 672
- Polydipsia, post-operative, 731
- Polymyxins, 1316
- Polypsis, adenomatous, diffuse, of stomach, 20  
 multiple (familial), 144-145, 155  
 synovial, 1079
- Polytan sponge, 542
- Polythene bags, for plombage, 542  
 balls, for plombage, 542
- Polyuria, 320  
 post-operative, 731
- Pool's topectomy, 763
- Popliteal palsy, external, 1265
- Porencephaly, traumatic, 648
- Porphyria, acute, diagnosis from intestinal obstruction, 126
- Portal-caval shunt, 91-92
- Portal hypertension, 87  
 -systemic venous shunt, 90-92
- Port wine stain, 1019
- Post-concussion syndrome, 635
- Posterior fossa, angioreticuloma of, 721  
 astrocytoma of, 720-721  
 ependymoma of, 721  
 haemangioblastoma of, 721  
 investigations, 719-720  
 lesions of, 692  
 medulloblastoma of, 721  
 meningioma of, 722  
 tumours of, 707, 718-720
- Post-gastrectomy syndrome, 45-46
- Proctitis, 314
- Posture, defects of, 1082-1083  
 in foot, 1143-1143
- Post-valvotomy syndrome, 604
- Potassium deficiency, 1305-1306  
 in fluid therapy, 297-299  
 iodide, 901, 902
- Pott's coarctation clamp, 633
- Pott's disease, 786-787, 1090-1094
- Pott's operation for Fallot's tetralogy, 613  
 post-operative care, 623  
 technique of, 618-619
- Pott's puffy tumour, 664, 769
- Pouch, pharyngeal, 591, 592
- Prednisone, in pancreatitis, 69
- Prefrontal leucotomy, 783
- Preiser's disease, 1107
- Prepatellar bursitis, 1133-1134
- Prepuce, in new born, 312  
 inflammation of, 314
- Priapism, 421
- Proctitis, 169-170
- Procto-colectomy, total, for familial polyposis, 144
- Proctotomy, for stricture, 189
- Prolactin, 724, 725
- Prolapse, rectal, 166-168
- Prostate, adenomatous hypertrophy, simple, 394  
 benign hyperplasia of, 393

- Scrotum, carcinoma of, 433  
   dermatitis of, 433  
   disease of, 433-434  
   elephantiasis of, 434  
   epithelioma of, 433  
   idiopathic gangrene of, 433  
   sebaceous cysts of, 433  
 Scurvy, 1067  
 Sebaceous cyst, 829  
   horn, 828  
 Sedileau's bands, 537  
 Seddon's classification of peripheral nerve injuries, 803  
 Semb's space, 537  
 Sentinel pile, 162  
 Septal defects, 624  
 Sequestrectomy in osteomyelitis, 1012  
 Serratus anterior, nerve to, lesions of, 814-815  
 Serum acid phosphatase estimation, in carcinoma of prostate, 401  
   sickness, 1193  
 Sesamoids, fractures of, 1153  
   lesions of, 1153  
 Sever's disease, 1146  
 Shédé's operation, 488  
 Shingles, 678-679  
 Shock, irreversible, 1303-1304  
   traumatic, 1302-1303  
 Shoulder, dislocation of, 1160-1163  
   posterior, 1163  
   recurrent, 1163  
   frozen, 1096  
   joint, arthrodesis of, 1099  
   tuberculosis of, 1098-1099  
   pain in, 1096  
 Sialadenitis, 426  
 Sialiectasis, 839  
   diagnosis from parotitis, 837-838  
 Sibson's fascia, 536-537  
 Sigmoidoscopy, in diagnosis of intestinal amebiasis, 183-184  
   in ulcerative colitis, 139  
 Signe de rideau, 691  
 Sinusitis, ethmoid and sphenoid, papilloedema and, 671, 672  
   nasal, frontal lobe abscess and, 659  
 Sinus(es), branchial, 864, 866-867  
   cervical, lateral, medial, 865, 866-867  
   dermal, congenital, 286  
   in sacrococcygeal region, 286  
   pilonidal, 179-181, 286  
   sphenoid, mucocele and empyema of, 668-669  
   thyroglossal, 865  
   venous, injuries of, 651-652  
 Sjogren's syndrome, 837  
 Sjoqvist's operation, 678  
 Skin grafts for burns, 1296-1297  
   for fractures, 1191-1192  
   malignant disease of, 831  
   melanotic lesions of, 833-835  
   operative closure of, 194  
 "Skip" areas, in Crohn's disease, 138  
   in small gut, 113  
 Skull, carcinoma of, secondary, 768, 769  
 Skull, chondroma and chondrosarcoma of, 767  
   chordoma of, 767  
   eosinophilic granuloma of, 765-766, 769  
   epidermoid tumour of, 766-767, 769  
   fractures of, 659  
     cranial nerve injury in, 683  
     extradural hematoma and, 639, 640  
     types of, 647-648  
   frontal impression in new-born, diagnosis from fractures, 647, 648  
   giant-cell tumour of, 765, 769  
   hemangioma of, 765, 769  
   in myelomatosis, 769  
   opening of, operative techniques, 732-736  
     incisions for, 735-736  
   osteogenic sarcoma of, 767  
   osteoma of, 764-765, 769  
   osteomyelitis of, 655, 658, 660, 664  
   periosteal fibrosarcoma of, 767  
   tumours of, 764-769  
     diagnosis of, 769  
     secondary, 768-769  
 Sling, supine, in spinal fracture, 1233  
 Smith-Petersen arthroplasty, 1121-1122  
   pin, 1198, 1242  
 Smith's fracture, 1214  
 Smoking, bronchial carcinoma and, 497  
 Sodium tetra-iodo-phenol-phthalein, in diagnosis of gall bladder disease, 47, 48  
 Sounding of urethra, 408-409  
 Spasm, arterial, 992, 993  
 Speech training, in cleft lip and palate, 249  
 Spermatocord, hydrocele of, 428-429  
 Spermatocelo, 429  
 Spider naevus, 1019  
 Spiller-Frazier operation, for tic douloureux, 676-678  
 Spina bifida, 791-792  
   management of, 792-793  
   occulta, 791, 1036, 1085  
 Spinal column, radiography, 772  
   cord, and nerve roots, 770-793  
   bladder in lesions of, 776-778  
   compression, 781  
   syndrome, 770-771  
   ependymomas in, 791  
   extradural lesions, 780, 783  
   injuries, 789  
   lesions, metastatic tumours and, 788  
   myelography for space-occupying lesions, 772  
   syndromes of various levels, 773-776  
   tumours, 791, 818  
 Spine, 1080-1095  
   cervical, fractures of, 1230-1231  
   clinical examination of, 1080-1082  
   congenital defects of, 1084-1086  
   fractures of, 1229-1233  
     first aid and transport of, 1229  
   injuries of, 789  
   lumbar, fractures of, 1231-1233  
     complications of, 1233  
   meningiomas of, 788  
   muscle tone defects, 1083-1084  
   neoplasm of, diagnosis from Pott's disease, 1092-1093

- Rectosigmoidectomy, in Hirschsprung's disease, 273-274  
     in rectal prolapse, 168  
 Rectum, amœboma of, 183  
     and anus, malformations of, clinical appearances, 279-280  
     differential diagnosis of, 280  
     management of, 281-283  
     restorative operation, 283  
     types of, 276-277  
     anatomy of, 156-157  
     carcinoma of, 145-150, 170-176  
         clinical features of, 172-173  
         complications of operation, 174-176  
         differential diagnosis of, 173  
         mortality of operation, 175  
         pathology of, 170-172  
         radiotherapy in, 175  
         sacral plexus infiltration of, 786  
         treatment of, 173-174  
     duplication of, 285  
     excision of, for stricture, 160  
     imperforate, 276, 312  
     prolapse of, complete, 168  
         in childhood, diagnosis from intussusception, 283  
         partial, 166-168  
     resection of, operations for, 176-179  
         anterior, 178-179  
         combined excision, 177-178  
         perineal excision, 176-177  
         restorative, 178  
     stricture of, 169-169  
     tumours of, 143-145  
 Rectus abdominis musculo, divarication of, 210  
 Reflex, reno-renal, 310  
 Rehabilitation after fractures, 1186-1187  
 Reiter's syndrome, 411  
 Renal failure, acute, 1307  
     function tests, 326-327  
     pelvis, tumours of, 347, 351-352  
 Reticulosarcoma, 1028  
 Reticuloses, affecting mediastinal glands, 527  
 Retina, angioma of, 721  
     central artery of, occlusion of, 667  
 Retroperitoneal tumours, 105-106  
 Rhagades, 827-828  
 Rheumatic fever, diagnosis from hæmatogenous osteomyelitis, 1040  
 Rheumatism, senile, 1061  
 Rh factor, 1310-1311, 1312  
 Rhinophyma, 829  
 Rhinorrhœa, cerebrospinal fluid, in head injuries, 650-651  
 Rib, cervical, 813-814  
 Ribs, fractures of, 1227-1228  
 Riches' apparatus, for suprapubic cystostomy, 392  
 Richter's hernia, 198-199, 224  
     strangulation of bowel in, 122  
 Rickets, 1066-1067, 1136  
     cœliac, 1067  
     renal, 1067  
 Rickety rosary, 1067  
 Riedel's lobe, diagnosis from tumour, 85  
 Riedel's thyroiditis, 894-895, 902  
 Rinné's test, 686  
 Roberts' operation, 488  
 Rob's surgical procedure for occlusion of internal carotid artery, 754  
 Rodent ulcers of face, 831  
     diagnosis of, 832-833  
 Röntgen, definition of, 1321  
 Rovsing's operation for polycystic disease of kidney, 353  
 Rubella, during pregnancy, 1036-1037  
 Rutherford Morison's incision, 8
- S
- Sacral segments and cauda equina syndrome, 775-776  
 Sacralisation, 1086  
 Sacrococcygeal teratoma, 284  
     diagnosis from meningocele, 285  
     tumours, 284-285  
 Sacro-iliac joint, subluxation of, sciatica in, 786  
     tuberculosis of, sciatica in, 786  
 Sacrum, fractures of, 1238  
 St. Mark's Hospital, lotion, for pruritus ani, 170  
 Salivary glands, infections of, 835-840  
     submandibular, 838  
     tumours, 843  
         ectopic, of palate, 856  
         of mouth, 845  
         *See also* Parotid tumours.  
 Salmon patch, 1019  
 Scleritis, diagnosis from appendicitis, 5  
 Salt and water balance, in head injuries, 637-638  
 Saphenous varix, simulating hernia, 216  
 Sarcoidosis, facial nerve lesions in, 684. *See also* Boeck's sarcoid.  
 Sarcoma, osteogenic, 1041, 1071-1073, 1074  
     *See also* under names of organs and regions.  
 Scalenus anterior syndrome, 814  
     medius syndrome, 814  
 Scalp, missile wounds of, 648  
 Scapula, fractures of, 1227  
 Scars, keloid, treatment of, 900, 1323  
 Schummelbusch's disease, 946  
 Schistosomiasis, cystitis in, 366  
 Schizophrenia, surgery of, 763  
 Schlatter's disease, 1132, 1133  
 Schmorl's nodes, 1087  
 Schönlein-Henoch syndrome, 95  
 Schwannoma, 721, 788, 823. *See also* Neurofibroma.  
 Sciatica, 782  
     causes of, 785-786  
 Sclerosis, disseminated, 667, 690  
     lateral, amyotrophic, 819  
     optic "neuritis" in, 872  
 Scoliosis, idiopathic, 1083-1084  
     diagnosis from Pott's disease, 1092  
 Scotoma, 747  
 Scoville's frontal lobe operation, 763  
 Scrotum, 424  
     burns of, 1297

- Scrotum, carcinoma of, 433  
   dermatitis of, 433  
   diseases of, 433-434  
   elephantiasis of, 434  
   epithelioma of, 433  
   idiopathic gangrene of, 433  
   sebaceous cysts of, 433  
 Scurvy, 1067  
 Sebaceous cyst, 828  
   horn, 828  
 Seheleau's bands, 537  
 Seddon's classification of peripheral nerve injuries, 803  
 Semli's space, 537  
 Sentinel pile, 162  
 Septal defects, 624  
 Sequestrectomy in osteomyelitis, 1042  
 Serratus anterior, nerve to, lesions of, 814-815  
 Serum acid phosphatase estimation, in carcinoma of prostate, 401  
   rickets, 1103  
 Sesamoids, fractures of, 1163  
   lesions of, 1163  
 Sever's disease, 1146  
 Shédé's operation, 488  
 Shingles, 678-679  
 Shock, irreversible, 1303-1304  
   traumatic, 1302-1303  
 Shoulder, dislocation of, 1160-1163  
   posterior, 1163  
   recurrent, 1163  
   frozen, 1096  
   joint, arthrodesis of, 1099  
   tuberculosis of, 1098-1099  
   pain in, 1096  
 Sialadenitis, 426  
 Sialectasia, 839  
   diagnosis from parotitis, 837-838  
 Sibson's fascia, 536-537  
 Sigmoidoscopy, in diagnosis of intestinal amebiasis, 183-184  
   in ulcerative colitis, 139  
 Signe de rideau, 691  
 Sinusitis, ethmoid and sphenoid, papilloedema and, 671, 672  
   nasal, frontal lobe abscess and, 659  
 Sinus(es), branchial, 864, 866-867  
   cervical, lateral, medial, 865, 866-867  
   dermal, congenital, 286  
   in sacrococcygeal region, 286  
   pilonidal, 179-181, 286  
   sphenoid, mucocele and empyema of, 668-669  
   thyroglossal, 865  
   venous, injuries of, 651-652  
 Sjogren's syndrome, 837  
 Sjoqvist's operation, 678  
 Skin grafts for burns, 1296-1297  
   for fractures, 1191-1192  
   malignant disease of, 831  
   melanotic lesions of, 833-835  
   operative closure of, 194  
 "Skip" areas, in Crohn's disease, 138  
   in small gut, 113  
 Skull, carcinoma of, secondary, 768, 769  
   chondroma and chondrosarcoma of, 767  
   chordoma of, 767  
   eosinophilic granuloma of, 765-766, 769  
   epidermoid tumour of, 766-767, 769  
   fractures of, 659  
     cranial nerve injury in, 683  
     extradural hematoma and, 639, 640  
     types of, 647-648  
   frontal impression in new-born, diagnosis from fractures, 647, 648  
   giant-cell tumour of, 765, 769  
   hemangioma of, 765, 769  
   in myelomatosis, 769  
   opening of, operative techniques, 732-736  
     incisions for, 735-736  
   osteogenic sarcoma of, 767  
   osteoma of, 764-765, 769  
   osteomyelitis of, 655, 658, 660, 664  
   periosteal fibrosarcoma of, 767  
   tumours of, 764-769  
     diagnosis of, 769  
     secondary, 768-769  
 Sling, supine, in spinal fracture, 1233  
 Smith-Petersen arthroplasty, 1121-1122  
   pin, 1198, 1242  
 Smith's fracture, 1214  
 Smoking, bronchial carcinoma and, 497  
 Sodium tetra-iodo-phenol-phthalein, in diagnosis of gall bladder disease, 47, 48  
 Sounding of urethra, 408-409  
 Spasm, arterial, 902, 903  
 Speech training, in cleft lip and palate, 249  
 Spermatocoele, 429  
 Spider naevus, 1010  
 Spiller-Frazier operation, for tic douloureux, 676-678  
 Spina bifida, 791-792  
   management of, 792-793  
   occulta, 791, 1036, 1085  
 Spinal column, radiography, 772  
   cord, and nerve roots, 770-793  
   bladder in lesions of, 776-778  
   compression, 781  
   syndrome, 770-771  
   ependymomas in, 791  
   extradural lesions, 780, 783  
   injuries, 780  
   lesions, metastatic tumours and, 788  
   myelography for space-occupying lesions, 772  
   syndromes of various levels, 773-776  
   tumours, 791, 818  
 Spine, 1080-1095  
   cervical, fractures of, 1230-1231  
   clinical examination of, 1080-1082  
   congenital defects of, 1084-1086  
   fractures of, 1229-1233  
     first aid and transport of, 1229  
   injuries of, 780  
   lumbar, fractures of, 1231-1233  
     complications of, 1233  
   meningiomas of, 788  
   muscle tone defects, 1083-1084  
   neoplasm of, diagnosis from Pott's disease, 1092-1093

- Rectosigmoidectomy*, in *Hirschsprung's* disease, 273-274  
 in rectal prolapse, 168  
*Rectum*, amœboma of, 183  
 and anus, malformations of, clinical appearances, 279-280  
 differential diagnosis of, 280  
 management of, 281-283  
 restorative operation, 283  
 types of, 276-277  
 anatomy of, 156-157  
 carcinoma of, 145-150, 170-176  
   clinical features of, 172-173  
   complications of operation, 174-176  
   differential diagnosis of, 173  
   mortality of operation, 175  
   pathology of, 170-172  
   radiotherapy in, 175  
   sacral plexus infiltration of, 786  
   treatment of, 173-174  
 duplication of, 285  
 excision of, for stricture, 169  
 imperforate, 276, 312  
 prolapse of, complete, 168  
   in childhood, diagnosis from intussusception, 293  
   partial, 166-168  
 resection of, operations for, 176-179  
   anterior, 178-179  
   combined excision, 177-178  
   perineal excision, 176-177  
   restorative, 178  
 stricture of, 188-189  
 tumours of, 143-145  
*Rectus abdominis* muscle, divarication of, 219  
 Reflex, reno-renal, 319  
 Rehabilitation after fractures, 1186-1187  
 Reiter's syndrome, 411  
*Renal* failure, acute, 1207  
   function tests, 326-327  
   pelvis, tumours of, 347, 351-352  
*Reticulosarcoma*, 1028  
*Reticuloses*, affecting mediastinal glands, 527  
*Retina*, angioma of, 721  
   central artery of, occlusion of, 667  
*Retroperitoneal* tumours, 105-106  
*Rhagades*, 827-828  
*Rheumatic* fever, diagnosis from hæmatogenous osteomyelitis, 1040  
*Rheumatism*, senile, 1061  
*Rh* factor, 1310-1311, 1312  
*Rhinophyma*, 829  
*Rhinorrhœa*, cerebrospinal fluid, in head injuries, 650-651  
*Rib*, cervical, 813-814  
*Ribs*, fractures of, 1227-1228  
*Riches'* apparatus, for suprapubic cystostomy, 392  
*Richter's* hernia, 198-199, 224  
   strangulation of bowel in, 122  
*Rickets*, 1066-1067, 1136  
   cœliac, 1067  
   renal, 1067  
*Rickety* rosary, 1067  
*Riedel's* lobe, diagnosis from tumour, 83  
*Riedel's* thyroiditis, 894-895, 902  
*Rinne's* test, 686  
*Roberts'* operation, 488  
*Rob's* surgical procedure for occlusion of internal carotid artery, 754  
*Rodent* ulcers of face, 831  
   diagnosis of, 832-833  
*Rœntgen*, definition of, 1321  
*Rovsing's* operation for polycystic disease of kidney, 353  
*Rubella*, during pregnancy, 1036-1037  
*Rutherford Morison's* incision, 8
- S
- Sacral segments and cauda equina syndrome, 775-776  
*Sacralisation*, 1086  
*Sacrococcygeal* teratoma, 284  
   diagnosis from meningocele, 285  
   tumours, 284-285  
*Sacro-iliac* joint, subluxation of, sciatica in, 786  
   tuberculosis of, sciatica in, 786  
*Sacrum*, fractures of, 1238  
*St. Mark's* Hospital, lotion, for pruritus ani, 170  
*Salivary* glands, infections of, 835-840  
   submandibular, 838  
   tumours, 843  
   ectopic, of palate, 850  
   of mouth, 845  
   *See also* Parotid tumours.  
*Salmon* patch, 1019  
*Salpingitis*, diagnosis from appendicitis, 5  
 Salt and water balance, in head injuries, 637-638  
*Saphenous* varix, simulating hernia, 216  
*Sarcoidosis*, facial nerve lesions in, 684. *See also* Boeck's sarcoid.  
*Sarcoma*, osteogenic, 1041, 1071-1073, 1074  
   *See also* under names of organs and regions.  
*Scalenus* anterior syndrome, 814  
   medius syndrome, 814  
*Scalp*, missile wounds of, 648  
*Scapula*, fractures of, 1227  
*Scars*, keloid, treatment of, 909, 1323  
*Schimmelbusch's* disease, 946  
*Schistosomiasis*, cystitis in, 366  
*Schizophrenia*, surgery of, 763  
*Schlatter's* disease, 1132, 1133  
*Schmorl's* nodes, 1087  
*Schönlein-Henoch* syndrome, 95  
*Schwannoma*, 721, 788, 823. *See also* Neurofibroma.  
*Sciatica*, 782  
   causes of, 785-786  
*Sclerosis*, disseminated, 667, 690  
   lateral, amyotrophic, 819  
   optic "neuritis" in, 673  
*Scoliosis*, idiopathic, 1083-1084  
   diagnosis from Pott's disease, 1092  
*Scotoma*, 747  
*Scoville's* frontal lobe operation, 763  
*Scrotum*, 424  
   burns of, 1297

- Synovitis, syphilitic, 1037  
 transient, of hip, 1115  
 Syphilis, congenital, 1037  
   orchitis in, 233  
   of brain, diagnosis from frontal lobe tumours, 707  
   of face, 827  
   of hip, 1119  
   of jaw, 855  
   of knee, 1135-1136  
   of palate, 855  
   of tongue, 850, 852  
   tenosynovitis in, 1105  
   tertiary, bones in, 1037  
 Syringobulbia, 690  
 Siringomyelia, 818-819  
   intramedullary tumours and, 776  
 Siringomyelocoele, 792

## T

- Talpa dorsalis, 666, 667  
   gastric crisis of, diagnosis from perforated peptic ulcer, 33  
 Tale granuloma, 106  
   diagnosis from Crohn's disease, 115  
 Talipes calcaneo-valgus, 1139, 1141  
   calcanus, 1139  
   congenital, 1138  
   equinovarus, 1139-1140  
   equinus, 1139  
   valgus, 1139  
   varus, 1139  
 Talus, dislocations of, 1171  
   neck of, fractures of, 1279-1280  
 Tarsorrhaphy, 679, 892  
 Tarsus, fractures of, 1279-1282  
 TSH, 724, 725  
 Taxis, definition of, 224  
 Tay Sachs' disease, 667. *See* Idiocy, amaurotic family.  
 Teeth, cysts of, 857-858  
   Hutchinson's, 1037  
   Moon's 1037  
 Telangiectasis, familial multiple, 1021-1022  
 Temporal cortex, 711  
   lobe epilepsy, 756, 760  
   herniation, 711  
   syndrome, 711-713  
   tumours, diagnosis from frontal tumours, 707  
     differential, 712  
     investigations, 712-713  
     pathology of, 713-715  
 Tendinitis, bicipital, 1098  
   calcifying, 1096  
   degenerative, 1096, 1097-1098  
 Tendo achillis, rupture of, 1147  
 Tendon sheaths, giant-cell tumours of, 1103-1104  
   tuberculosis of, 1051  
 Tendons, extensor, repair of, 1288-1289  
   flexor, repair of, 1288  
   injuries to, 1200  
   of wrist and hand, rupture of, 1105-1106  
 Tennis elbow, 1062, 1101  
 Tenosynovitis, around wrist, 1104  
   in hand, 1104-1105  
   tuberculous, 1050-1051  
 Tenotomy of tendo achillis, 974  
 Terramycin, 1316  
   in acute cholecystitis, 51  
   in lymphogranuloma inguinale, 1025  
   in peritonitis, 9  
   in urethritis, non-gonococcal, 411  
 Testis, 423-424  
   appendix, torsion of, 233  
   biopsy of, 434  
   chorion-epithelioma of, 431  
   ectopic, 230-231  
   fibrosis of, 426  
   hematocoele of, 430  
     acute, diagnosis from orchitis, 428  
     diagnosis from inguinal hernia, 208  
   imperfect descent of, 229-233  
     tumours and, 431-432  
   infarction of, 233  
   inflammation of, 424-426  
   maldescended, diagnosis from inguinal hernia, 208  
   operations on, 435-436  
   retractile, 230  
   seminoma of, 431  
   syphilis of, 432  
     hydrocele and, 428  
   teratoma of, 431  
   torsion of, 232-233  
     diagnosis from inguinal hernia, 208  
     from orchitis, 426  
   tuberculosis of, 443  
   tumours of, 431-433  
     hydrocele and, 428  
     treatment of, 433  
   undescended, 231-232  
     malignancy in, 232  
 Tetanus, 1193-1194  
 Tetany, following thyroid operation, 909  
   in pancreatitis, 67, 68, 69  
   parathyroid. *See* Hypoparathyroidism.  
 Tetracycline, 1316-1317  
 Tetracyclines, 1316-1317  
 Tetracycyn, 1316  
 Thiersch's operation, for rectal prolapse, 167-168  
   skin graft, for compound fractures, 1191  
 Thiouracil, 901  
 Thomas's splint, 1184  
 Thoracic disc protrusion, 781  
   treatment of, 784  
   syndrome, lower, middle and upper, 775  
 Thoracoplasty, failed, resection after, 540, 544  
   technique of, 533-540  
 Thoracoscopy, and section of adhesions, technique of, 530-532  
 Thoracotomy, in bronchial carcinoma, 504, 507  
   positions and technique for, 549-551  
 Thorium X, for port wine stain, 1019  
 Threadworms, 170  
 Thromboangitis obliterans, 108, 966-967  
   lumbar sympathectomy for, 798



- Spine, osteo-arthritis of, 1094-1095  
 posture of, 1082-1083  
 thoracic, fractures of, 1231  
 tuberculous disease of, 786-787, 1090-1094  
   diagnosis of, 1091-1092  
     differential, 1092-1093  
     treatment of, 1093-1094  
   *See also* Pott's disease of spine.
- Spinohalamic tractotomy, 793
- Spleen, 94-98  
   accessory, 94  
   cysts of, 98  
   enlarged, 97-98  
   rupture of, 94-95
- Splenectomy, complications of, 98  
   during other operations, 95  
   in blood disease, 95-98  
   indications, 94  
   technique of, 98
- Splenomegaly, 88  
   congestive, 92, 97-98
- Splenorenal shunt, 92
- Spondylitis ankylopoetica, 1061, 1087-1089, 1324  
   diagnosis from Pott's disease, 1092  
   tuberculous, 786-787
- Spondylolisthesis, 1085-1086  
   diagnosis from embryological mal-development, 1036  
   sacral and, 783
- Spondylolysis, 1085, 1094-1095
- Spongioblastoma multiforme, 708. *See also* Glioblastoma multiforme.
- Sprain, 1158  
   chronic, 1160
- Spurling's test, 781
- Steatorrhoea, 70, 1067
- Steinmann's pin and stirrup, 1188
- Sterility, male, 434-435  
   epididymitis as cause of, 426
- Sterno-clavicular joint, arthritis of, 1096
- Sternomastoid tumour, 869-870
- Sternum, fractures of, 1228
- Stewart (Hamilton), operation for hydro-nephrosis, 342
- Stillbaestrol, in carcinoma of prostate, 396
- Still's disease, 1061, 1092
- Stöffel's operation, 822-823
- Stomach, angioma of, 20  
   carcinoma of, 13-19  
     aetiology of, 13  
     clinical features of, 15  
     diagnosis of, differential, 18  
       from peptic ulcer, 27  
     investigations, 16-18  
     microscopic appearance of, 14  
     pathology of, 13-14  
     perforated, 16  
     peritoneal tumour secondary to, 105  
     physical examination in, 15  
     prognosis of, 16, 19  
     treatment of, 18-19  
     types of, 13  
   fibroma of, 20  
   hour-glass, complicating gastric ulcer, 39  
   leiomyoma of, 20
- Stomach, lipoma of, 20  
   lymphosarcoma of, 20  
   neurilemmoma of, 20  
   operations, technique of, 39-45  
   papilloma of, 20  
   sarcoma of, 20  
   tumours of, 13-20  
     benign, 20  
   ulcer of, differentiation between benign and malignant, 27  
     perforated, technique of closure, 39-40  
     *See also* Peptic ulcer.
- Stomatitis, 844-845
- Strawberry mark, 1019. *See also* Ham-angioma, strawberry.
- Streptomycin, 875, 1316  
   deafness caused by, 687  
   in appendicitis, 11  
   in bowel sterilisation, 150-151  
   in cold abscess, 1046, 1093  
   in constrictive pericarditis, 607  
   in peritonitis, 101, 102-103  
   in soft chancre, 419  
   in tuberculoma, 723  
   in tuberculosis, 1046, 1049  
     meningitic, 659  
     pulmonary, 543, 544, 565  
     ulcerative, 117  
     urinary, 442  
   labyrinthine symptoms caused by, 689
- Stress incontinence, 321  
   syndrome, 1062
- Subacromial bursitis, 1096-1097
- Subarachnoid-ureterostomy, 744
- Subhyoid bursa, 868
- Sudeck's atrophy, 1204
- Sulphonamides, in appendicitis, 9  
   in carcinoma of colon, 150  
   in perforated peptic ulcer, 35  
   in peritonitis, 101  
   in soft chancre, 419  
   in spinal injuries, 780  
   in urinary tract infections, 1314  
   powder, 1314
- Suprarenal glands, 930
- Sweating, excessive, 798, 799  
   post operative test, 799
- Sweat-test, 806-807
- Sylvian fissure, meningiomas of, 715
- Sympathicoblastoma, undifferentiated, 800.  
   *See also* Neuroblastoma.
- Sympathectomy, for causalgia, 806  
   for chronic occlusive disease, 973-974  
   for post-thrombotic oedema, 1032  
   for Sudeck's atrophy, 1204  
   lumbar, 1014  
   post-operative course and tests, 799  
   thoraco-lumbar, 797-798
- Syndesmosis, tibiofibular, diastasis of, 1273-1274
- Synkinesia, 684
- Synovectomy, in tuberculosis, 1030
- Synovial cysts, 1079  
   polypoid, 1079
- Synovioma, 1078-1079  
   benign, of fingers, 1103-1104

- Synovitis, syphilitic, 1037  
transient, of hip, 1115  
Syphilis, congenital, 1037  
  orchitis in, 233  
  of brain, diagnosis from frontal lobe tumours, 707  
  of face, 827  
  of hip, 1119  
  of jaw, 855  
  of knee, 1135-1136  
  of palate, 856  
  of tongue, 850, 852  
  tenosynovitis in, 1103  
  tertiary, bones in, 1037  
Syringobulbia, 690  
Syringomyelia, 818-819  
  intramedullary tumours and, 776  
Syringomyelocoele, 792

## T

- Tabes dorsalis, 666, 667  
  gastric crisis of, diagnosis from perforated peptic ulcer, 33  
Tale granuloma, 106  
  diagnosis from Crohn's disease, 115  
Talipes calcaneo-valgus, 1139, 1141  
  calcaneus, 1139  
  congenital, 1138  
  equinovarus, 1139-1140  
  equinus, 1139  
  valgus, 1139  
  varus, 1139  
Talus, dislocations of, 1171  
  neck of, fractures of, 1279-1280  
Tarsorrhaphy, 670, 802  
Tarsus, fractures of, 1279-1282  
TBII, 724, 725  
Taxis, definition of, 224  
Tay Sachs's disease, 667. *See* Idiocy, amaurotic family.  
Teeth, cysts of, 857-858  
  Hutchinson's, 1037  
  Moon's 1037  
Telangiectasia, familial multiple, 1021-1022  
Temporal cortex, 711  
  lobe epilepsy, 756, 760  
  herniation, 711  
  syndrome, 711-713  
  tumours, diagnosis from frontal tumours, 707  
  differential, 712  
  investigations, 712-713  
  pathology of, 713-715  
Tendinitis, bicipital, 1098  
  calcifying, 1096  
  degenerative, 1096, 1097-1098  
Tendo achillis, rupture of, 1147  
Tendon sheaths, giant-cell tumours of, 1103-1104  
  tuberculosis of, 1051  
Tendons, extensor, repair of, 1288-1289  
  flexor, repair of, 1288  
  injuries to, 1200  
  of wrist and hand, rupture of, 1105-1106  
Tennis elbow, 1062, 1101  
Tenosynovitis, around wrist, 1101  
  in hand, 1101-1105  
  tuberculous, 1050-1051  
Tenotomy of tendo achillis, 974  
Terramycin, 1316  
  in acute cholecystitis, 51  
  in lymphogranuloma inguinale, 1025  
  in peritonitis, 9  
  in urethritis, non-gonococcal, 411  
Testis, 423-424  
  appendix, torsion of, 233  
  biopsy of, 431  
  chorion-epithelioma of, 431  
  ectopic, 230-231  
  fibrosis of, 426  
  hematocoele of, 430  
    acute, diagnosis from orchitis, 426  
    diagnosis from inguinal hernia, 208  
  imperfect descent of, 229-233  
    tumours and, 431-432  
  infarction of, 233  
  inflammation of, 424-426  
  mal descended, diagnosis from inguinal hernia, 208  
  operations on, 435-436  
  retractile, 230  
  seminoma of, 431  
  syphilis of, 432  
    hydrocele and, 428  
  teratoma of, 431  
  torsion of, 232-233  
    diagnosis from inguinal hernia, 208  
    from orchitis, 426  
  tuberculosis of, 443  
  tumours of, 431-433  
    hydrocele and, 428  
    treatment of, 433  
  undescended, 231-232  
    malignancy in, 232  
Tetanus, 1193-1194  
Tetany, following thyroid operation, 909  
  in pancreatitis, 67, 68, 69  
  parathyroid. *See* Hypoparathyroidism.  
Tetracycline, 1316-1317  
Tetracyclines, 1316-1317  
Tetracyan, 1316  
Thiersch's operation, for rectal prolapse, 167-168  
  skin graft, for compound fractures, 1191  
Thiouracil, 901  
Thomas's splint, 1184  
Thoracic disc protrusion, 781  
  treatment of, 784  
  syndrome, lower, middle and upper, 775  
Thoracoplasty, failed, resection after, 540, 544  
  technique of, 533-540  
Thoracoscopy, and section of adhesions, technique of, 530-532  
Thoracotomy, in bronchial carcinoma, 504, 507  
  positions and technique for, 549-551  
Thorium X, for port wine stain, 1019  
Threadworms, 179  
Thromboangitis obliterans, 108, 966-967  
  lumbar sympathectomy for, 798

- Spine, osteo-arthritis of, 1094-1095  
 posture of, 1082-1083  
 thoracic, fractures of, 1231  
 tuberculous disease of, 786-787, 1090-1094  
   diagnosis of, 1091-1092  
   differential, 1092-1093  
   treatment of, 1093-1094  
   *See also* Pott's disease of spine.
- Spinthalamic tractotomy, 793
- Spleen, 94-98  
   accessory, 94  
   cysts of, 98  
   enlarged, 97-98  
   rupture of, 94-95
- Splenectomy, complications of, 98  
   during other operations, 95  
   in blood disease, 95-98  
   indications, 94  
   technique of, 98
- Splenomegaly, 88  
   congestive, 92, 97-98
- Splenorenal shunt, 92
- Spondylitis ankylopoetica, 1061, 1087-1089, 1324  
   diagnosis from Pott's disease, 1092  
   tuberculous, 786-787
- Spondylolisthesis, 1085-1086  
   diagnosis from embryological mal-development, 1036  
   sciatica and, 783
- Spondylolysis, 1085, 1094-1095
- Spongioblastoma multiforme, 708. *See also* Glioblastoma multiforme.
- Sprain, 1188  
   chronic, 1160
- Spurling's test, 781
- Steatorrhoea, 70, 1067
- Steinmann's pin and stirrup, 1186
- Sterility, male, 434-435  
   epididymitis as cause of, 426
- Sterno-clavicular joint, arthritis of, 1096
- Sternomastoid tumour, 869-870
- Sternum, fractures of, 1228
- Stewart (Hamilton), operation for hydro-nephrosis, 342
- Stillbaestrol, in carcinoma of prostate, 396
- Still's disease, 1081, 1092
- Stöffel's operation, 822-823
- Stomach, angioma of, 20  
   carcinoma of, 13-19  
     etiology of, 13  
     clinical features of, 15  
     diagnosis of, differential, 18  
       from peptic ulcer, 27  
     investigations, 16-18  
     microscopic appearance of, 14  
     pathology of, 13-14  
     perforated, 16  
     peritoneal tumour secondary to, 105  
     physical examination in, 15  
     prognosis of, 16, 19  
     treatment of, 18-19  
     types of, 13  
   fibroma of, 20  
   hour-glass, complicating gastric ulcer, 39  
   leiomyoma of, 20
- Stomach, lipoma of, 20  
   lymphosarcoma of, 20  
   neurilemmoma of, 20  
   operations, technique of, 39-45  
   papilloma of, 20  
   sarcoma of, 20  
   tumours of, 13-20  
     benign, 20  
   ulcer of, differentiation between benign and malignant, 27  
     perforated, technique of closure, 39-40  
     *See also* Peptic ulcer.
- Stomatitis, 844-845
- Strawberry mark, 1010. *See also* Hæm-angioma, strawberry.
- Streptomycin, 875, 1316  
   deafness caused by, 687  
   in appendicitis, 9  
   in bowel sterilisation, 150-151  
   in cold abscess, 1046, 1093  
   in constrictive pericarditis, 607  
   in peritonitis, 101, 102-103  
   in soft chancre, 419  
   in tuberculoma, 723  
   in tuberculosis, 1048, 1049  
     meningitic, 659  
     pulmonary, 543, 544, 565  
     ulcerative, 117  
     urinary, 442  
   labyrinthine symptoms caused by, 689
- Stress incontinence, 321  
   syndrome, 1062
- Subacromial bursitis, 1096-1097
- Subarachnoid-ureterostomy, 744
- Subhyoid bursa, 868
- Sudeck's atrophy, 1204
- Sulphonamides, in appendicitis, 9  
   in carcinoma of colon, 150  
   in perforated peptic ulcer, 35  
   in peritonitis, 101  
   in soft chancre, 410  
   in spinal injuries, 780  
   in urinary tract infections, 1314  
   powder, 1314
- Suprarenal glands, 930
- Sweating, excessive, 798, 799  
   post operative test, 799
- Sweat-test, 806-807
- Sylvian fissure, meningiomas of, 715
- Sympathicoblastoma, undifferentiated, 800.  
   *See also* Neuroblastoma.
- Sympathectomy, for causalgia, 506  
   for chronic occlusive disease, 973-974  
   for post-thrombotic edema, 1033  
   for Sudeck's atrophy, 1204  
   lumbar, 1014  
   post-operative course and tests, 799  
   thoraco-lumbar, 797-798
- Syndesmosis, tibiofibular, diastasis of, 1273-1274
- Synkinesia, 684
- Synovectomy, in tuberculosis, 1050
- Synovial cysts, 1079  
   polyposis, 1079
- Synovioma, 1078-1079  
   benign, of fingers, 1103-1104

Tricuspid atresia, 614  
 Trigeminal tractotomy, 678  
 Trigger finger and thumb, 1165  
 Trousseau's sign, 929  
 Trusses, 209  
     for infants and children, 224-225  
 TSH, 724, 725  
 Tublin's technique in coarctation of aorta, 634  
 Tuberculoma, 544  
     intracranial, 722  
 Tuberculosis, aneurysms in, 995  
     buritis in, 1050-1051  
     deafness in, 697  
     diagnosis from anal fissure, 162  
     epididymitis in, 424, 426  
     erythema induratum in, 990  
     facial palsy in, 693  
     fistula-in-ano in, 165  
     genital (male), 443-444  
     genito-urinary, 437-444  
     hyperplastic, 116-117  
         diagnosis from carcinoma, 150  
         from Crohn's disease, 114  
     of anal canal, diagnosis from carcinoma, 176  
     of bone, 1046-1051, 1078  
         diagnosis of, 1046-1047  
         treatment of, general, 1049-1051  
         operative, 1050  
     of breast, 945-946  
     of face, 827  
     of intestine, 116-117  
     of jaw, 855  
     of kidney, 437  
     of knee-joint, 1134  
     of neck, 871-875  
     of shoulder-joint, 1098-1099  
     of spine, 1090-1094  
     of tongue, 850, 852  
     primary, 872-873  
     pulmonary, "destroyed" lung in, ex-  
         cision of, 544-545  
         diagnosis from bronchial carcinoma, 500  
         excision in, 543-546  
         indications, 544  
     extra-periosteal plastic plombage in, 541-543  
     extra-pleural pneumothorax for, 541  
     lobectomy for, 558  
     phrenic crush for, 532-533  
     surgery of, 529-548  
     thoracoplasty for, 533-540  
     thoracoscopy and section of adhesions, 530-532  
     treatment of, preventive, 873-874  
         with anti-tuberculous drugs, 875  
     ulcerative, of intestine, 117  
         diagnosis from Crohn's disease, 114  
     urinary, 439-444  
 Tumours, radiosensitivity of, 1324-1326.  
     *See also under names of organs and regions.*  
 Turban tumour, 829-830  
 Two-table method, in spinal fractures, 1232-1233

## U

Ulcer, anastomatic, 30-31  
     artefacta, 1012  
     carcinomatous, 1013  
     gravitational, 1010-1011  
     hypertensive, 1012  
     Marjolin's 1011  
     of leg, sympathectomy for, 798  
     peptic. *See* Peptic ulcer.  
     post-thrombotic, 1010-1011, 1013  
     rodent, 831  
     syphilitic, 1012  
     trophic, 1012  
     tropical, 1012  
     tuberculous, cauterisation of, 456  
     varicose. *See* Varicose ulcers.  
     venous, 1009-1014  
 Ulcerative colitis, 137-143. *See also* Colitis, ulcerative.  
 Ulna, upper end of, fractures of, 1223  
 Umbilicus, discharge from, 237-238  
 Unconscious patient, management of, 636-637  
 Unna's bandage, 1013  
 Urachus, persistent, 309  
 Uremia, diagnosis from intestinal obstruction, 126-127  
     in senile enlargement of prostate, 394  
     renal, in head injuries, 639  
 Urea clearance test, of renal function, 326-327  
 Ureter, abnormalities of, congenital, 306-309, 357-358  
     calculi of, 358-361  
     congenital dilatation of, 308-309  
     diseases of, 357-364  
     double, 328  
     duplication of, 306-307, 357  
     ectopia of, 307  
     failure of development of, 306  
     injuries of, 361-363  
     obstruction of, 307-309  
     operations on, 363-364  
     orifice of, congenital stenosis of, 357  
     papilloma of, 361  
     structure of, congenital, 307-308  
     tumours of, 361  
 Ureteric reflux, 363  
 Ureterocele, 307  
 Ureterocolic anastomosis, 364  
 Ureterolithotomy, 363  
 Ureterostomy, cutaneous, 364  
 Urethra, abnormalities of, congenital, 310  
     anatomy of, 407  
     bulbous, rupture of, 411  
     calculus of, 417  
     diseases of, 407-417  
     examination of, 408-410  
     foreign bodies in, 417  
     injuries of, 411-416  
         by instruments, 412  
     membranous, avulsion of, 411-412  
     operations on, 421-422  
     rupture of, complicating pelvic fracture, 1237

- Thrombophlebitis**, 996, 997-998  
*migrans*, 997  
 of *spermatic cord*, complicating operations for hernia, 213-214  
 portal, 80-81
- Thrombosis**, 981, 987  
 arterial primary, 108  
 coronary, 966  
 diagnosis from embolism, 982  
 iliofemoral, 998-999  
 intravascular, 961  
 mesenteric, intestinal obstruction and, 121  
   vascular occlusion and, 107  
 of cavernous and lateral sinuses, 657  
 of intra-cranial venous sinuses, 671  
 of retinal veins, papilloedema and, 671  
 "silent", 998  
 venous, 996-1001  
   appendectomy complicated by, 10  
   cholecystectomy complicated by, 59  
   deep, sequelæ of, 1001
- Thrush**, 845
- Thumb**, Bennett's stave fracture of, 1217-1218
- Thyrectomy**, anæsthesia for, 916-917  
 complications of, 919-921  
 results of, 920-921  
 technique of, 917-919
- Thymo-lympho-epithelioma**, 913
- Thymoma**, 520-521, 913-914
- Thymus**, in relation to myasthenia gravis, 912-921  
 tumours of, 912-921
- Thyroglossal cysts**, 865-866
- Thyroid gland**, adenoma of, 892-893, 910  
 anatomy of, 885-886  
 carcinoma of, 895-899  
   classification of, 896  
   clinical groups, 896-898  
   diagnosis from Riedel's thyroiditis, 895  
   endotracheal anæsthesia in, 902  
   treatment of, 898-899, 900  
   examination of, 886-887  
   investigations, 886-887  
   toxic manifestations of antithyroid drugs, 901  
   tumour of, ectopic, of tongue, 851
- Thyroidectomy**, for carcinoma, 899  
 in cardiac failure, 911  
 laryngeal nerve damage during, 692  
 partial, contraindication, 892  
 subtotal, indications for, 887, 888  
   post-operative care of, 907-908  
   complications, 908-909  
   preparation for, 901-902  
   technique of, 902-907
- Thyroiditis**, 893-895  
 acute, 893  
 chronic, 894  
 non-suppurative, subacute, 894
- Thyrototoxic crisis**, 908
- Thyrototoxicosis**, 890-892  
 preparation for operation, 901  
 primary, 890-892, 900  
 secondary, 890, 892, 900
- Thyrotropic hormone**. *See* TSH
- Tibia**, sabre, 1037
- Tibial plateau**, 1255-1256  
 shaft, fractures of, 1260-1265  
   continuous traction for, 1262-1263  
   delayed union, 1263-1264  
   fixed distraction for, 1263  
   oblique and partly oblique, 1261  
   open operation for, 1263  
   plaster wedging in, 1262  
   skin loss in, 1264-1265  
   spiral (helical) oblique and comminuted, 1261-1262  
   transverse, 1261  
 syndrome, anterior, 821-822
- Tic douloureux**, 675-678  
 treatment of, by alcoholic injections, 675-676  
   by division of sensory root of trigeminal nerve, 676-678
- Tiemann's catheter**, 381, 382, 392
- Tinel's sign**, 804, 808
- Tinnitus**, causes of, 688  
 acoustic nerve tumours and, 722
- Tissue grafts**, 904
- "Tobruk" splint**, 1249
- Todd's paralysis**, 757
- Toe(s)**, claw, 1149-1150  
 congenital deformities of, 1141-1142  
 fractures of phalanges, 1282-1283  
 nail, ingrowing, 1154  
 painful lesions of, 1154  
 second, dorsal dislocation of, 1153
- Tongue**, bifid, 848  
 black hairy, 849  
 carcinoma of, 845, 851-853  
 congenital abnormalities of, 848-849  
 fissured, malignant change in, 851  
 hemiatrophy of, 694  
 inflammation of, 849  
 leukoplakia of, 849  
 short frenulum, 848-849  
 syphilis of, 850, 852  
 tie, 848  
 tuberculosis of, 850, 852  
 tumours of, benign, 850-851  
 ulcers of, acute, 850  
   chronic, 850  
   dyspeptic, 849
- Tonsillectomy**, in tuberculous cervical adenitis, 875
- Tonsils**, malignant growths of, 882
- Topectomy**, 763
- Torkildsen's operation**, 743
- Torticollis**, acquired, 1090  
 congenital, 1089-1090
- Tracheitis**, treatment of, 907
- Tracheotomy**, 878-880
- Trauma**, arterial, chronic, 967  
 lymphoedema following, 1032  
 metabolic response to, 1304-1305  
 venous, 1014-1015
- Trench foot**, 981
- Trendelenberg's operation** for pulmonary embolism, 962  
 test, 1003, 1109-1110
- Troves**, bloodless fold of, 2

Water balance, in infancy and childhood, 297  
 deficiency, in traumatic coma, 637  
 Watt's lobotomy or leucotomy, 763  
 Weber-Klippel disease, 1021  
 Weber's test, 690  
 Wen, 828  
 Wharton's duct, stone in, 839  
 "White" leg, 098  
 Willis's classification of parotid tumours, 810  
     of swellings of jaws, 856  
 Wilms' tumour, 301-306, 347  
     *diagnosis from neuroblastoma*, 801  
     treatment of, 306  
 Worms, 170  
 Wounds, clinical examination of, 636  
     repairs of, complicating intracranial operations, 738

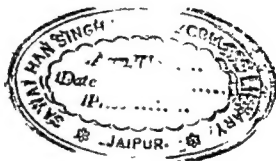
Wrist, burns of, 1297  
     dislocations of, 1166-1167, 1217  
     epidermoid cysts of, 1103  
     fractures of, 1215-1217  
     ganglia of, 1102  
     joint, 1106  
         dislocations of, 1166-1167, 1217  
     tenosynovitis around, 1104

## X

X-ray therapy, deep, 1319  
     superficial, 1319

## Z

Zenker's fluid, formula of, 767  
 Zondek-Aschheim reaction, in testicular tumours, 432



- Urethra, scales of calibration, 407-408  
 strictures of, 412-416  
   "diaphragm", 413  
   instrumental, 415-416  
   post-gonorrhœal, 412-413  
   post-inflammatory, 412-415  
   "tunnel", 413  
   valvular obstruction in, 310-311  
 Urethritis, 410-411  
   gonococcal, 410  
   non-gonococcal (non-specific), 410-411  
     abacterial pyuria and, 367  
     stricture following, 412  
 Urethrography, 409-410  
 Urethroscopy, 409-410  
   in stricture, 414  
 Urethrotiginitis, in female, 405-406  
 Urinary steroids, 930, 933  
   tract infections, treatment of, with anti-  
     biotics and sulphonamides, 1314  
 Urine, extravasation of, 416  
   incontinence of, 320-321  
   operative diversion from bladder, 363-364  
   retention of, acute, "immediate" pros-  
     tatectomy in, 399-400  
     bladder neck (including prostatic)  
       obstruction with, 399  
     effects and complications of, 390  
     treatment of, 390-393, 777-778  
     with overflow, 320
- V
- Vagotomy for duodenal ulcer, 28, 29, 30  
   technique of, 41  
 Valvotomy, for aortic stenosis, 625  
   for mitral stenosis, 594, 598-603  
     sequelæ, 604  
     pulmonary, 619-622  
 Van Slyke's urea clearance test, 326-327  
 Varicocele, 430-431  
   diagnosis from inguinal hernia, 208  
 Varicose ulcers, 1009-1010  
   veins, 1001  
     complications of, 1008-1009  
     of legs, 1001-1009  
       clinical features of, 1003  
       differential diagnosis of 1005  
       physical signs of, 1004-1005  
       primary, 1001-1003  
       secondary, 1003, 1006  
     recurrence of, causes of, 1008  
     stripping of, 1007  
     treatment of, 1005-1008  
 Varix, aneurysmal, 990  
 Vascular complications of bony injuries,  
   1203  
   occlusion, 107-108  
 Vasodilator drugs, 973  
 Vasopressin, 724. *See also* ADH.  
 Vasotomy, 436  
   for epididymitis, 425  
   for tuberculosis, 444  
 Veau-Wardill operation, 246-247  
 Vein(s), grafts, 994  
   inflammation of, 996-1001

- Vein(s), ligation, in varicose veins, 1007-  
   1008  
   peripheral, occlusion of, causing  
     lymphœdema, 1030  
   thrombosis of 996-1001  
 Venous obstruction, thoracic, 672  
 Ventricular septal defects, 624  
 Ventriculo-cisternostomy, 743  
 Ventricleulography, advantages of angio-  
   graphy over, 700  
   for epilepsy, 759  
   for meningioma, 710  
   for parieto-occipital tumours, 717  
   for tumours of frontal lobe, 707  
     of posterior fossa, 720  
     of temporal lobe, 713  
   indications for, 703  
   technique of, 701-702  
 Ventriculo-ureterostomy, technique of, 744  
 Verruæ, 828  
 Vertebrae, sciatica in lesions of, 786  
   metastasis to, 783  
 Vertigo, 688-690  
   causes of, central, 689  
   post-traumatic, 689  
 Vestibular function, tests of, 688  
 Viomycin, in pulmonary excision, 543  
 Viscera, injuries to, 1200  
   complicating fractures, 1237-1238  
 Visual fields, 672-673  
   defects of, in head injuries, 654  
   in temporal lobe syndrome, 711  
 Vitamin C treatment of scurvy, 1087  
   D treatment of osteomalacia, 1067  
   K in jaundice, 77  
 Vocal cords, paralysis of, King's operation,,  
   909  
 Vogt's classification of abnormalities of  
   œsophagus, 250  
 Volkmann's contracture, 982, 1201-1203  
   fibrosis, 965  
 Vulvulus, 132-134  
   intestinal obstruction and, 125  
 Vomiting, 688  
   in appendicitis, differential diagnosis, 6  
     obstructive, 3  
   in brain hæmorrhage, 753  
   in duodenal ulcer, 25  
   in extra-peritoneal disease, 126-127  
   in head injuries, 640, 643  
   in hydronephrosis, 288  
   in raised intracranial pressure, 706  
   in pancreatic carcinoma, 74  
   in paralytic ileus, 100  
   renal pain and, 320  
 Von Hippel's disease, 721

## W

- Wallerian degeneration, in peripheral nerve  
 injury, 803  
 Warts, penile, 419  
   plantar, 1145  
   seborrhœic, 829  
   virus, 828

- Water balance, in infancy and childhood, 297  
 deficiency, in traumatic coma, 637  
 Watt's lobotomy or leucotomy, 763  
 Weber-Klippel disease, 1021  
 Weber's test, 686  
 Wen, 828  
 Wharton's duct, stone in, 839  
 "White" leg, 998  
 Willis's classification of parotid tumours, 840  
     of swellings of jaws, 836  
 Wilms' tumour, 304-306, 347  
     diagnosis from neuroblastoma, 801  
     treatment of, 306  
 Worms, 170  
 Wounds, clinical examination of, 636  
     repairs of, 636  
     types of, 73
- Wrist, burns of, 1297  
 dislocations of, 1166-1167, 1217  
 epidermoid cysts of, 1103  
 fractures of, 1215-1217  
 ganglia of, 1102  
 joint, 1106  
     dislocations of, 1166-1167, 1217  
 tenosynovitis around, 1104
- X**
- X-ray therapy, deep, 1319  
 superficial, 1319
- Z**
- Zenker's fluid, formula of, 767